second edition

Handbook of EMOTION REGULATION

edited by **James J. Gross**

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SECOND EDITION

Edited by

James J. Gross

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To Paul, Mark, and Anne

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Preface

By any measure, the field of emotion regulation is thriving. Books, articles, special journal issues, and conferences related to emotion regulation seem to be everywhere. This high (and growing) level of interest in emotion regulation is reflected in citation trends. Until the early 1990s, there were just a few citations each year containing the term *emotion regulation*. By 2007—the year in which the first edition of this handbook was published—there were more than 3,000 Google Scholar citations for that year alone. In 2012, the citation count passed 8,000 for that year alone. Doing the math, it turns out that many more papers have been published on emotion regulation in the years since the first edition of this handbook was published than in all years up to—and including—2007. Hence, this second edition of the *Handbook of Emotion Regulation*.

The goal of this edition is to provide an authoritative and up-to-date account of findings in this field that will (1) encourage cumulative science by drawing together and integrating the specialized literatures on emotion regulation that exist in each of the subareas of psychology; and (2) facilitate cross-disciplinary dialogue about one of the most fascinating puzzles regarding the human condition, namely, that we are at once governed by—and governors of—our emotions. Of the 36 chapters in this new edition, two-thirds are brand new chapters for this edition, and the remaining chapters have been thoroughly updated. To reflect exciting new developments in the field, three new sections have been added in this edition: Psychopathology, Interventions, and Health Implications.

The *Handbook* begins with a section on foundations, in which I provide a conceptual and empirical orientation to the field. To this end, I first set emotion in the context of other affective processes. Next, I relate emotion regulation to other forms of affect regulation. I then selectively review what is known about emotion regulation goals, strategies, and outcomes. In the final part of the chapter, I highlight several fundamental questions and associated directions for future research on emotion and emotion regulation.

The second section considers biological bases of emotion regulation. Ochsner and I draw upon the human neuroimaging literature to present an integrated framework that links emotion and emotion regulation to other important forms of valuation, including affective learning, affective decision making, and expectancies, beliefs, and placebo effects. Proudfit, Dunning, Foti, and Weinberg review electroencephalographic studies of the temporal dynamics of emotion generation and emotion regulation, demonstrating that one particular event-related brain potential—the late positive potential—is sensitive to attentional and cognitive emotion regulation manipulations. Johnstone and

Walter describe the network of prefrontal and subcortical brain structures that are crucial for optimal health and show how this network is dysfunctional in clinical disorders. Finally, Gyurak and Etkin present a process-oriented neurobiological framework that seeks to integrate a wide range of implicit and explicit emotion regulation processes that are crucial to understanding mental health and illness.

The third section examines cognitive aspects of emotion regulation. Miller, Rodriguez, Kim, and McClure present a multiple systems account of delay discounting, and argue that delay discounting provides a framework for studying important instances of emotion regulation, including responses to temptations, procrastination, willpower, and addictions. Luerssen and Ayduk review the literature on delay of gratification, with an emphasis on identifying precursors, assessing the role of current affective state, linking delay of gratification to emotion regulation, and discussing insights from neuroimaging. Sheppes introduces the notion of emotion regulation choice; explains why it is crucial for healthy functioning; and explores the emotional, cognitive, and motivational determinants of emotion regulation choice. Finally, Grecucci and Sanfey review the literature on emotion and decision making, and make the case that emotion regulation influences our decision making in a number of important ways.

The fourth section focuses on developmental considerations, ranging from childhood through old age. Eisenberg, Hofer, Sulik, and Spinrad examine the development of effortful control in childhood and assess its impact on socioemotional, academic, and moral development. Thompson reviews the literature on the socialization of emotion and emotion regulation in the family, framing the socialization process in terms of an interaction between bottom-up and top-down influences. Riediger and Klipker consider emotion regulation in adolescence, with particular attention to the acquisition of emotion regulation skills, the motivation to regulate, and emotion regulation effectiveness. Finally, Charles and Carstensen take a lifespan perspective, and show how biological and motivational changes interact to shape the trajectory of emotion regulation throughout adulthood and into older age.

The fifth section considers social aspects of emotion regulation. Coan and Maresh present social baseline theory, which they use to explain how and why each of us uses other people to help regulate our emotions. Shaver and Mikulincer apply attachment theory to adolescents and adults, and show how this theory illuminates basic processes and individual differences in interpersonal emotion regulation. Jones, Kirkland, and Cunningham link the literature on attitudes and evaluation to emotion regulation, with particular attention to their iterative reprocessing model. Levenson, Haase, Bloch, Holley, and Seider consider emotion regulation in couples, with particular attention to the nature, development, and consequences of this form of social emotion regulation. Finally, Mesquita, De Leersnyder, and Albert present a cultural perspective on emotion regulation and show that regulatory patterns are aligned with cultural ideas and practices.

The sixth section examines personality processes and individual differences. Rothbart, Sheese, and Posner present a temperament systems approach, which provides a context for understanding individual differences, as well as the development of emotion regulation. John and Eng consider individual differences in emotion regulation, with particular attention to individual differences in coping, emotional competence, and specific regulatory processes such as reappraisal and suppression. Hofmann and Kotabe argue that desires share many similarities with emotion and show how an emotion regulation perspective can be used to organize findings relative to the regulation

of desire. Mauss and Tamir argue that emotion goals are critical to emotion regulation, and consider their content, hierarchical structure, and modes of operation. Finally, Leary and Gohar analyze the many ways self-awareness influences emotion regulation.

The seventh section focuses on emotion regulation and psychopathology. Campbell-Sills, Ellard, and Barlow review behavioral and neurobiological findings relative to the role of emotion regulation in the development, phenomenology, and treatment of anxiety disorders. Joormann and Siemer apply an emotion regulation perspective to mood disorders, with an eye toward understanding the factors that govern the onset and maintenance of these disorders. Kober discusses the role of emotion regulation in substance use disorders, featuring both the ways drugs regulate emotions and the ways in which emotion dysregulation is at once a cause for and a consequence of drug use. Finally, Barrett, Wilson-Mendenhall, and Barsalou examine emotion regulation from the perspective of situated conceptualizations and show how this perspective sheds new light on emotion dysregulation.

The eighth section considers clinical interventions designed to change emotion regulation. Mennin and Fresco present emotion regulation therapy, and show how this integrative intervention can be applied to generalized anxiety disorder and major depression. Neacsiu, Bohus, and Linehan describe dialectical behavior therapy, a treatment designed for individuals with severe and pervasive disorders of emotion regulation, such as borderline personality disorder. MacLeod and Grafton review research on attentional bias modification procedures and show how these procedures help individuals with conditions involving deficient emotion regulation. Berking and Schwarz describe the development of affect regulation training, which seeks to enhance emotional competence by training specific affect regulation skills. Finally, Farb, Anderson, Irving, and Segal review the literature on mindfulness meditation, showing how mindfulness meditation engages basic emotion regulation processes.

The ninth section of this handbook focuses on emotion regulation and health. Cole draws upon research on social genomics to show how emotional suppression might affect the molecular underpinnings of disease. Chen and Miller document how socioeconomic status predicts health outcomes, and argue that one reason for this association may be that low-socioeconomic-status children fail to learn adaptive forms of emotion regulation. Appleton and Kubzansky employ an emotion regulation framework to explain the association between negative emotions and cardiovascular disease. Finally, Wagner and Heatherton show how negative affect—and its misregulation—can lead to self-control failures ranging from binge eating to aggression.

Although this handbook is divided into sections, one of its major goals is breaking down barriers to cross-area communication. For this reason, there are considerably more cross-chapter links and citations than is typical in a handbook of this kind. There are also many points at which an author in one section will present material that makes contact with ideas, methods, and evidence from another section (e.g., developmental considerations in the health section; neuroscience in the cognitive section; analyses of individual differences in the social section). My hope is that these carefully assembled chapters—written by leading scholars in the field—will bring the field of emotion regulation together in a way that will be productive and new.

A large number of wonderful people helped to bring this handbook into being. I am grateful to Seymour Weingarten, Editor-in-Chief at The Guilford Press, for convincing me that the time was right for this handbook, and to Robert Levenson, with whom my work on emotion regulation began. I am also grateful to my many friends and col-

leagues who have helped shape my thinking about emotion regulation, and I would like to particularly thank the generous reviewers who provided helpful feedback on each of these chapters. Finally, I would like to acknowledge the contributions of the members of the Stanford Psychophysiology Laboratory, who help make Stanford such a fun place to be.

JAMES J. GROSS

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PART I

FOUNDATIONS

CHAPTER 1

Emotion Regulation: Conceptual and Empirical Foundations

James J. Gross

Emotions often are wonderfully helpful. They can direct attention to key features of the environment, optimize sensory intake, tune decision making, ready behavioral responses, facilitate social interactions, and enhance episodic memory. However, emotions can harm as well as help, particularly when they are of the wrong type, intensity, or duration for a given situation.

At such moments, we may try to *regulate* our emotions. This fundamental insight—that emotions can and should be regulated in certain situations—is well represented over the centuries in each of the major world traditions (for a more detailed historical overview of the field, see Gross, 1999).

In the past century, psychological investigations of emotion regulation have focused on psychological defenses (Freud, 1926/1959), stress and coping (Lazarus, 1966), attachment (Bowlby, 1969), and self-regulation (Mischel, 1996). However, until the early 1990s, there were relatively few papers each year containing the term *emotion regulation* (see Figure 1.1). Now there are thousands of new publications each year, making emotion regulation one of the fastest growing areas within the field of psychology (Koole, 2009; Tamir, 2011).

What is needed is a framework for organizing this bewildering array of findings. My aim in this chapter is to provide such a conceptual and empirical framework. Because a discussion of emotion regulation presupposes an understanding of what emotion is, in the first section I present the modal model of emotion and relate emotion to other affective processes. In the second section, I describe the process model of emotion regulation and distinguish emotion regulation from other forms of self-regulation. This prepares the way for the third section, in which I discuss key findings regarding emotion regulation goals, strategies, and outcomes. In the final section, I highlight three of the biggest challenges—and opportunities—for those interested in emotion regulation.

Emotions and Related Processes

One of the toughest questions in the field of affective science is one of the simplest, namely: What *is* an emotion? Theorists have tried to address this question by posing two other questions: What attributes are shared by all emotions (necessary conditions)? What attributes—if present—guarantee that something is an emotion (sufficient conditions)? Unfortunately, efforts to derive this kind of tidy classical definition of emotion are thwarted by the fact that *emotion* refers to an astonishing array of responses, from the mild to the intense, the brief to the extended,

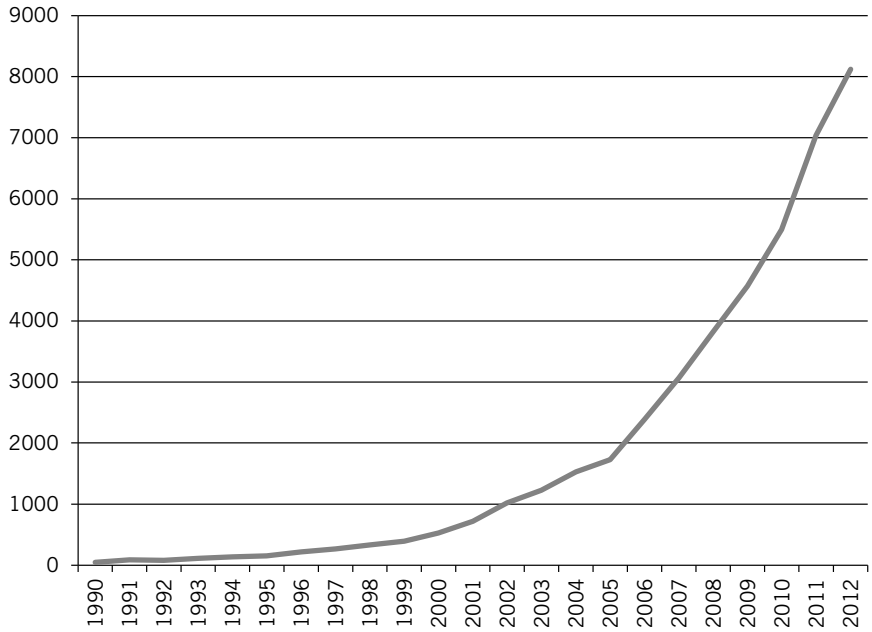

FIGURE 1.1. Number of publications containing the exact term *emotion regulation* in Google Scholar each year from 1990 to 2012 (Gross, 2013). Note that this is *not* a cumulative plot; each point represents 1 year's citations.

the simple to the complex, and the private to the public. Disgust at a prejudiced comment counts as an emotion. So does amusement at a funny mishap, anger at social injustice, joy at the prospect of receiving a promotion, surprise at a friend's "new look," grief at the death of a spouse, and embarrassment at a child's misbehavior. What are the core features of these diverse emotions?

Core Features of Emotion

The first core feature of emotion has to do with *when it occurs*. According to appraisal theory, emotions arise when an individual attends to and evaluates (appraises) a situation as being relevant to a particular type of currently active goal (Lazarus, 1991; Scherer, Schorr, & Johnstone, 2001). The goals that underlie this evaluation may be enduring (staying alive) or transient (wanting another piece of cake). They may be conscious and complicated (aspiring to become a professor) or unconscious and simple (trying to avoid stepping in puddles). They may be widely shared (having close friends) or highly idiosyncratic (finding a new way of tying one's shoes). Whatever the goal, and whatever meaning the situation has in light of the goal, it is this meaning that gives rise to emotion. As this meaning changes over time—due either to changes in the situation itself or changes in the meaning the situation holds for the individual—the emotion will also change.

The second core feature of emotion has to do with its *multifaceted nature*. Emotions are whole-body phenomena that involve loosely coupled changes in the domains of subjective experience, behavior, and central and peripheral physiology (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). The subjective aspect of emotion is so central to many instances of emotion that the terms "emotion" and "feeling" often are used interchangeably. But emotions not only make us feel, they also incline us to act (Frijda, 1986). These impulses to act in certain ways (and not act in others) include changes in facial behavior and body posture, as well as situation-specific instrumental actions such as staring, hitting, or running.

These changes in experience and behavior are associated with autonomic and neuroendocrine responses that both anticipate emotion-related behaviors (thereby providing metabolic support for the action) and follow them, often as a consequence of the motor activity associated with the emotional response (Lang & Bradley, 2010). As functionalist accounts of emotion make clear, the multifaceted responses that comprise emotion often (but not always) are useful in helping to achieve the goals that gave rise to emotions in the first place (Levenson, 1999).

The Modal Model of Emotion

These core features constitute what has been referred to as the *modal model* of emotion—so called because these features are evident in many different approaches to emotion (Barrett, Ochsner, & Gross, 2007; Gross, 1998a). According to this model, emotions involve person–situation transactions that compel attention, have meaning to an individual in light of currently active goals, and give rise to coordinated yet flexible multisystem responses that modify the ongoing person–situation transaction in crucial ways. The modal model lies at the heart of lay intuitions about emotion and also represents key points of convergence among researchers and theoreticians concerned with emotion.

In Figure 1.2, I present the situation–attention–appraisal–response sequence specified by the modal model of emotion in highly abstracted and simplified form. This sequence begins with a psychologically relevant situation. Often, this is a situation that can be specified by referring to features of the external environment (e.g., the snake slithering into my tent). However, psychologically relevant "situations" can also be internal (e.g., the sneaking suspicion that I'll never amount to anything). Whether external or internal, situations are attended to in various ways, giving rise to appraisals that constitute the individual's assessment of what the situation means in light of relevant goals (Ellsworth & Scherer, 2003). The emotional responses generated by these unfolding appraisals involve changes in experiential, behavioral, and neurobiological response systems.

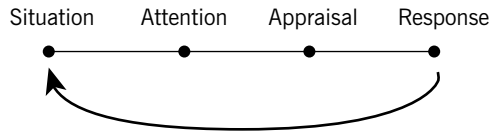

FIGURE 1.2. The modal model of emotion.

Like many other responses we make, emotional responses often change the situation that gave rise to the response in the first place. Figure 1.2 depicts this aspect of emotion by showing the response looping back to (and modifying) the situation that gave rise to the emotion. To make this idea more concrete, imagine a husband and wife heatedly disagreeing about whether household chores are being fairly divided. Several minutes into the discussion, the husband starts to cry (yes, the husband). This emotional response dramatically alters the interpersonal situation. This new situation now gives rise to a new response from the wife—now no longer angry but instead feeling compassion. This compassionate response itself further changes the interpersonal situation, giving rise to other emotions in each of them. The key idea here is that emotional responses often lead to changes in the environment that alter the probability of subsequent instances of that and other emotions (for a more detailed discussion of this point, see Gross & Thompson, 2007).

Emotions and Other Affective Processes

One thing that makes the emotion literature challenging is that many different terms are used to refer to emotion-related processes, including affect, emotion, stress, and mood (Davidson, 1994). Unfortunately, these terms are used in different ways by different researchers, leading at times to some degree of "conceptual and definitional chaos" (Buck, 1990, p. 330). To organize this chaotic landscape, I find it useful to view *affect* as the umbrella term for states that involve relatively quick good–bad discriminations (Scherer, 1984). These affective states include (1) *emotions* such as anger and sadness, (2) *stress responses* to circumstances that exceed an individual's ability to

cope, and (3) *moods* such as depression and euphoria.

How are these various affective processes distinguished? Although both emotion and stress involve whole-body responses to significant events, "stress" typically refers to negative (but otherwise unspecified) affective responses, whereas "emotion" refers to both negative and positive affective states (Lazarus, 1993). Emotions also may be distinguished from moods (Parkinson, Totterdell, Briner, & Reynolds, 1996). Moods often last longer than emotions, and compared to moods, emotions are typically elicited by specific objects and give rise to behavioral response tendencies relevant to these objects. By contrast, moods are more diffuse, and although they may give rise to broad action tendencies such as approach or withdrawal (Lang, 1995), moods bias cognition more than they bias action (Siemer, 2001).

Lest these distinctions seem academic, consider the term *affect*. From my perspective, *affect* belongs at the top of the hierarchy, as the superordinate term in this set of emotion-related terms. However, others take a different view. For example, some use the terms *affect* and *emotion* interchangeably (Zajonc, 1984). For others, *affect* refers to the experiential component of emotion (Buck, 1993; MacLean, 1990). Still others use *affect* to refer to the behavioral component of emotion (American Psychiatric Association, 2013; Kaplan & Sadock, 1991). As these observations suggest, clarity regarding how each of these constructs is being used is a necessary prerequisite for an analysis of how these various processes are (or are not) regulated (Gross, 2010).

Emotion Regulation and Related Processes

Emotion regulation refers to shaping which emotions one has, when one has them, and how one experiences or expresses these emotions (Gross, 1998b). Thus, emotion regulation is concerned with how emotions themselves are regulated (regulation *of* emotions), rather than how emotions regulate something else (regulation *by* emotions). Defined in this way, many different activities count as emotion regulatory. These include pounding your pillow when you're angry at a boss, imagining your audience naked when you're nervous about performing in a piano recital, picking up the phone to call a friend when you're feeling sad, telling a child who is having a tantrum not to act like such a baby, anticipating going to a fun party on the weekend to reenergize yourself midweek, going for a run after an upsetting fight with a friend, playing calming music after a long day at work, leaving a tense meeting early to cool down, going to a club to have a drink, and watching *It's a Wonderful Life* for the 600th time. Because there seems to be no limit to the activities that may qualify as emotion regulatory, what is needed—as with emotion—is a description of its core features.

Core Features of Emotion Regulation

The first core feature of emotion regulation is the *activation of a goal* to modify the emotion-generative process (Gross, Sheppes, & Urry, 2011). This goal may be activated either in oneself or in someone else. To mark this distinction, it is useful to refer to *intrinsic emotion regulation* in the first case (James regulates his own emotions: emotion regulation *in self*) and to *extrinsic emotion regulation* in the second case (James regulates Sarah's emotions: emotion regulation *in another*). Researchers who work with adults typically focus on intrinsic emotion regulation (Gross, 1998b; but see Levenson, Haase, Bloch, Holley, & Seider, this volume). By contrast, researchers who work with infants and children typically focus on extrinsic emotion regulation (e.g., Cole, Martin, & Dennis, 2004). Although this distinction is often helpful, it is worth noting that in some situations intrinsic and extrinsic emotion regulation co-occur, such as when James regulates Sarah's emotions (extrinsic regulation) in order to calm himself down (intrinsic regulation).

The second core feature of emotion regulation is the *engagement of the processes that are responsible for altering the emotion trajectory*. Many different processes can be recruited to regulate emotions, and these vary considerably in the degree to which they are explicit versus implicit. Many prototypical instances of emotion regulation are explicit, and thus conscious, such as when

we try hard to look calm even though we are very anxious before a talk, or when we try to look on the bright side of a bad outcome to cheer ourselves up. However, emotion regulatory activity can also be implicit and take place without conscious awareness. Examples include hiding the affection one feels for another person due to a fear that one will be rejected, or quickly turning one's attention away from potentially upsetting material. Previous discussions have distinguished categorically between explicit and implicit processes (Masters, 1991). However, it may be more useful to think of a continuum of emotion regulation possibilities that range from explicit, conscious, effortful, and controlled regulation to implicit, unconscious, effortless, and automatic regulation (Gyurak & Etkin, this volume; Gyurak, Gross, & Etkin, 2011).

The third core feature of emotion regulation is its *impact on emotion dynamics* (Thompson, 1990), or the latency, rise time, magnitude, duration, and offset of responses in experiential, behavioral, or physiological domains. Depending on the individual's goals, emotion regulation may increase or decrease the latency, rise time, magnitude, duration, or offset of the emotional response (compared to the emotional response that would have occurred in the absence of emotion regulation) (Gross, 1998b). Emotion regulation also may change the degree to which emotion response components cohere as the emotion unfolds, such as when changes in emotion experience and physiological responding occur in the absence of facial behavior (Dan-Glauser & Gross, 2013).

These three core features of emotion regulation—the activation of a regulatory goal, the engagement of regulatory processes, and the modulation of the emotion trajectory—are common features of many diverse types of emotion regulation. In a later section, I turn to a more complete discussion of each of these three core features, as I review what is known about emotion regulation goals, emotion regulation strategies, and emotion regulation outcomes. Before elaborating upon each of these core features, however, we need to consider what makes different forms of emotion regulation so different from one another.

The Process Model of Emotion Regulation

One framework that has proven useful for addressing this question is the process model of emotion regulation (Gross, 1998b). This information-processing model takes as its starting point the modal model (Figure 1.2), which—as we have seen—specifies the sequence of processes involved in emotion generation. The process model of emotion regulation builds on the modal model, and treats each step in the emotion-generative process that is described in the modal model as a potential target for regulation. In Figure 1.3, I present the process model, which highlights five points at which individuals can regulate their emotions.

These five points represent five families of emotion regulation processes: situation selection, situation modification, attentional deployment, cognitive change, and response modulation (Gross, 1998b). These

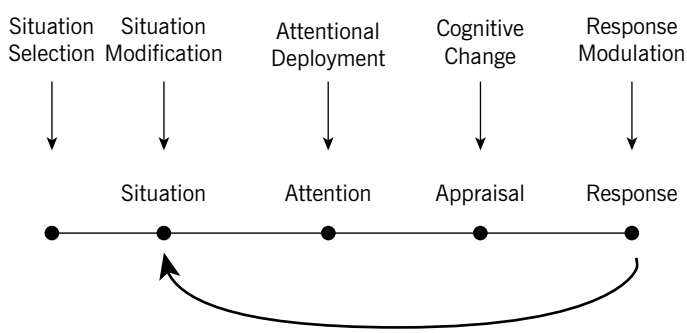

FIGURE 1.3. The process model of emotion regulation.

families are distinguished by the point in the emotion-generative process at which they have their primary impact. Movement from left to right in Figure 1.3 represents movement through time: A particular situation is selected, modified, attended to, appraised, and yields a particular set of emotional responses. However, as emphasized in Figure 1.2, emotion generation is an ongoing process, extending beyond a single episode. This dynamic aspect of emotion and emotion regulation is signaled by the feedback arrow in Figure 1.3 from the emotional response back to the situation (there may in fact be many such points of feedback). I describe these five families of regulatory strategies in more detail below.

Emotion Regulation and Related Constructs

Before considering emotion regulation processes in greater detail, however, it is important to note in passing that—paralleling the distinctions drawn among members of the affective family presented earlier—emotion regulation can be seen as subordinate to the broader construct of *affect regulation*. Under this broad heading fall all manner of efforts to influence our valenced responses (Westen, 1994), including (1) *emotion regulation*, (2) *coping*, and (3) *mood regulation*. Because virtually all goal-directed behavior can be construed as maximizing pleasure or minimizing pain—and thus as affect regulatory in a broad sense—it is frequently useful to sharpen the focus by examining one or more of these three second-level families of processes.

Coping is distinguished from emotion regulation both by its predominant focus on decreasing negative affect and its emphasis on much larger periods of time (e.g., coping with bereavement). As noted earlier, moods are typically of longer duration and are less likely to involve responses to specific "objects" than are emotions (Parkinson et al., 1996). In part due to their less well-defined behavioral response tendencies, in comparison with emotion regulation, mood regulation and mood repair are more concerned with altering emotion experience than emotion behavior (Larsen, 2000). It is not yet known whether the regulation of emotion, stress responses, and moods are more similar than different, more different than similar, or somewhere in between. It therefore is usually a good idea to pay close attention to the type of affect targeted for regulation.

Emotion Regulation Goals, Strategies, and Outcomes

As we have seen, emotion regulation has three core features. The first—the emotion regulation *goal*—is what people are trying to accomplish. The second—the emotion regulation *strategy*—is the particular processes that are engaged in order to achieve that goal. The third—the *outcome*—refers to the consequences of trying to achieve that particular emotion regulation goal using that particular strategy. In the following sections, I review each of these three core features of emotion regulation in turn, selectively highlighting what we know about each.

Emotion Regulation Goals

If asked about times they have tried to regulate their emotions, people often describe efforts to down-regulate negative emotions (i.e., diminish their intensity or duration), especially anger, sadness, and anxiety, with a particular focus on decreasing the experiential and behavioral aspects of negative emotions (Gross, Richards, & John, 2006). People also report trying to up-regulate positive emotions (i.e., increase their intensity or duration), especially love, interest, and joy, often by sharing their positive experiences with others (Quoidbach, Berry, Hansenne, & Mikolajczak, 2010). These reports of everyday emotion regulation are consistent with traditional hedonic accounts of affect regulation, which assume that individuals are motivated to decrease negative emotional states and increase positive emotional states (Larsen, 2000).

It turns out, however, that there is more to emotion regulation than this. Indeed, the down-regulation of negative emotions and the up-regulation of positive emotions can be seen as just two cells in the 2×2 matrix shown in Figure 1.4. It may seem odd to imagine people wanting more of a "bad" thing or less of a "good" thing, but there are many reasons people might want

	Decrease	Increase
Negative emotion	Trying to calm oneself down when angry (Int) Helping a tearful child untangle his kite (Ext)	Firing oneself up before a big game (Int) Reframing a friend's "little fight" with a spouse as serious (Ext)
Positive emotion	Wiping a smile off one's face at a funeral (Int) Helping giggling girls calm down at bedtime (Ext)	Sharing great news with close friends (Int) Telling someone a joke to cheer her up (Ext)

FIGURE 1.4. Emotion regulation goals can include efforts to decrease or increase either the magnitude or duration of negative or positive emotion. Decreasing negative emotion appears to be the most common regulation goal in everyday life, followed by increasing positive emotion. Emotion regulation may be either intrinsic (Int) or extrinsic (Ext).

to up-regulate negative emotions or down-regulate positive emotions, as Parrott (1993) has observed in the context of mood regulation. Motives for up-regulating negative emotions include promoting a focused, analytic mindset; fostering an empathic stance; and influencing others' actions. Motives for down-regulating positive emotions include maintaining a realistic mindset; being mindful of social conventions; and concealing one's feelings from others.

This broader view of emotion regulation goals suggests something important about what people are trying to accomplish when they regulate their emotions. Sometimes—and perhaps often—people are motivated by "hedonic considerations," or the wish to increase short-term pleasure and decrease short-term pain. At other times, however, people are motivated by "instrumental considerations"; that is, they are motivated to change their emotions in order to achieve some other, nonemotional outcome (Mauss & Tamir, this volume; Tamir, 2009). Sometimes, these instrumental goals may be related to specific work demands, such as appearing relaxed and upbeat for nervous airline passengers (Hochschild, 1983), seeming calm yet empathic for nervous medical patients (Larson & Yao, 2005), showing high levels of interest in students (R. E. Sutton, 2004), or sounding angry when trying to collect payment on debts (R. I. Sutton, 1991). At other times, these instrumental goals are related to broader cultural imperatives (Mesquita, De Leersnyder, & Albert, this volume); these may dictate that people show (Szczurek, Monin, & Gross, 2012) or feel (Tsai, 2007) particular emotions and not others in a given situation.

Emotion Regulation Strategies

Whatever emotion regulation goals people may have, they can do many different things to achieve them. They can even do many different things at once—or at least in quick succession. For example, after a stressful day, some people might turn off their cell phone, have a beer, and watch an entertaining program on television while holding hands with their partner. This kind of mixing of regulation strategies is probably common in everyday life. For analytic purposes, however, the process model distinguishes five families of regulatory processes.

The most forward-looking approach to emotion regulation is *situation selection*. This type of emotion regulation involves taking actions that make it more (or less) likely that one will end up in a situation that one expects will give rise to desirable (or undesirable) emotions. Examples include avoiding a grumpy neighbor, arranging a play date for a child, or seeking out a friend with whom one can have a good cry. Despite the commonness of situation selection, it is hard to tell how one will feel in different situations (in the case of intrinsic regulation), and harder still to be sure how another person will feel in various situations (in the case of extrinsic regulation).

Situation modification refers to directly modifying a situation so as to alter its emotional impact. When one's parents visit at college, situation modification may take the form of hiding piles of dirty laundry or questionable artwork. Parents also engage in their share of situation modification, which ranges from helping with frustrating math problems to suggesting games to play on a rainy day. Because efforts to modify a

situation may effectively call a new situation into being, it is sometimes difficult to distinguish between situation selection and situation modification. Also, although "situations" can be external or internal, situation modification—as I mean it here—has to do with modifying external, physical environments. Modifying "internal" environments (i.e., thoughts) will be considered later, in the context of cognitive change.

Attentional deployment refers to directing attention within a given situation in order to influence one's emotions. Attentional deployment is one of the first emotion regulatory processes to appear during development (Rothbart, Ziaie, & O'Boyle, 1992), and it is used from cradle to grave, particularly when it is not possible to modify one's situation. One of the most common forms of attentional deployment is *distraction*, which focuses attention on other aspects of the situation or moves attention away from the situation altogether; distraction also may involve changing internal focus, such as when someone calls to mind thoughts or memories that help to instantiate the desired emotional state (Thiruchselvam, Hajcak, & Gross, 2012).

Cognitive change refers to modifying how one appraises a situation so as to alter its emotional significance, either by changing how one thinks about the situation or about one's capacity to manage the demands it poses. Sometimes, cognitive change is applied to an external situation (e.g., "This interview is a chance for me to learn more about the company"). At other times, cognitive change is applied to an internal situation (e.g., "I'm not anxious—I'm getting 'pumped up' for a game, and this will help me play my best"). One particularly well-studied form of cognitive change is *reappraisal*; this form of cognitive change is often used to decrease negative emotions, but it can also be used to increase or decrease negative or positive emotions (Samson & Gross, 2012).

The fifth family of emotion regulatory processes, *response modulation*, occurs late in the emotion-generative process, after response tendencies have already been initiated, and refers to directly influencing experiential, behavioral, or physiological components of the emotional response. Physical exercise and deep-breathing relaxation techniques can be used to decrease experiential and physiological aspects of negative emotions, and alcohol, cigarettes, drugs, and even food also may be used to modify emotion experience. Another common form of response modulation involves regulating emotion-expressive behavior. One well-researched example of response modulation is *expressive suppression*, in which a person tries to inhibit ongoing negative or positive emotion-expressive behavior.

Emotion Regulation Outcomes

At the heart of the process model is the intuition that different forms of emotion regulation might have different consequences, both immediately and over the long term. This prediction flows from the idea that if emotions develop over time, then intervening at different points in the emotion-generative process should lead to different outcomes.

To test this idea, researchers have used both experimental and correlational approaches to investigate the affective, cognitive, and social consequences of different types of emotion regulation. This work is yielding a rich and nuanced understanding of how specific forms of emotion regulation affect both the people who are doing the regulating and the people around them.

To illustrate this rapidly growing body of work, I focus on one of the most well-researched contrasts in the field, namely, the contrast between reappraisal (from the cognitive change family) and suppression (from the response modulation family). This contrast is an interesting one because although both suppression and reappraisal are commonly employed to down-regulate emotion, *suppression* is a behaviorally oriented form of emotion regulation in which a person decreases emotion-expressive behavior while emotionally aroused, whereas *reappraisal* is a cognitively oriented form of emotion regulation in which a person tries to think about a situation in a way that alters the emotional response (for a more comprehensive review of the effects of different emotion regulation strategies, see Webb, Miles, & Sheeran, 2012).

Affectively, experimental studies have shown that suppression leads to decreased positive but not negative emotion experience (Gross, 1998a; Gross & Levenson, 1993, 1997; Stepper & Strack, 1993; Strack, Martin, & Stepper, 1988), increased sympa-

thetic nervous system responses (Demaree et al., 2006; Gross, 1998a; Gross & Levenson, 1993, 1997; Harris, 2001; Richards & Gross, 2000), and greater activation in emotion-generative brain regions such as the amygdala (Goldin, McRae, Ramel, & Gross, 2008). Correlational studies are largely congruent with these experimental findings and, if anything, suggest a more negative profile of affective consequences for suppression, in that compared to people who do not report using suppression, people who report using suppression experience less positive emotion and more negative emotion, including painful feelings of inauthenticity as well as depressive symptoms (Gross & John, 2003; Moore, Zoellner, & Mollenholt, 2008; Nezlek & Kuppens, 2008).

By contrast, experimental studies have shown that reappraisal leads to decreased levels of negative emotion experience and increased positive emotion experience (Gross, 1998a; Feinberg, Willer, Antonenko, & John, 2012; Lieberman, Inagaki, Tabibnia, & Crockett, 2011; Ray, McRae, Ochsner, & Gross, 2010; Szasz, Szentagotai, & Hofmann, 2011; Wolgast, Lundh, & Viborg, 2011), has no impact on or even decreases sympathetic nervous system responses (Gross, 1998a; Kim & Hamann, 2012; Stemmler, 1997; Shiota & Levenson, 2012; Wolgast et al., 2011), and leads to lesser activation in emotion-generative brain regions such as the amygdala (Goldin et al., 2008; Kanske, Heissler, Schonfelder, Bongers, & Wessa, 2011; Ochsner & Gross, 2008; Ochsner et al., 2004) and ventral striatum (Staudinger, Erk, Abler, & Walter, 2009). Correlational studies suggest that compared to people who do not use reappraisal, people who use reappraisal experience and express more positive emotion and less negative emotion, including fewer depressive symptoms (Gross & John, 2003; Nezlek & Kuppens, 2008). Reappraisers' reports of less negative emotion are corroborated by functional imaging studies that show less activation in emotion-related regions such as the amygdala (Drabant et al., 2009).

Cognitively, experimental studies have shown that suppression leads to worse memory (Johns, Inzlicht, & Schmader, 2008; Richards, Butler, & Gross, 2003; Richards & Gross, 1999, 2000, 2006). Correlational findings support these conclusions: Individuals who typically use suppression have worse memory for emotional interactions than do individuals who use suppression less frequently (Richards & Gross, 2000).

By contrast, experimental studies have found that reappraisal either has no impact on subsequent memory or actually improves it (Richards & Gross, 2000; Hayes et al., 2011), and can enhance performance on standardized exams (Jamieson, Mendes, Blackstock, & Schmader, 2010). Correlational studies bear out these findings, showing that individuals who typically reappraise have comparable or even enhanced memory compared to others (Richards & Gross, 2000).

Socially, experimental studies have reported that suppression leads to less liking from social interaction partners, and to an increase in partners' blood pressure levels (Butler et al., 2003). Correlational studies support these laboratory findings. Individuals who typically use suppression report avoiding close relationships and having less positive relations with others; this dovetails with peers' reports that suppressors have relationships with others that are less emotionally close (English, John, & Gross, 2013; Gross & John, 2003; Srivastava, Tamir, McGonigal, John, & Gross, 2009).

Reappraisal, by contrast, has no detectable adverse consequences for social affiliation in a laboratory context (Butler et al., 2003). Correlational studies support these findings: Individuals who typically use reappraisal are more likely to share their emotions—both positive and negative—and report having closer relationships with friends, which matches their peers' reports of greater liking (Gross & John, 2003; Mauss et al., 2011).

Across these three outcome domains, reappraisal seems preferable to suppression. However, caution is required here, because the effects of emotion regulation vary by context. Thus, the adverse social consequences of suppression are not evident in individuals with bicultural European–Asian values (Butler, Lee, & Gross, 2007; Soto, Perez, Kim, Lee, & Minnick, 2011). Similarly, some of the benefits of reappraisal are moderated by context. For example, if emotional intensity is already high when reappraisal is engaged, it no longer has the experiential or physiological benefits seen in other contexts (Sheppes, Catran, & Meiran, 2009).

The context specificity of the effects of suppression and reappraisal (and, presumably, other forms of emotion regulation) means that global conclusions about one strategy being "better" than another are likely to be misleading. Indeed, any given emotion regulation strategy may be used to make things either better or worse, depending on whose point of view is adopted, on the outcome of interest, and on details regarding the context. For example, cognitive strategies that dampen negative emotions may help a medical professional operate efficiently in stressful circumstances, but they also may neutralize negative emotions associated with empathy and thereby decrease helping. It also bears emphasizing that regulatory strategies may accomplish one person's goals at the expense of another's. For example, a mother may accomplish her goals when she stops a child from crying for candy in the supermarket, but this success may come at the expense of the child's failure to achieve his or her goal of getting candy.

Fundamental Questions and Directions for Future Research

As is the case with any new and vital area of science, the study of emotion regulation has generated many more questions than answers (Gross, 2013). In the following sections, I describe three of the questions I think are particularly important to the field of emotion regulation.

How Separable Are Emotion and Emotion Regulation?

One of the most intuitively compelling distinctions in the field of emotion research is that between emotion and emotion regulation. We feel angry, and try not to show it. A child cries, and we comfort her. We are discouraged, and try to find hope. In each case, it seems utterly obvious that one set of psychological processes governs the emergence of an emotion, and another governs whether and how we manage these emotions.

However, the closer one looks, the harder it is to draw a bright line between emotion and emotion regulation (Gross et al., 2011). Many situations seem to call forth both emotion and emotion regulation (Campos, Frankel, & Camras, 2004), and many of the brain systems that give rise to emotion are also engaged by emotion regulation (Ochsner et al., 2009). This has led some commentators to argue that the two sets of processes are so intertwined that no clear distinction can be made between them (Kappas, 2011; Thompson, 2011).

Part of the problem here is that there are many different ways to define emotion, each of which suggests a different take on how (and whether) emotion and emotion regulation should be distinguished (Gross & Barrett, 2011). From my perspective, the crucial distinction between emotion and emotion regulation is a functional one. As we have seen, an emotion arises when a person attends to a situation that he or she evaluates as relevant to a particular type of goal. For example, I may feel angry at others when they throw garbage from their cars. I may even have an emotion about my anger response. For example, I may feel proud that I feel anger at others who are degrading the environment. Emotion regulation may be said to occur when (1) an emotional response itself is subject to valuation as good or bad, *and* (2) this valuation leads to the activation of a goal to change that particular emotion response trajectory. To continue the earlier example, if I find myself getting so annoyed at others who pollute the environment that I snap at my children, I may negatively value my anger and feel upset that I am snapping at my children. If this is all that happens, there's no emotion regulation—just two overlapping instances of emotion (anger at polluters, and upset at myself for snapping at my children). But if this feeling of upset leads me to try to curb my anger, then this would be an instance of emotion regulation.

A more general way of putting this idea is to say that emotion regulation involves the valuation of a valuation. That is, an emotional response is itself judged to be good or bad—hence leading to an affective response about the target emotional response—and this second affective response motivates an effort to modify the first affective response. This perspective on emotion regulation is functional in that it doesn't define a priori what should "count" as emotion versus emotion regulation; instead, the question of separability hinges on whether a goal has been activated to influence the emotion-

generative process itself (for a more detailed exposition of this valuation perspective on emotion regulation, see Ochsner & Gross, this volume).

Why Do People Regulate Their Emotions as They Do?

Anyone who has ever seen two grown men step out of their cars to fight over who is the bigger idiot has likely wondered why on earth the two can't manage to regulate their emotions in more productive ways. This puzzlement points to a more general question about why people regulate (or fail to regulate) their emotions as they do. Answering this question requires a more complete analysis of the emotion regulatory process than I have provided so far, and one of the most pressing challenges for researchers in this area is to contribute to this analysis. In answering this overarching question, a number of more specific questions must be addressed:

1. What leads people to activate a goal to regulate emotion?
2. What determines the fate of this regulatory goal?
3. Which strategy is employed to achieve a given emotion regulatory goal?

Why do some people activate a goal to regulate emotions when others do not? One reason may be differences in awareness of the person's own ongoing (or anticipated) emotional responses—in the case of intrinsic emotion regulation—or in awareness of another person's ongoing (or anticipated) emotional response—in the case of extrinsic emotion regulation. People differ substantially in their ability to track subtle emotion dynamics and represent these in a differentiated fashion; some do this very well, but others (e.g., those who have alexithymia or low levels of emotion awareness) have little or no awareness of ongoing emotional responses (Salovey & Mayer, 1990; Taylor, 1994). Emotional awareness appears to be a crucial rate-limiting factor in successfully regulating emotions (Barrett, Gross, Conner, & Benvenuto, 2001; Samson, Huber, & Gross, 2012), but much remains to be learned about the precise role of emotional awareness in activation of the goal to engage in emotion regulation.

Even after a person has become aware of an emotion and activated a goal to regulate that emotion, there remains the question: What determines how this regulatory goal will fare in its competition with other currently active goals? As discussed earlier, people have both hedonic and instrumental goals, but it is far from clear how these various goals interact. What is known suggests that emotion regulation often involves tradeoffs between hedonic and instrumental motives. Avoidance that may bring short-term relief ("If I skip the cocktail party, I can avoid feeling anxious") may have a substantial long-term price tag ("If I skip the cocktail party, I may miss out on developing helpful professional contacts"). Over the course of development, it appears that the balance of motives shifts repeatedly, first from hedonic to instrumental goals, then, later in life, away from instrumental motives and toward hedonic motives, reflecting an awareness of the reduced value of long-term investments as one moves toward the end of one's life (Carstensen, Isaacowitz, & Charles, 1999; Charles & Carstensen, this volume). Just how people flexibly manage competing regulatory goals is likely to be an important determinant of healthy adaptation.

Once an emotion regulatory goal has been activated and has survived a competition with other currently active goals, there remains the question: Which emotion regulatory strategy (or strategies) will be selected in order to achieve that particular emotion regulatory goal? Part of the answer may hinge on context-specific factors, such as the type and intensity of emotion that needs regulating. For example, people prefer reappraisal to distraction when emotion intensity is low, but prefer distraction to reappraisal when emotion intensity is high, because at high-intensity levels, reappraisal is often no longer effective (Sheppes, this volume; Sheppes, Scheibe, Suri, & Gross, 2011). Another important context-specific factor may be a person's perceptions of his or her currently available social and/or psychological resources (Coan & Maresh, this volume; Opitz, Gross, & Urry, 2012). Other factors that govern strategy selection may be more stable across situations. For example, some people have incremental beliefs about emotion, and see emotions as the kinds of

things that can be changed. Others have entity beliefs about emotion, and see emotions as relatively immutable. Perhaps not surprisingly, those with incremental beliefs seem to be more adept at emotion regulation than those with entity beliefs (Mauss & Tamir, this volume; Tamir, John, Srivastava, & Gross, 2007). Another important kind of belief has to do with whether one believes one is able to engage in a particular form of emotion regulation when one wishes to do so. This type of belief is referred to as *emotion regulation self-efficacy*, and self-efficacy beliefs can be modified. For example, in the context of generalized social anxiety disorder, patients who received cognitive-behavioral therapy (vs. those randomized to a wait-list group) showed increased reappraisal self-efficacy, and these changes in self-efficacy mediated the effects of therapy on clinical improvement (Goldin et al., 2012; John & Eng, this volume).

It is evident that these three questions—regarding the activation of a regulatory goal, the relative dominance of that regulation goal compared to other goals, and the emotion regulation strategy that is selected—represent a small subset of the many questions that need to be answered before we fully understand whether (and how) a particular person will regulate emotion in a particular situation, and whether he or she will do so successfully. Other determinants include person-based factors, such as working memory capacity, as well as situation-based factors that make some forms of emotion regulation easier to implement than others. One pressing challenge for future research is to clarify the rules that govern the skillful application of emotion regulation. This work is important, because it will create a framework for understanding individual and group differences in emotion regulation, and suggest strategies for intervention when regulation is deficient.

How Can We Use What We Know to Make the World a Better Place?

Although we still have much to learn about the psychological processes that are necessary for skillful and flexible emotion regulation, we now know enough to begin thinking about how to use what we know about emotion regulation to make the world a better place. Efforts in this direction are justified by not only their ends but also what they can teach us about basic processes as we apply what we think we know to real-world situations.

One type of application—and perhaps the most obvious—is individual-level interventions designed to teach healthier patterns of emotion regulation (Gross & Munoz, 1995). Such interventions might take the form of crafting instructional materials, teacher workshops, classroom-based interventions, and parenting classes designed to increase awareness of the importance of emotion and skillful emotion regulation. Interventions may target individuals at heightened risk of adverse outcomes, such as daughters of depressed mothers, children who live in abusive families, members of underrepresented minorities in work or academic contexts, or those with high temperamental levels of negative emotion. More specific interventions will target individuals who have clinical diagnoses (Barrett, Wilson-Mendenhall, & Barsalou, this volume; Campbell-Sills, Ellard, & Barlow, this volume; Joormann & Siemer, this volume; Kober, this volume). These are the inventions that come to mind most easily, and many of our pharmacological and psychosocial interventions for psychiatric disorders have an emotion regulation component, although much remains to be learned about exactly how each type of intervention influences particular aspects of emotion regulation (Berking & Schwarz, this volume; Neacsiu, Bohus, & Linehan, this volume; MacLeod & Grafton, this volume; Mennin & Fresco, this volume; Farb, Anderson, Irving, & Segal, this volume).

A second type of application involves making larger changes in the physical and social worlds in which we live. An example of this class of interventions comes from applying an emotion regulation perspective to seemingly intractable global conflicts (Halperin, 2013). These conflicts are characterized by high levels of negative emotions that powerfully shape attitudes and behaviors of each of the parties to the conflict. In particular, negative intergroup emotions—emotions that arise as a result of belonging to a certain group—can lead to the commencement and maintenance of hostilities, then block progress toward a peaceful solution to the ongoing conflict. To

assess the role of emotion regulation in one such conflict, namely, the ongoing Israeli–Palestinian conflict, a nationwide survey of Jewish-Israeli adults was conducted during the Gaza War between Israelis and Palestinians. This survey assessed both reappraisal use and attitudes toward providing humanitarian aid to Palestinian citizens. Findings indicated that Israelis who regulated their negative emotions during the war by using reappraisal were more supportive of providing humanitarian aid than Israelis who did not use reappraisal (Halperin & Gross, 2011). Building on this foundation, a second study randomized Israeli participants to either a reappraisal training condition or a control condition just before the Palestinian United Nations (UN) bid in 2011. Findings indicated that a week following the training, participants who had been trained to use reappraisal showed greater support for conciliatory policies and less support for aggressive policies toward Palestinians. These effects persisted when assessed 5 months after training, and at each time point, negative emotion mediated the effects of reappraisal on conflict-related attitudes (Halperin, Porat, Tamir, & Gross, 2012). These findings hint at the broader, real-world relevance of an emotion regulation perspective, and in future work it will be interesting to investigate how such a perspective might be applied in other arenas.

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References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Barrett, L. F., Wilson-Mendenhall, C. D., & Barsalou, L. W. (this volume, 2014). A psychological construction account of emotion dysregulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 447–465). New York: Guilford Press.

Barrett, L. F., Gross, J. J., Conner, T., & Benvenuto, M. (2001). Knowing what you're feeling and knowing what to do about it: Mapping the relation between emotion differentiation and emotion regulation. *Cognition and Emotion, 15*, 713–724.

Barrett, L. F., Ochsner, K. N., & Gross, J. J. (2007). On the automaticity of emotion. In J. Bargh (Ed.), *Social psychology and the unconscious: The automaticity of higher mental processes* (pp. 173–217). New York: Psychology Press.

Berking, M., & Schwarz, J. (this volume, 2014). Affect regulation training. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 529–547). New York: Guilford Press.

Bowlby, J. (1969). *Attachment and loss: Attachment*. New York: Basic Books.

Buck, R. (1990). Mood and emotion: A comparison of five contemporary views. *Psychological Inquiry, 1*, 330–336.

Buck, R. (1993). What is this thing called subjective experience?: Reflections on the neuropsychology of qualia. *Neuropsychology, 7*, 490–499.

Butler, E. A., Egloff, B., Wilhelm, F. W., Smith, N. C., Erickson, E. A., & Gross, J. J. (2003). The social consequences of expressive suppression. *Emotion, 3*, 48–67.

Butler, E. A., Lee, T. L., & Gross, J. J. (2007). Emotion regulation and culture: Are the social consequences of emotion suppression culture-specific? *Emotion, 7*, 30–48.

Campbell-Sills, L., Ellard, K. K., & Barlow, D. H. (this volume, 2014). Emotion regulation in anxiety disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 393–412). New York: Guilford Press.

Campos, J. J., Frankel, C. B., & Camras, L. (2004). On the nature of emotion regulation. *Child Development, 75*, 377–394.

Carstensen, L. L., Isaacowitz, D. M., & Charles, S. T. (1999). Taking time seriously. *American Psychologist, 54*, 165–181.

Charles, S. T., & Carstensen, L. L. (this volume, 2014). Emotion regulation and aging. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 203–218). New York: Guilford Press.

Coan, J. A., & Maresh, E. L. (this volume, 2014). Social baseline theory and the social regulation of emotion. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 221–236). New York: Guilford Press.

Cole, P., Martin, S., & Dennis, T. (2004). Emotion regulation as a scientific construct: Meth-

odological challenges and directions for child development research. *Child Development, 75*, 317–333.

Dan-Glauser, E. S., & Gross, J. J. (2013). Emotion regulation and emotion coherence: Evidence for strategy-specific effects. *Emotion*.

Davidson, R. J. (1994). On emotion, mood, and related affective constructs. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 51–55). New York: Oxford University Press.

Demaree, H. A., Schmeichel, B. J., Robinson, J. L., Pu, J., Everhart, D., & Berntson, G. G. (2006). Up- and down-regulating facial disgust: Affective, vagal, sympathetic, and respiratory consequences. *Biological Psychology, 71*, 90–99.

Drabant, E. M., McRae, K., Manuck, S. B., Hariri, A. R., & Gross, J. J. (2009). Individual differences in typical reappraisal use predict amygdala and prefrontal responses. *Biological Psychiatry, 65*, 367–373.

Ellsworth, P. C., & Scherer, K. R. (2003). Appraisal processes in emotion. In R. J. Davidson, K. R., Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 572–595). New York: Oxford University Press.

English, T., John, O. P., & Gross, J. J. (2013). Emotion regulation in close relationships. In J. A. Simpson & L. Campbell (Eds.), *The Oxford handbook of close relationships* (pp. 500–513). New York: Oxford University Press.

Farb, N. A. S., Anderson, A. K., & Segal, Z. V. (this volume, 2014). Mindfulness interventions and emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 548–568). New York: Guilford Press.

Feinberg, M., Willer, R., Antonenko, O., & John, O. P. (2012). Liberating reason from the passions. *Psychological Science, 23*, 788–795.

Freud, S. (1959). *Inhibitions, symptoms, anxiety* (A. Strachey, Trans.; J. Strachey, Ed.). New York: Norton. (Original work published 1926)

Frijda, N. H. (1986). *The emotions*. Cambridge, UK: Cambridge University Press.

Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The neural bases of emotion regulation: Reappraisal and suppression of negative emotion. *Biological Psychiatry, 63*, 577–586.

Goldin, P., Ziv, M., Jazaieri, H., Werner, K., Kraemer, H., Heimberg, R. G., et al. (2012). Cognitive reappraisal self-efficacy mediates the effects of individual cognitive-behavioral therapy for social anxiety disorder. *Journal of Consulting and Clinical Psychology, 80*, 1034–1040.

Gross, J. J. (1998a). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224–237.

Gross, J. J. (1998b). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*, 271–299.

Gross, J. J. (1999). Emotion regulation: Past, present, future. *Cognition and Emotion, 13*, 551–573.

Gross, J. J. (2010). The future's so bright, I gotta wear shades. *Emotion Review, 2*, 212–216.

Gross, J. J. (2013). Emotion regulation: Taking stock and moving forward. *Emotion, 13*, 359–365.

Gross, J. J. & Barrett, L. F. (2011). Emotion generation and emotion regulation: One or two depends on your point of view. *Emotion Review, 3*, 8–16.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*, 348–362.

Gross, J. J., & Levenson, R. W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64*, 970–986.

Gross, J. J., & Levenson, R. W. (1997). Hiding feelings: The acute effects of inhibiting positive and negative emotions. *Journal of Abnormal Psychology, 106*, 95–103.

Gross, J. J., & Munoz, R. F. (1995). Emotion regulation and mental health. *Clinical Psychology: Science and Practice, 2*, 151–164.

Gross, J. J., Richards, J. M., & John, O. P. (2006). Emotion regulation in everyday life. In D. K. Snyder, J. A. Simpson, & J. N. Hughes (Eds.), *Emotion regulation in couples and families: Pathways to dysfunction and health*. Washington, DC: American Psychological Association.

Gross, J. J., Sheppes, G., & Urry, H. L. (2011). Emotion generation and emotion regulation: A distinction we should make (carefully). *Cognition and Emotion, 25*, 765–781.

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York: Guilford Press.

Gyurak, A., & Etkin, A. (this volume, 2014). A neurobiological model of implicit and explicit

emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 76–90). New York: Guilford Press.

Gyurak, A., Gross, J. J., & Etkin, A. (2011). Explicit and implicit emotion regulation: A dual-process framework. *Cognition and Emotion, 25*, 400–412.

Halperin, E. (2013). Current and potential contributions of the study of emotion and emotion regulation to the study and practice of conflict resolution. *Emotion Review.*

Halperin, E., & Gross, J. J. (2011). Emotion regulation in violent conflict: Reappraisal, hope, and support for humanitarian aid to the opponent in war time. *Cognition and Emotion, 25*, 1228–1236.

Halperin, E., Porat, R., Tamir, M., & Gross, J. J. (2012). Can emotion regulation change political attitudes in intractable conflict?: From the laboratory to the field. *Psychological Science, 24*(1), 106–111.

Harris, C. R. (2001). Cardiovascular responses of embarrassment and effects of emotional suppression in a social setting. *Journal of Personality and Social Psychology, 81*, 886–897.

Hayes, J. P., Morey, R. A., Petty, C. M., Seth, S., Smoski, M. J., McCarthy, G., et al. (2011). Staying cool when things get hot: Emotion regulation modulates neural mechanisms of memory encoding. *Frontiers in Human Neuroscience, 4*, 1–10.

Hochschild, A. R. (1983). *The managed heart.* Berkeley: University of California Press.

Jamieson, J. P., Mendes, W. B., Blackstock, E., & Schmader, T. (2010). Turning the knots in your stomach into bows: Reappraising arousal improves performance on the GRE. *Journal of Experimental Social Psychology, 46*, 208–212.

John, O. P., & Eng, J. (this volume, 2014). Individual differences in emotion regulation: Conceptualization, measures, and findings. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 321–345). New York: Guilford Press.

Johns, M. J., Inzlicht, M., & Schmader, T. (2008). Stereotype threat and executive resource depletion: Examining the influence of emotion regulation. *Journal of Experimental Psychology: General, 137*, 691–705.

Joormann, J., & Siemer, M. (this volume, 2014). Emotion regulation in mood disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 413–427). New York: Guilford Press.

Kanske, P., Heissler, J., Schonfelder, S., Bongers, A., & Wessa, M. (2011). How to regulate emotion?: Neural networks for reappraisal and distraction. *Cerebral Cortex, 21*, 1379–1388.

Kaplan, H. I., & Sadock, B. J. (1991). *Synopsis of psychiatry* (6th ed.). Baltimore: Williams & Wilkins.

Kappas, A. (2011). Emotion and regulation are one! *Emotion Review, 3*, 17–25.

Kim, S. H., & Hamann, S. (2012). The effect of cognitive reappraisal on physiological reactivity and emotional memory. *International Journal of Psychophysiology, 83*, 348–56.

Kober, H. (this volume, 2014). Emotion regulation in substance use disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 428–446). New York: Guilford Press.

Koole, S. L. (2009). The psychology of emotion regulation: An integrative review. *Cognition and Emotion, 23*, 4–41.

Lang, P. J. (1995). The emotion probe: Studies of motivation and attention. *American Psychologist, 50*, 372–385.

Lang, P. J., & Bradley, M. M. (2010). Emotion and the motivational brain. *Biological Psychology, 84*, 437–450.

Larsen, R. J. (2000). Toward a science of mood regulation. *Psychological Inquiry, 11*, 129–141.

Larson, E. B., & Yao, X. (2005). Clinical empathy as emotional labor in the patient–physician relationship. *Journal of the American Medical Association, 293*, 1100–1106.

Lazarus, R. S. (1966). *Psychological stress and the coping process.* New York: McGraw-Hill.

Lazarus, R. S. (1991). *Emotion and adaptation.* Oxford, UK: Oxford University Press.

Lazarus, R. S. (1993). From psychological stress to the emotions: A history of changing outlooks. *Annual Review of Psychology, 44*, 1–21.

Levenson, R. W. (1999). The intrapersonal functions of emotion. *Cognition and Emotion, 13*, 481–504.

Levenson, R. W., Haase, C. M., Bloch, L., Holley, S. R., & Seider, H. (this volume, 2014). Emotion regulation in couples. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 267–283). New York: Guilford Press.

Lieberman, M. D., Inagaki, T. K., Tabibnia, G., & Crockett, M. J. (2011). Subjective responses to emotional stimuli during labeling, reappraisal, and distraction. *Emotion, 11*, 468–80.

Masters, J. C. (1991). Strategies and mechanisms for the personal and social control of emotion.

In J. Garber & K. A. Dodge (Eds.), *The development of emotion regulation and dysregulation* (pp. 182–207). Cambridge, UK: Cambridge University Press.

MacLean, P. D. (1990). *The triune brain in evolution: Role in paleocerebral functions*. New York: Plenum.

MacLeod, C., & Grafton, B. (this volume, 2014). Regulation of emotion through modification of attention. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 508–528). New York: Guilford Press.

Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. H., & Gross, J. J. (2005). The tie that binds?: Coherence among emotion experience, behavior, and physiology. *Emotion, 5*, 175–190.

Mauss, I. B., & Tamir, M. (this volume, 2014). Emotion goals: How their content, structure, and operation shape emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 361–375). New York: Guilford Press.

Mauss, I. B., Shallcross, A. J., Troy, A. S., John, O. P., Ferrer, E., Wilhelm, F. H., et al. (2011). Don't hide your happiness!: Positive emotion dissociation, social connectedness, and psychological functioning. *Journal of Personality and Social Psychology, 100*, 738–748.

Mennin, D. S., & Fresco, D. M. (this volume, 2014). Emotion regulation therapy. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 469–490). New York: Guilford Press.

Mesquita, B., De Leersnyder, J., & Albert, D. (this volume, 2014). The cultural regulation of emotions. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 284–302). New York: Guilford Press.

Mischel, W. (1996). From good intentions to willpower. In P. Gollwitzer & J. Bargh (Eds.), *The psychology of action* (pp. 197–218). New York: Guilford Press.

Moore, S. A., Zoellner, L. A., & Mollenholt, N. (2008). Are expressive suppression and cognitive reappraisal associated with stress-related symptoms? *Behaviour Research and Therapy, 46*, 993–1000.

Neacsiu, A. D., Bohus, M., & Linehan, M. (this volume, 2014). Dialectical behavior therapy: An intervention for emotion dysregulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 491–507). New York: Guilford Press.

Nezlek, J. B., & Kuppens, P. (2008). Regulating positive and negative emotions in daily life. *Journal of Personality, 76*, 561–579.

Ochsner, K. N., & Gross, J. J. (2008). Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. *Current Directions in Psychological Science, 17*, 153–158.

Ochsner, K. N., & Gross, J. J. (this volume, 2014). The neural bases of emotion and emotion regulation: A valuation perspective. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 23–42). New York: Guilford Press.

Ochsner, K. N., Ray, R. R., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D. E., et al. (2004). For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *NeuroImage, 23*, 483–499.

Ochsner, K. N., Ray, R. R., Hughes, B., McRae, K., Cooper, J. C., Weber, J., et al. (2009). Bottom-up and top-down processes in emotion generation: Common and distinct neural mechanisms. *Psychological Science, 20*, 1322–1331.

Opitz, P., Gross, J. J., & Urry, H. L. (2012). Selection, optimization, and compensation in the domain of emotion regulation: Applications to adolescence, older age, and major depressive disorder. *Social and Personality Psychology Compass, 6*, 142–155.

Parkinson, B., Totterdell, P., Briner, R. B., & Reynolds, S. (1996). *Changing moods: The psychology of mood and mood regulation*. London: Longman.

Parrott, W. G. (1993). Beyond hedonism: Motives for inhibiting good moods and for maintaining bad moods. In D. M. Wegner & J. W. Pennebaker (Eds.), *Handbook of mental control* (pp. 278–305). Englewood Cliffs, NJ: Prentice Hall.

Quoidbach, J., Berry, E. V., Hansenne, M., & Mikolajczak, M. (2010). Positive emotion regulation and well-being: Comparing the impact of eight savoring and dampening strategies. *Personality and Individual Differences, 49*, 368–373.

Ray, R. D., McRae, K., Ochsner, K. N., & Gross, J. J. (2010). Cognitive reappraisal of negative affect: Converging evidence from EMG and self-report. *Emotion, 10*, 587–592.

Richards, J. M., Butler, E., & Gross, J. J. (2003). Emotion regulation in romantic relationships:

The cognitive consequences of concealing feelings. *Journal of Personal and Social Relationships, 20*, 599–620.

Richards, J. M., & Gross, J. J. (1999). Composure at any cost?: The cognitive consequences of emotion suppression. *Personality and Social Psychology Bulletin, 25*, 1033–1044.

Richards, J. M., & Gross, J. J. (2000). Emotion regulation and memory: The cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology, 79*, 410–424.

Richards, J. M., & Gross, J. J. (2006). Personality and emotional memory: How regulating emotion impairs memory for emotional events. *Journal of Research in Personality, 40*, 631–651.

Rothbart, M. K., Ziaie, H., & O'Boyle, C. G. (1992). Self-regulation and emotion in infancy. In N. Eisenberg & R. A. Fabes (Eds.), *Emotion and its regulation in early development* (pp. 7–23). San Francisco: Jossey-Bass.

Salovey, P., & Mayer, J. D. (1990). Emotional intelligence. *Imagination, Cognition and Personality, 9*, 185–211.

Samson, A., & Gross, J. J. (2012). Humor as emotion regulation: The differential consequences of negative versus positive humor. *Cognition and Emotion, 26*, 375–384.

Samson, A. C., Huber, O., & Gross, J. J. (2012). Emotion regulation in Asperger's syndrome and high functioning autism. *Emotion, 12*(4), 659–665.

Scherer, K. R. (1984). On the nature and function of emotion: A component process approach. In K. R. Scherer & P. E. Ekman (Eds.), *Approaches to emotion* (pp. 293–317). Hillsdale, NJ: Erlbaum.

Scherer, K. R., Schorr, A., & Johnstone, T. (2001). *Appraisal processes in emotion: Theory, methods, research*. New York: Oxford University Press.

Sheppes, G. (this volume, 2014). Emotion regulation choice: Theory and findings. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 126–139). New York: Guilford Press.

Sheppes, G., Catran, E., & Meiran, N. (2009). Reappraisal (but not distraction) is going to make you sweat: Physiological evidence for self-control effort. *International Journal of Psychophysiology, 71*, 91–96.

Sheppes, G., Scheibe, S., Suri, G., & Gross, J. J. (2011). Emotion regulation choice. *Psychological Science, 22*, 1391–1396.

Shiota, M. N., & Levenson, R. W. (2012). Turn down the volume or change the channel?: Emotional effects of detached versus positive reappraisal. *Journal of Personality and Social Psychology, 103*(3), 416–429.

Siemer, M. (2001). Mood-specific effects on appraisal and emotion judgements. *Cognition and Emotion, 15*, 453–485.

Soto, J. A., Perez, C. R., Kim, Y.-H., Lee, E. A., & Minnick, M. R. (2011). Is expressive suppression always associated with poorer psychological functioning?: A cross-cultural comparison between European Americans and Hong Kong Chinese. *Emotion, 11*, 1450–1455.

Srivastava, S., Tamir, M., McGonigal, K. M., John, O. P., & Gross, J. J. (2009). The social costs of emotional suppression: A prospective study of the transition to college. *Journal of Personality and Social Psychology, 96*, 883–897.

Staudinger, M. R., Erk, S., Abler, B., & Walter, H. (2009). Cognitive reappraisal modulates expected value and prediction error encoding in the ventral striatum. *NeuroImage, 47*, 713–721.

Stemmler, G. (1997). Selective activation of traits: Boundary conditions for the activation of anger. *Personality and Individual Differences, 22*, 213–233.

Stepper, S., & Strack, F. (1993). Proprioceptive determinants of emotional and nonemotional feelings. *Journal of Personality and Social Psychology, 64*, 211–220.

Strack, F., Martin, L. L., & Stepper, S. (1988). Inhibiting and facilitating conditions of the human smile: A nonobtrusive test of the facial feedback hypothesis. *Journal of Personality and Social Psychology, 54*, 768–777.

Sutton, R. E. (2004). Emotional regulation goals and strategies of teachers. *Social Psychology of Education, 7*, 379–398.

Sutton, R. I. (1991). Maintaining norms about expressed emotions: The case of bill collectors. *Administrative Science Quarterly, 36*, 245–268.

Szasz, P. L., Szentagotai, A., & Hofmann, S. G. (2011). The effect of emotion regulation strategies on anger. *Behaviour Research and Therapy, 49*, 114–119.

Szczurek, L., Monin, B., & Gross, J. J. (2012). The *stranger* effect: The rejection of affective deviants. *Psychological Science, 23*(10), 1105–1111.

Tamir, M. (2009). What do people want to feel and why?: Pleasure and utility in emotion regulation. *Current Directions in Psychological Science, 18*, 101–105.

Tamir, M. (2011). The maturing field of emotion regulation. *Emotion Review, 3*, 3–7.

Tamir, M., John, O. P., Srivastava, S., & Gross, J. J. (2007). Implicit theories of emotion: Affective and social outcomes across a major life transition. *Journal of Personality and Social Psychology, 92*, 731–744.

Taylor, G. J. (1994). The alexithymia construct: Conceptualization, validation, and relationship with basic dimensions of personality. *New Trends in Experimental and Clinical Psychiatry, 10*, 61–74.

Thiruchselvam, R., Hajcak, G., & Gross, J. J. (2012). Looking inwards: Shifting attention within working memory representations alters emotional responses. *Psychological Science, 23*(12), 1461–1466.

Thompson, R. A. (1990). Emotion and self-regulation. In *Socioemotional development: Nebraska Symposium on Motivation* (Vol. 36, pp. 367–467). Lincoln: University of Nebraska Press.

Thompson, R. (2011). Emotion and emotion regulation: Two sides of the developing coin. *Emotion Review, 3*, 53–61.

Tsai, J. L. (2007). Ideal affect: Cultural causes and behavioral consequences. *Perspectives on Psychological Science, 2*, 242–259.

Webb, T. L., Miles, E., & Sheeran, P. (2012). Dealing with feeling: A meta-analysis of the effectiveness of strategies derived from the process model of emotion regulation. *Psychological Bulletin, 138*(4), 775–808.

Westen, D. (1994). Toward an integrative model of affect regulation: Applications to social-psychological research. *Journal of Personality, 62*, 641–667.

Wolgast, M., Lundh, L.-G., & Viborg, G. (2011). Cognitive reappraisal and acceptance: An experimental comparison of two emotion regulation strategies. *Behaviour Research and Therapy, 49*, 858–866.

Zajonc, R. B. (1984). On the primacy of affect. *American Psychologist, 39*, 117–123.

PART II

BIOLOGICAL BASES

CHAPTER 2

The Neural Bases of Emotion and Emotion Regulation: A Valuation Perspective

Kevin N. Ochsner
James J. Gross

The observation that emotions can be powerful forces for good and for ill has motivated researchers' efforts to understand how emotions arise and how they are regulated (Gross, 2007; Kalisch, 2009; Ochsner & Gross, 2005, 2008; Phillips, Ladouceur, & Drevets, 2008; Quirk & Beer, 2006). In particular, neuroscience research recently has made great strides in describing the neural systems that give rise to emotional responses and that permit their regulation. At the same time, parallel progress has been made in delineating the neural bases of related abilities, including affective learning, affective decision making, and expectancies, beliefs, and placebo effects (Cunningham & Zelazo, 2007; Hartley & Phelps, 2010; Murray, O'Doherty, & Schoenbaum, 2007; Pessoa, 2008; Rangel, Camerer, & Montague, 2008). It is becoming evident that the neural systems implicated across these various literatures—including those concerned with emotion and emotion regulation—are strikingly similar. This suggests that any account of the neural bases of emotion and its regulation—or related abilities—should be informed by these similarities. Such an integrated framework would be both more robust and more translatable to multiple basic and clinical contexts. Our goal in this chapter is to provide such a framework.

Our starting point is the assertion that *goal-directed (motivated) behavior* may be defined as behavior that aims to decrease the probability of states of either our bodies or of the world that have negative value for us (e.g., putting on a sweater when we are cold; picking up trash in the park), or increase the probability of states that have positive value for us (e.g., opening a can of soup to eat when we are hungry; arranging to have coffee with a friend).

The determination of value occurs dynamically at many levels of the brain, at different time scales, and with respect to many features in the environment (Leventhal, 1984; Rangel et al., 2008; Scherer, 2001). A sudden loss of blood pressure may occasion a valuation, as may the smell of dinner being prepared, an aggressive driver cutting one off, or a new way of thinking about a poem. If valuations are assessments of what is bad for me (negative value) or good for me (positive value), computed for many different objects, then different types of valuation might be expected to give rise to different types of responses, and indeed, they do (Ortony, Clore, & Collins, 1988;

Scherer, Schorr, & Johnstone, 2001). One particularly important type of valuation is *emotion*, which arises when a situation is evaluated as relevant to an individual's goals, thereby triggering a loosely coordinated set of experiential, behavioral, and peripheral physiological responses (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). Emotional responses such as joy, anger, and disgust all have at their core an evaluation of whether something is good for me or bad for me.

Given the major role that emotions play in shaping how we feel about and respond to the world around us, it is no surprise that emotions themselves can become the target of valuation. For example, we may feel angry at a child's impolite behavior, and judge our anger as either inappropriate (having negative value) or appropriate (having positive value), depending on the age of the child. In this and in similar cases, we assign negative or positive value to the valuation process itself, thereby energizing processes that tend to make the emotion in question either less or more likely to occur, depending on whether the valuation is negative or positive. When individuals influence their emotions in this way, they are engaging in *emotion regulation*.

Our framework therefore holds that emotions arise via the valuation of internal or external stimuli, and that emotion regulation arises via the valuation of the emotion itself. From this perspective, emotion and emotion regulation both have valuation at their core.

This three-part chapter uses a valuation perspective to integrate diverse findings in current neuroscience research regarding emotion, emotion regulation, affective learning, decision making, and expectancy. In the first part, we propose a multilevel framework that analyzes emotion and emotion regulation in terms of valuation. In the second part we use this framework to understand current research on emotion and its regulation. Finally, in the third part we explore the general utility of this framework by giving examples of how it can be extended to account for current research on related phenomena, including affective learning, decision making, and expectancy effects.

The Functional Architecture of Valuation

According to the framework we propose here, valuation can be schematized as the three-stage processing cycle outlined in Figure 2.1A. As detailed below, a *perception stage* takes various kinds of stimuli as inputs; a *valuation stage* dynamically appraises the value of these stimuli given current goals, context, and prior experience with similar stimuli; and an *action stage* comprises valuation-appropriate responses ranging from covert adjustments of low-level sensory (e.g., increased pupil dilation) or higher-level cognitive processes (e.g., shifts in effortful attention) to overt adjustments of a wide range of response systems (e.g., facial behavior, postural adjustments, sympathetic nervous system activation). This perception–valuation–action (PVA) sequence repeats as the new state of the world, resulting from the action, becomes the input for the next PVA sequence, thus setting in motion a new PVA cycle. Because multiple PVA cycles are typically running at any given time, these cycles interact, and it is these processing dynamics that give rise to behavior.

PVA Components: The Perception Stage

A PVA sequence is initiated by an external or internal stimulus that can vary in complexity from low-level perceptual features (like eye whites or low spatial frequencies) or physiological responses (e.g., a racing heart) to organized perceptual exemplars (e.g., objects or scenes) to abstract constructs such as the self. In this initial *perception* stage of the sequence, sensory systems (e.g., thalamus plus primary and secondary sensory cortices) encode these types of sensory inputs and pass them along to systems for computing value (Kravitz, Saleem, Baker, & Mishkin, 2011).

PVA Components: The Valuation Stage

Valuations are subserved by an overlapping set of interacting brain systems that compute the badness or goodness of perceptual inputs (Hamann, Ely, Hoffman, & Kilts, 2002; Ochsner & Barrett, 2001; Rangel et

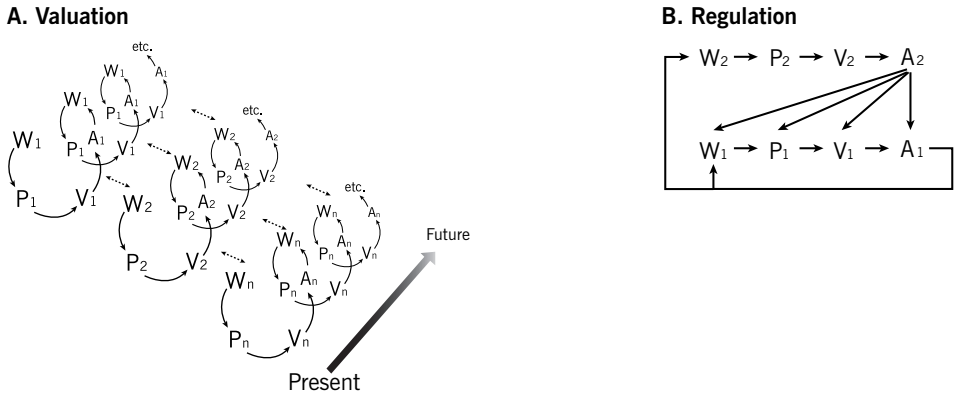


FIGURE 2.1A. The perception–valuation–action (PVA) processing cycle that comprises the fundamental building block of emotion and other types of valuation. As described in the text, multiple PVA cycles can operate at once, here represented by subscripts 1 to *n*. Each cycle involves individual PVA sequences that iteratively feed into one another across time (shown here progressively spiraling into the future), thereby comprising a PVA cycle. For each cycle, some set of internal and external stimuli comprising an initial state of the world (W) is represented perceptually (P), values are placed upon the stimuli (V), and associated action links (A) that are activated result in a new state of the world that feeds into the next iteration of the PVA processing cycle. Note that here we place emphasis on the three-stage (P-V-A) processing cycle, and the world (W) is considered to be the result of a prior action and the input for the next PVA sequence. PVAs can interact with one another, exciting or inhibiting each other's activation (schematically shown here by double-headed arrows between PVAs). *Emotions* are specific types of PVA sequences that involve specific types of perceptions, valuations, and actions. The neural systems supporting valuation are shown in Figure 2.2A and elaborated in the text and in Figure 2.3.

FIGURE 2.1B. How PVA sequences instantiate regulation. To illustrate how PVA sequences enable regulation, we zoom in on two PVA sequences, PVA_1 and PVA_2. As shown here, *emotion regulation* (or value regulation more generally) is a functional relationship between two PVA sequences in which one (PVA_1) is "generating emotion" and the other (PVA_2) is taking the first PVA as its "P," valuing it (negatively or positively), and targeting that first PVA for change via its "A." The "A" of PVA_2 enacts an emotion regulation strategy that influences one or more steps of PVA_1. As described in the text, five types of regulatory strategies may be distinguished. Each regulatory strategy depends on different combinations of cognitive control systems (see text, Figure 2.2B, and Figure 2.3) whose regulatory effects can be understood in terms of the stage of the PVA sequence that is targeted for change. On this view, *emotion regulation* is a type of valuation in which the valuation process is itself the target of valuation.

al., 2008; Rolls, 1999), thereby providing a common currency for comparing various objects and events (Levy & Glimcher, 2011). In this chapter we use *valuation* as an umbrella term to connote the same kinds of underlying processes that emotion theorists would describe using the term *appraisal* and attitude researchers would describe using the term *evaluation*. Targets of valuation range from primary reinforcers—objects that are innately seen as "bad" or "good," such as a sweet drink, to secondary reinforcers—objects that derive their negative or positive value from their association with primary reinforcers, such as an A+ written at the top of one's term paper (Rangel et al., 2008; Rolls, 1999). Figure 2.2A shows the brain regions associated with valuation processes.

Multiple valuations are computed for a given stimulus, and these vary along a continuum of representational complexity, with more complex valuations typically taking

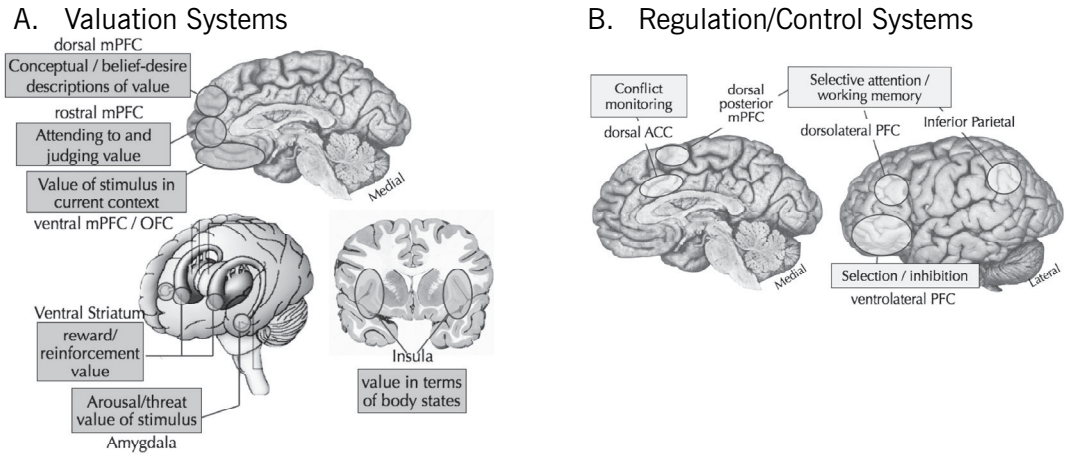


FIGURE 2.2A. Neural systems supporting valuation. Medial (top), lateral (bottom left), and coronal (bottom right) views of the brain showing systems implicated in different types of valuation processes that can be arrayed along a continuum from *core level valuations* (amygdala, ventral striatum) that consist of links between stimuli and reinforcers, to *contextual level valuations* (vmPFC/OFC) that place these S-R links in their historical, social, and motivational context, to *conceptual level valuations* that represent the value of stimuli in belief–desire terms (rostral and dorsal medial PFC) that may be verbalizable and consciously reportable. The insula, implicated in the representation of body states, may play a role in representing the body states associated with all three types of valuation. See text and Figure 2.3 for examples of how these systems play roles in specific types of valuation and emotion.

FIGURE 2.2B. Neural systems supporting regulation. Medial (left) and lateral (right) views of the brain showing systems implicated in regulatory strategies that can be used to regulate valuation in general, and emotion in particular Regions include: the dorsal anterior cingulate cortex (dACC), implicated in monitoring conflicts between desired and actual actions, posterior and dlPFC and inferior parietal cortex, implicated in holding control strategies and goals in mind and directing attention to relevant perceptual inputs, and vlPFC, implicated in selecting context-appropriate responses and inhibiting context-inappropriate responses. See text and Figure 2.3 for examples of how these systems play roles in specific types of emotion regulation and related phenomena.

longer to compute than less complex valuations (Leventhal, 1984; Scherer, 2001).

At the lowest level of this continuum, *core valuations* are made. These represent relatively direct associations between percepts and basic physiological and behavioral responses at the action stage (e.g., snake → fear response). Core valuation involves primarily subcortical and brainstem systems implicated in affective learning and responding. While many of these systems, including the ventral striatum, amygdala, and periaqueductal gray (PAG), receive inputs from a variety of sensory inputs (Keay & Bandler, 2001; Packard, 2009), and as such can be involved in the valuation of a variety of stimuli, there is evidence that core valuations for pain sensations involve dedicated pathways from the thalamus to nociceptive regions of the midcingulate cortex and anterior insula (Willis & Westlund, 1997). In addition, while the ventral striatum and amygdala are typically linked with positive/appetitive and negative/aversive valuations, respectively, both human and animal studies suggest that subregions of each structure may play roles in both kinds of valuations (Delgado, Jou, LeDoux, & Phelps, 2009; Holland & Gallagher, 2004; Wager, Barrett, et al., 2008). The associations underlying core valuations can be (but are not always) activated automatically and

without conscious intent, and are implicit insofar as they are not directly accessible to awareness, although one can be aware of the actions they trigger, and thereby become aware of them indirectly. Core valuations typically are linked to stereotyped action impulses (Kober et al., 2008; LeDoux, 2000; Rolls, 1999; Russell & Barrett, 1999), and as such, can provide the basis for stimulus–response (S-R) links of the sort that underlie Pavlovian conditioning and other basic forms of affective responding that involve pleasure and pain (Rangel et al., 2008).

At an intermediate level, *contextual valuations* evaluate inputs that represent combinations of S-R links and at least three types of contextual information: the historical as well as current social and motivational contexts of the person (for an illustrative example, see the section "Emotion as a Type of PVA Sequence"). This computational step involves at least three regions. The first comprises the orbitofrontal cortex (OFC) and ventromedial prefrontal cortex (vmPFC) (Ongur, Ferry, & Price, 2003; Price, 1999), whose inputs include the output of both the core valuation level and the medial temporal lobe (MTL) and the cortical associative memory systems, which provide temporal and spatial context (Davachi, 2006; Murray et al., 2007). Second is the superior temporal sulcus/temporoparietal junction (STS/TPJ), which itself is a multisensory zone that integrates expectancies with feedback, and reorients attention accordingly, including when expectations must be adjusted about the beliefs, actions, and intentions of others (Saxe, 2006; Young, Camprodon, Hauser, Pascual-Leone, & Saxe, 2010). Third is the anterior insula (AI), which integrates and makes available to awareness information about current body states, especially as they pertain to one's current affective state (Craig, 2003; Harrison, Gray, Gianaros, & Critchley, 2010; Kurth, Zilles, Fox, Laird, & Eickhoff, 2010; Zaki, Davis, & Ochsner, 2012). Contextual valuations indicate whether an object is good or bad in the present context, and therefore whether it should be sought or avoided at the present time. One commonly studied form of contextual valuation is fear extinction, in which an organism learns that a stimulus that previously predicted an aversive outcome (and was therefore negatively valued) no longer does so (and in the present temporal context, can be valued less negatively; Quirk & Beer, 2006). More generally, contextual valuations play key roles in other forms of affective learning, in determining whether the value of stimuli change across contexts, and in subjective awareness of one's affective states (Craig, 2009; Cunningham, Raye, & Johnson, 2004; Holland & Gallagher, 2004; Lieberman, Jarcho, & Satpute, 2004; Rangel et al., 2008; Schoenbaum, Saddoris, & Stalnaker, 2007). Contextual valuations influence behavior either by activating action impulses themselves or—as detailed below in the section on emotion regulation—by influencing which core valuations are expressed via actions (Ochsner, Ray, et al., 2009).

At the highest level of this continuum, *conceptual valuations* represent appraisals of stimuli that are abstract and often verbalizable. By this we mean representations of evaluations and affective states that abstracted across exemplars and contexts and are accessible to awareness in the form of "belief–desire" language. For example, a conceptual valuation of a snake may involve activation of a conceptual representation of "fear," which one can verbalize using that word.

We propose that this level involves at least four regions. First, the rostromedial (rmPFC) and dorsomedial (dmPFC) prefrontal regions implicated in attending to and explicitly judging the value of stimuli and use of categories and belief–desire language to elaborate semantically the affective value of a wide range of stimuli, from simple objects to the self (Cato et al., 2004; Lindquist & Barrett, 2008; Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012; Mitchell, 2009; Olsson & Ochsner, 2008; Zysset, Huber, Ferstl, & von Cramon, 2002). An unresolved question about medial PFC (mPFC) is whether different subregions are involved in making judgments (whether evaluative or not) about others, the self, and/or stimuli in general (Denny, Kober, Wager, & Ochsner, 2012; Ferstl & von Cramon, 2002; Zysset, Huber, Samson, Ferstl, & von Cramon, 2003). For our present purposes, we consider mPFC to be critical for using conceptual information to elaborate the affective meaning of stimuli, whether the stimulus triggering the valuation and emotion is the self, another person, or some other object/

event/situation. A third region involved in conceptual valuation is the ventrolateral prefrontal cortex (vlPFC), which helps select desired and inhibit undesired value representations (Aron, Robbins, & Poldrack, 2004; Badre & Wagner, 2007; Barrett, 2006; Gallagher & Frith, 2003; Lieberman et al., 2007; Lindquist & Barrett, 2008; Mitchell, 2009; Olsson & Ochsner, 2008; Thompson-Schill, Bedny, & Goldberg, 2005). Finally, regions of the anterior insula that support introspective awareness of body states also may be integral to awareness of body states and use of conceptual knowledge about them to make judgments about one's current affective states (Craig, 2009; Damasio, Damasio, & Tranel, 2013; Gray et al., 2012; Harrison et al., 2010; Zaki et al., 2012). Conceptual valuations influence behavior either by activating action impulses themselves or—as detailed below in the section on regulation—influencing which contextual and core valuations are expressed via actions (Ochsner, Ray, et al., 2009). We propose that conceptual valuations play key roles in introspection about and self-reports of affective states, in mental state attribution, and in judgments about the values of stimuli and actions that involve conscious reasoning about their value (Kalisch, Wiech, Critchley, & Dolan, 2006; Mitchell, 2009; Olsson & Ochsner, 2008).

PVA Components: The Action Stage

At any given level of valuation, the action impulses associated with a PVA sequence can be either *mental* (e.g., retrieving information from memory, forming a mental image, or introspecting about one's mood) or *physical* (e.g., including overt behaviors such as shifts of gaze or starting to run, and autonomic/physiological responses such as heart rate increases or the release of stress hormones; Levenson, 1999). Although elaborating the brain systems supporting the action stage is not the focus of this chapter, it likely involves subcortical and cortical regions involved in selecting motor actions, as well as initiating autonomic responses (e.g., the PAG, primary motor and supplementary motor areas, cingulate motor regions, and insula) (Buhle et al., 2012; Critchley, 2005; Dum, Levinthal, & Strick, 2009; Mobbs et al., 2009).

PVA Operating Principles: Processing Dynamics

As multiple valuations are computed at different levels and time scales—each with its own associated action impulses—only a subset of the possible actions associated with a percept and its valuations can be enacted. What determines which actions are expressed, whether mental (e.g., thoughts and feelings) or physical (e.g., smiling and hugging)?

One factor is the existing structure of the PVA sequences an individual possesses at any given moment in time. This factor has been addressed primarily in psychological and computational models of associative memory networks that suggest the P's, V's, and A's of all currently activated PVAs mutually excite and/or inhibit one another in such a way that the most activated action tendency or (a set of equally activated) tendencies "win" and are manifested as mental and/or physical actions (Desimone & Duncan, 1995; Barrett, Ochsner, & Gross, 2007; Maas, 2000; Miller & Cohen, 2001). The schematic PVA sequences of Figure 2.1A illustrate the possible kinds of links that may exist between P, V, and A nodes.

A second factor is the multiple contextual and historical considerations that determine the level of activation for each PVA sequence and whether they have inhibitory or excitatory links with other PVAs—including the stimuli that are (or have been) present as inputs, their activation history (which determines the strength of links within and across PVAs, and hence their relative ease of activation [Anderson, 1983; Neely, 1991]), and whether they are in the focus of attention (which enhances activity, especially at the perception stage [Pessoa, Kastner, & Ungerleider, 2003; Polk, Drake, Jonides, Smith, & Smith, 2008]).

At any given moment, an individual's affective response comprises the profile of activation across all PVAs—at all levels—that may combine or cancel one another depending on the nature of their connections and levels of activation (Barrett, Mesquita, Ochsner, & Gross, 2007; Scherer, 2001). As described below, depending on the circumstances, core, contextual, and/or conceptual PVAs may be activated most strongly and lead to action.

PVA Operating Principles: Interacting Networks

As our process-level description makes clear, the PVA sequence is continually unfolding in real time for multiple stimuli as multiple levels of analysis. This means that networks of interacting brain systems underlie each stage of the PVA sequence, as well as the interactions among stages. This follows from the fact that an individual's affective response comprises the profile of activation across all PVAs, at all levels, which in turn follows from the idea that the P, V, and A stages all involve multiple brain systems working together to compute the perceptual, evaluative, and action components of one's response to a given stimulus.

Thus, the totality of one's valuation of a stimulus cannot be understood in terms of the activation of a single brain system. That said, most of what we know about the functions of brain systems implicated in the P, V, and A stages comes from studies employing analytic techniques (e.g., simple contrasts) designed to isolate the contributions to behavior of single regions rather than integrated networks. Increasingly, however, various kinds of connectivity, network, and multivoxel pattern analyses are being used to describe the task-varying functional relationships among regions that define them as critical for aspects of the P, V, and A stages (Kober et al., 2008). For example, we and others have used mediation and structural equation modeling to describe the ways in which prefrontal control regions regulate emotional response via their impact on subcortical regions that trigger affective responses (Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007; Kober et al., 2010; Urry et al., 2006; Wager, Barrett, et al., 2008; for review, see Ochsner, Silvers, & Buhle, 2012). As such analytic techniques mature, we expect that our framework will be able to describe more precisely the functional interactions governing the P, V, and A stages, as well as their interactions.

A Valuation Perspective on Emotion and Emotion Regulation

Emotions are particular types of valuation that (1) have a well-specified object (i.e., one is angry about something), (2) unfold over seconds to minutes, and (3) involve coordinated changes in subjective experience, behavior, and physiology (Barrett et al., 2007; Mauss et al., 2005; Scherer et al., 2001). In keeping with our overall goal of showing how the valuation framework is broadly applicable, in the sections that follow we employ an expansive view of emotion.

Emotion as a Type of PVA Sequence

Imagine you are a commuter in a crowded New York subway car. Across from you sit a sleepy-eyed old man, a muscular teen, and an attractive woman. As the subway rattles toward your stop, the teen removes a knife from his pocket, shifting it from hand to hand.

In our framework, emotional reactions to the knife-wielding teen may be conceived of as specific kinds of PVA sequences derived from particular perceptions, valuations, and associated action impulses (Ortony et al., 1988; Scherer et al., 2001). Thus, an initial response may reflect a core-level valuation of the teen and his knife as potentially threatening by the amygdala and related regions, which triggers corresponding action impulses that mobilize you to avoid harm (e.g., increased heart rate, behavioral readiness to fight or flee; LeDoux, 2000; Phelps, 2006). At the contextual level, the action outputs of core-level PVA sequences become perceptual inputs that are integrated with other inputs representing the *historical* (episodic) context of, for example, having prior subway conversations with the teen (via MTL inputs), the *social* context of multiple other passengers being present (via STS/TPJ inputs), and the *motivational* context of current stress and bodily complaints (via AI and other subcortical inputs). As time passes, activation of the contextual-level PVAs, which dictate other courses of action, can begin to build, and the initial valuation may evolve dynamically into valuations of the teen as relatively innocuous or highly dangerous, depending on whether the teen previously indicated he has a role as a thug in a school play or is on medication for a delusional disorder (historical context), whether he elicits calm or anxious reactions from

other passengers (social context), or whether you are stressed from work or just had a great day (motivational context). Then, the action outputs of activated contextual-level PVAs are taken as inputs to systems (rmPFC, and/or vlPFC) that compute a valuation of the teen in belief–desire terms that can—at the action stage—be introspectively accessed or reported to others as the thoughts and feelings you attribute to yourself or others, including, for example, the thoughts that you yourself are brave, that the knife-wielder looks aggressive, and that the old man and young woman seem calm.

The order in which these valuation systems is activated, and their interplay, is not fixed and depends on the circumstances of your encounter with a stimulus. For example, if you are sitting on the subway, and the teen enters from the opposite end of the car and does not pose an immediate threat, then conceptual valuation systems might evaluate his intentions ("Is he dangerous?") and your own level of fear ("I'm not scared—yet"). As the teen moves closer, contextual systems might be most active as you evaluate the goodness or badness of potential courses of action based on your changing motivational state (increasing anxiety), history (the seat next to you was just vacated, and the teen moves toward this open seat) and the apparent anxiety of your fellow passengers (who look increasingly afraid). Finally, as the teen moves even closer and the threat level is very high, activation in core valuation systems may escalate to promote defensive actions such as freezing, escape or fighting (Mobbs et al., 2007).

The key idea is that, taken together, all of these PVAs, however they were activated, and each with their associated mental and physical action tendencies, comprise an emotional response.

Emotion Regulation as a Type of PVA Sequence

As noted earlier, emotions themselves are sometimes the target of valuation. For example, in the previous subway example, we might wish to protect our view of ourselves as brave and, as a consequence, desire to decrease our fear responses. To do this, we can take as objects of valuation the action outputs of PVAs that comprise a fear response. When we do this—thereby activating a goal to influence the nature of the emotional response—we are engaging in emotion regulation. As described below, this involves interactions among regions implicated in cognitive control (i.e., regulation) and/or valuation.

In our framework, emotion regulation is initiated when a PVA cycle that gives rise to emotion becomes the object of valuation (see Figure 2.1B). We propose that this typically happens across levels of valuation, as a higher-level PVA places a good or bad valuation on a lower-level PVA (although it also can happen between PVAs at a single level). It also can happen if there is a high level of conflict between active PVAs, such as whether the impulse to flee a potentially dangerous situation conflicts with the impulse to freeze, and a clear set of emotional responses isn't activated. We propose that when this happens, the level of conflict constitutes an input to the next PVA cycle, and evaluation of that conflict triggers an appropriate course of regulatory action.

One key feature of our framework is the idea that some of the prefrontal systems that support emotion regulation are involved in the control of nonaffective forms of behavior as well (Miller & Cohen, 2001; Ochsner & Gross, 2005). These systems (see Figure 2.2B) include dorsal and ventrolateral prefrontal regions that support selective attention, working memory, and retrieval from semantic memory; cingulate regions that monitor conflicts between competing As and the need for continued control; medial regions that support mental state attribution; and ventromedial prefrontal regions that place contextual constraints on the expression of core-level PVAs (Miller, 2000; Ochsner & Gross, 2005; Olsson & Ochsner, 2008; Wager, Jonides, & Reading, 2004; Wager & Smith, 2003). As detailed below, the regulatory actions supported by these systems comprise different types of "A's" in PVA sequences that place a value on one's current affective state.

Distinguishing among Emotion Regulation Processes

We have previously argued that emotion regulation processes can be differentiated into five families according to which stage

of the emotion generation sequence they target. In the context of the present framework, this idea is expressed by suggesting that emotion regulatory processes differ in the stage of the PVA sequence at which they have their primary impact (see Figure 2.3). Some strategies influence the situation-dependent perceptual inputs (*situation selection, situation modification*, and *attention deployment*). Others influence the valuation step itself (*cognitive change*). Still others influence the response output associated with activated action sequences (*response modulation*). By impacting different states of the PVA cycle, different strategies impact emotional responding in different ways, as detailed below.

Situation selection refers to altering the inputs to the PVA sequence through decisions about whether to expose oneself to a given situation/stimulus based on its projected affective impact. For example, calling to mind the image of the subway might lead to a negative evaluation, and a feeling of fear. This feeling might motivate a higher-level PVA that would trigger a decision to take an alternative means of transportation in order to decrease the probability of the negative experiences that one associates with taking the subway. More generally, situation selection can take many forms, for example, when a socially anxious individual avoids a social event. To date, the neural bases of situation selection have been studied only with avoidance conditioning tasks in which an animal learns to select an action (e.g., running in a wheel when a light is illuminated predicts an upcoming shock) that enables it to avoid experiencing a noxious stimulus (that prevents shock administration). Rodent studies have shown this involves modulation of two core valuation systems, the striatum and amygdala (Everitt et al., 1999; LeDoux & Gorman, 2001), and one human imaging study (Delgado et al., 2009) indicates that it also engages vlPFC and dorsolateral prefrontal cortex (dlPFC) regions involved in cognitive control that presumably modulate the core valuation systems.

Situation modification refers to altering the situation one is in, thereby modifying inputs to the PVA sequence, and changing the emotion (e.g., sitting further away from the teen or exiting the subway). We would expect that prefrontal systems should be involved in the selection of escape behaviors—especially in the kinds of emotionally arousing situations humans face in everyday life. Although this hypothesis has not been tested, the involvement of prefrontal regions is suggested by behavioral studies in humans showing that emotion can be regulated by deliberately changing situation-dependent stimulus inputs in the service of explicit regulatory goals. For example, either physically or mentally, using visual imagery, one can move closer to or further away from an emotion-eliciting stimulus (e.g., making oneself feel more positive by approaching a pleasant stimulus or less negative by withdrawing from an unpleasant one; Davis, Gross, & Ochsner, 2011; Muhlberger, Neumann, Wieser, & Pauli, 2008; Williams & Bargh, 2008).

Attentional deployment refers to altering the inputs to the PVA sequence by increasing or decreasing attention to them (e.g., looking away from the teen and at the man or woman). While this can gate specific stimuli wholly into or out of the PVA stream, thereby promoting or preventing responses to them, we propose that more graded changes in attention to stimuli may result in correspondingly graded levels of activation of their associated PVA sequences. This strategy involves interactions between cognitive control systems and valuation systems, with particular involvement of dorsal PFC and inferior parietal regions associated with selective attention (Pessoa et al., 2003), and in some cases rmPFC regions implicated in attending to and explicitly judging the value of stimuli (Bishop, 2007; Egner, Etkin, Gale, & Hirsch, 2008; Etkin, Egner, Peraza, Kandel, & Hirsch, 2006; Lane et al., 1998; Ochsner, Hughes, Robertson, Cooper, & Gabrieli, 2009). A growing but somewhat inconsistent imaging literature shows that, by and large, when a task manipulation diminishes attention to an affectively arousing stimulus, activation increases in PFC regions implicated in cognitive control (suggesting that cognitive control systems are involved in manipulating attention), and activity decreases in regions implicated in core (e.g., amygdala, PAG), contextual (e.g., insula) or conceptual (e.g., mPFC) valuation (e.g., Ochsner & Gross, 2005; Pessoa, 2009). While it is clear that the specific valuation systems modulated by attentional deploy-

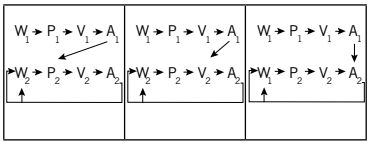
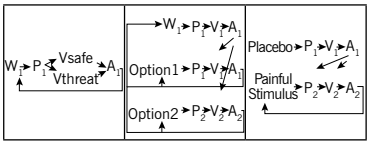
Neural Systems			Emotion Regulation Strategies			Related Phenomena		
Region	Type of System	Processes	Attentional Deployment	Cognitive Change (Reappraisal)	Response Modulation (e.g., Expressive Suppression)	Affective Learning (e.g., Fear Extinction)	Affective Decisions (e.g., Intertemporal Choice)	Expectancies (e.g., Placebo Effects)
dorsolateral PFC	Control	selective attention/ working memory	⇧	⇧	⇧		⇧	⇧
dorsal posterior mPFC			⇧	⇧	⇧			
inferior parietal			⇧	⇧				⇧
dorsal ACC		performance monitoring	⇧	⇧	⇧	⇧		⇩
ventrolateral PFC		selection/ inhibition	⇧	⇧	⇧		⇧	⇧
dorsal mPFC	Conceptual Valuation	conceptual/ categorical valuations		⇧				⇧
rostral mPFC	↑	attention to valuation	⇧	⇩				⇩
ventromedial PFC/OFC	Contextual Valuation	value of stimulus in current context	?	?		⇧	⇧⇩	⇧
ventral striatum	↓	reward/ reinforcement value		⇧⇩				⇧
amygdala	Core Valuation	arousal (and threat) value of stimulus	⇩	⇧⇩	⇧	⇩		⇩
insula	Valuation	representation and awareness of body states for all types of valuation		⇧⇩	⇧	⇧	⇧⇩	⇩
PVA Cycle Illustrations								

FIGURE 2.3. Neural systems for valuation and control postulated by the valuation framework presented in the chapter (left-hand columns), as well as the roles these neural systems play in three kinds of emotion regulation strategies (center columns, see text) and three kinds of related phenomena (right-hand columns, see text). Up arrows indicate increased activation, down arrows indicate decreased activation, and "?" indicates involvement in some (but not the majority) of the studies. The final row diagrams, in PVA terms, how each emotion regulation strategy or related phenomenon might operate (see text for details). The three center columns show, for each emotion regulation strategy, how control actions impact either attention paid to particular stimuli at the perception stage (attentional deployment), how one values those stimuli (reappraisal), or what actions one takes as a consequence of this valuation (response modulation). The three right-hand columns show for related phenomena how initial valuations (e.g., threat) may be overridden if one learns new valuations (e.g., safe) for a stimulus (extinction) one may select among choice options as a function of their relative valuations, with control actions coming into play when the choice options are similarly valued and/or in conflict (intertemporal choice), or a placebo may influence the valuation of a painful stimulus via the action of control processes (placebo effects).

ment depend on the sensory qualities of the stimulus, distraction from pain modulates nociceptive regions of insula and cingulate cortex (Frankenstein, Richter, McIntyre, & Remy, 2001; Tracey et al., 2002), whereas distraction from an aversive image modulates the amygdala (McRae et al., 2010; Pessoa, 2009); for example, cross-study variability in attentional deployment strategies and the lack of a common metric for determining how much any given strategy diminishes attention to a stimulus in one study compared to others (see Ochsner & Gross, 2005, for a detailed review) have limited the conclusions that can be drawn about when and how specific cognitive control systems are involved.

Cognitive change refers to altering the subjective meaning and/or perceived self-relevance of the present situation (e.g., thinking of the knife as a stage prop or that knife-tossing is an innocent way of passing time). The framework suggests that this strategy should involve interactions between cognitive control systems that can be used deliberately to change one's interpretation of a stimulus and valuation systems that trigger an affective response. Of note here is the fact that the framework predicts that conceptual valuation systems can play a role on either side of this regulatory equation: on the one hand, being the target of cognitive control systems that seek to change one's high-level conceptual valuation of a stimulus, and on the other, assisting those cognitive control systems in reformulating the attributions one makes about the nature of one's own beliefs, desires, and feelings (e.g., "I'm feeling less afraid now")—or those expressed by others (e.g., "The subway passengers are anxious about the crowding, not the teen")—as one deliberately changes his or her interpretation of an emotion-eliciting stimulus. Research on cognitive change—referred to in the literature as "reappraisal"—consistently supports the predictions of the framework: When engaging in a cognitive change strategy, activation is observed in lPFC and cingulate PFC regions associated with cognitive control, as well as mPFC regions associated with conceptual valuation (albeit primarily when up-regulating emotional responses) and at the same time increasing or decreasing activity in core (e.g., amygdala, striatum) and/or contextual (e.g., insula) valuation systems (e.g., Kober et al., 2010; Ochsner et al., 2004; Urry et al., 2006; Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008; reviewed in Kalisch, 2009; Ochsner & Gross, 2005, 2008) in accordance with one's regulatory goals.

Finally, *response modulation* refers to targeting behavioral manifestations of emotion (e.g., playing it cool by not showing fear of the knife-wielding teen). Human research primarily has focused on one exemplar of this strategy, *expressive suppression*, which involves hiding behavioral manifestations of emotion (Gross, 1998). Behaviorally, expressive suppression effectively reduces facial expressions of emotion, but the effort and attention required to do so trigger autonomic responses, impair memory for visual cues, and can negatively impact social interactions (Butler et al., 2003; Gross, 1998; Richards & Gross, 2000). In keeping with these findings, an initial imaging study showed that suppressing the expression of disgust activated two key PFC regions associated with cognitive control (dlPFC associated with maintaining goals, and vlPFC associated with response selection and inhibition more generally; Aron et al., 2004; Badre & Wagner, 2007; Thompson-Schill et al., 2005), and *increased* activation in core (amgydala) and contextual (insula) valuation regions associated with detection of threats and awareness of body states (Goldin, McRae, Ramel, & Gross, 2008). This supports the idea that expressive suppression, like other strategies, depends on interactions between cognitive control and valuation regions, and may have neural bases similar to those supporting response inhibition more generally (Aron et al., 2004).

Applications of the Valuation Perspective

The neural systems implicated in emotion and emotion regulation play key roles in other phenomena that involve valuation and cognitive control (Hartley & Phelps, 2010; Murray et al., 2007; Pessoa, 2008; Phillips et al., 2008; Rangel et al., 2008). We believe it is important that any account of emotion and emotion regulation use terminology and concepts that are broadly applicable to allied phenomena as well.

With this in mind, we illustrate in this section the broad applicability of our valuation perspective on emotion and emotion regulation by showing how it can provide a framework for describing the mechanisms underlying three types of related phenomena that traditionally are considered in relatively separate literatures. This has the dual benefits of broadening the framework to account for aspects of related phenomena it was not initially formulated to address, and in so doing, making the framework more robust and generally applicable.

Affective or Emotional Learning

As noted in the earlier section on the PVA processing dynamics, our valuation perspective allows learning to occur by updating the valuations placed on stimuli with each iteration of the PVA sequence. To account more broadly for various forms of affective or emotional learning, we can elaborate the way in which this updating process occurs.

When encountering a stimulus, one's current valuation of it sets an expectation for the outcome states of the world that should follow from execution of the associated action impulse(s). These outcomes become inputs to the next PVA, which evaluates discrepancies between the expected and actual outcomes. If this valuation is negative (i.e., when the discrepancy is large and/or important in light of currently active goals), this valuation triggers learning and updating processes that change links between a stimulus and its valuation (P-V) or between a valuation and an action (V-A)—or between separate PVA sequences—so that future valuations are more accurate (Delgado, Olsson, & Phelps, 2006; Rangel et al., 2008; Schultz, Dayan, & Montague, 1997). Each change is small, so that one's value expectations for a stimulus at a given moment in time are a function of one's prior experiences with it, biased more heavily toward recent experiences. While this value updating process typically is studied in the context of conditioning, reward, and affective learning, it fits neatly within our valuation framework as the way that changes in the contingency between actions and outcomes can adaptively alter the valuations that drive the actions.

To illustrate this, we can use our subway example to consider one of the most studied examples of value updating, namely, extinction of a fear response. We discussed extinction earlier as an example of contextual valuation in which an organism learns that a previously feared stimulus need no longer be feared in the current temporal context. In that section, however, we did not explain how the organism learns this contextual association. Here we propose that value updating is the learning mechanism.

The subway example can help make this concrete. Recall that the knife-wielding teen initially elicits a threat valuation and fear response involving amygdala-mediated core level PVAs. If the expected outcome does not transpire (i.e., the teen takes no harmful actions), however, then over time a new contextual-level PVA is acquired by ventromedial/orbitomedial PFC systems indicating the teen is not a threat. The longer the teen takes no harmful action, the stronger this PVA becomes. Ultimately, even though the knife still connotes threat at the core level, the contextual PVA wins out for expression in behavior (see the section on PVA processing dynamics). Because the core-level PVA itself remains unchanged, a fear response can be quickly reinstated in the future should the teen become truly threatening (Bouton, 2004; LeDoux, 1993). The framework can be similarly applied to other cases in which one learns, or already has learned, that a given emotional impulse is inappropriate or unnecessary in the current context, as in reversal of learned appetitive or aversive associations (Bouton, 2004; Corcoran & Quirk, 2007; Schoenbaum et al., 2007).

As this example makes clear, affective learning and the types of regulatory strategies reviewed earlier are not mutually exclusive and may in many contexts work together. For example, the act of reappraising can be seen as a way of cognitively creating a discrepancy between an expected internal outcome (e.g., a fear response) associated with a given percept (e.g., the knife-wielding teen) and the response that actually occurs (e.g., calmness). This discrepancy could activate learning processes that weaken core-level PVA representations of the teen as threatening, build new contextual-level representations of the teen as nonthreatening, and strengthen conceptual-level PVAs of the teen as an actor. In this way, reappraisal—and by extension other regulatory strategies—

can be seen as providing top-down "teaching" inputs to outcome-driven regulatory processes that typically are triggered by external cues (cf. Delgado, Gillis, & Phelps, 2008; Delgado, Nearing, LeDoux, & Phelps, 2008).

Affective Decision Making

Our valuation perspective also may be applied to affect-laden decision making. Affective decisions require a choice between options that are associated with different expected rewards or punishments. In our framework, these expectations are reflected in the values computed for choice options at various levels of the valuation hierarchy. To the extent that the play of activation and inhibition across PVA sequences associated with choice options results in a core-, contextual-, or conceptual-level valuation determining the behavioral output, then the choice option associated with that valuation will be selected. However, in some cases, this play of activation fails to determine clearly a most highly valued selection, and cognitive control processes may be engaged in order to construct, hold in mind, and implement top-down processes that influence PVAs associated with choice options. This commonly happens when choice options are similarly valued and/or conflict with one another, but it also may happen when the valuation process itself becomes a target of valuation (e.g., when there is a negative valuation of an attractive response option).

To illustrate (see Figure 2.3), consider how the framework accounts for a commonly studied choice dilemma in behavioral economics and neuroeconomics known as "intertemporal choice" (or as delay of gratification in the developmental literature; Mischel, Shoda, & Rodriguez, 1989). This dilemma involves choosing between a smaller reward available now or a larger reward available at some point in the future. In our framework, selection of the immediate reward would be promoted by core-level (striatal) or contextual-level (medial/orbital frontal) valuation systems that represent the reward value of the currently available stimulus. By contrast, picking the delayed reward would require the use of lateral prefrontal cognitive control systems in order to maintain a representation of the delayed reward in working memory and inhibit activation of PVAs for the immediately available choice option (Figner et al., 2011). In keeping with this account, human imaging studies have shown greater ventral striatal (VS) and/or vmPFC versus greater dlPFC activity when participants select immediate versus delayed rewards (McClure, Ericson, Laibson, Loewenstein, & Cohen, 2007; McClure, Laibson, Loewenstein, & Cohen, 2004), and a recent transcranial magnetic stimulation study showed that disruption of left—but not right—dlPFC led participants to "impulsively" select immediate rewards when they had shown a prior preference for the delayed reward (Figner et al., 2011). Strikingly, this result dovetails with the finding that a pathway from left dlPFC to the VS supports the use of reappraisal to diminish craving for desired substances (e.g., sugary/fattening foods) when participants think about the negative long term (e.g., diabetes) as opposed to the immediate (e.g., delicious taste) consequences of consuming them (Kober et al., 2010).

As these findings make clear, affective decision making and the regulatory strategies reviewed earlier may depend upon very similar neural systems and, as such, the line between them is not always clear. Indeed, intertemporal choices—and other choices that require selecting between options consistent with long- versus short-term goals—can be viewed as self-control tasks (Figner et al., 2011; Hare, Camerer, & Rangel, 2009; Wunderlich, Rangel, & O'Doherty, 2009) in which the decision to select an option consistent with a long-term goal is influenced by attention deployment and cognitive change strategies (Mischel et al., 1989). Our valuation perspective can also be applied to other types of choice dilemmas in which control and valuation processes interact to determine choice, including risky decision making (Gianotti et al., 2009), in which the choice is to be fair toward or to punish others (Knoch et al., 2008; Knoch, Pascual-Leone, Meyer, Treyer, & Fehr, 2006), and when the act of choice itself changes our valuations of stimuli via the value-updating process (Sharot, De Martino, & Dolan, 2009; Sharot, Shiner, & Dolan, 2010) as in cognitive dissonance reduction (Lieberman, Ochsner, Gilbert, & Schacter, 2001; Sharot et al., 2009; van Veen, Krug, Schooler, & Carter, 2009).

Expectancies, Beliefs, and Placebo Effects

Our valuation framework also helps make sense of the growing imaging literatures on the ways in which expectancies and beliefs of various sorts—including placebo effects—influence responses to various kinds of affective stimuli (Wager, 2005). In these tasks, participants are given one of two kinds of explicit expectations. In studies of expectancies or anticipation, participants are told that an upcoming stimulus—whether a painful sensation, an image, or something else—will be of a particular intensity or kind. In placebo experiments, participants are told that a drug (e.g., a cream or a pill) will increase or decrease their subsequent responses to a stimulus. In either case, these expectations lead participants to experience the stimulus, when presented, as subjectively more similar to what they expected than would have been the case had they held no expectations or beliefs about its nature or the protective properties of a drug.

From the perspective of our framework, these phenomena all involve the top-down influence of cognitive control systems on valuation systems or the influence of higher level valuation systems on lower level valuation systems. Our interpretation of these effects is consistent with results of imaging studies of expectancies and placebo effects on pain responses. Such studies indicate that expectancies and placebo beliefs about pain are maintained in a combination of lateral prefrontal/parietal working memory systems and/or medial prefrontal systems (Atlas, Bolger, Lindquist, & Wager, 2010; Lieberman, Jarcho, Berman, et al., 2004; Wager, 2005; Wager, Atlas, Leotti, & Rilling, 2011) that in the framework could be described as representing either conceptual-level beliefs (e.g., "The cream on my forearm should lessen the pain") or contextual-level expectations about the stimulus or placebo. According to our framework, these systems influence attention to and appraisal of the value of stimuli in contextual-level and/or core-level valuation systems (see Figure 2.3), modifying their levels of activation to be consistent with top-down beliefs (e.g., lessening activation of pain-sensitive valuations systems, including contextual-level regions (e.g., cingulate and insular cortex) and core-level regions (e.g., amygdala and PAG) (Ploghaus, Becerra, Borras, & Borsook, 2003; Wager, 2005).

Thus, from the viewpoint of the framework, expectancies and beliefs operate much like two of the emotion regulation strategies described earlier—attention deployment and cognitive change—in that they alter lower-level inputs to PVA systems and/or the valuation process.

Summary

One of the fundamental challenges faced by any animal is computing and expressing the value of stimuli in an accurate and timely manner. This is difficult, because the animal's internal state and external environment change over time, and its information acquisition, processing, and response resources and capabilities are limited. To address these challenges, humans (and other animals) have developed a complex set of interacting valuation systems, each of which can be described in terms of a simplified P-V-A sequence, in which a particular perceptual input is valued (negatively or positively to a given degree), leading to an impulse to alter ongoing behavioral or cognitive responses. These P-V-A sequences run in parallel at various levels in the brain and compete for expression.

This process-oriented valuation framework suggests a number of directions for future research. One direction concerns the valuation systems. While Figures 2.2A and 2.3 feature three kinds of valuation systems (core, contextual, and conceptual), future work should clarify the number and kind of valuation systems, as well as the rules that govern their engagement in particular contexts. A second direction has to do with how the often-competing action impulses associated with different P-V-A sequences are coordinated. We have emphasized the role of competitive activation and inhibition, but how this and other processes lead to coordinated and sustained adaptive behavior rather than erratic and conflicting behavior is not yet clear. A third direction concerns the inputs and outputs of valuation systems. We have suggested that the class of P-V-A sequences whose inputs are other P-V-A sequences, and outputs that include the engagement of cognitive control processes are fundamental to emotion regula-

tion and self-control more generally. That said, the range of relevant inputs and outputs, and the malleability of input–output relations requires further study. A fourth direction concerns the efficacy of various forms of value regulation and how they are intermixed in everyday life. Which "pure" or "hybrid" forms of value regulation are most effective? A fifth direction concerns translation of what we learn to illuminate individual differences. In our framework, a given emotional response and regulation profile could involve individual differences in (1) the initial valuations placed on specific classes of stimuli by systems at the core, contextual, and/or conceptual levels; (2) the speed with which these valuations are made; (3) how quickly and easily one resolves conflicts between them to express emotional responses; (4) how quickly and effectively learning processes update these valuations given that some emotional responses may be more difficult to change than others; (5) the knowledge of how and when to deploy emotion regulatory strategies; and (6) the capacity and ability to deploy top-down control systems to implement these strategies. One important direction for future research is examining how each of these differences—and others—may interact to produce various forms of psychopathology.

Our goal in presenting this valuation framework is to provide a common platform for analyzing the neural systems that are important for many different types of valuation. The impetus for this framework came from the observation that neural systems implicated in emotion generation and emotion regulation overlapped in important ways with neural systems implicated in other literatures that typically are not considered side by side (see Figure 2.3). Across all these research domains an organism's adaptive capacity crucially hinges on the coordination of multiple valuation systems in real time, and a key challenge for future research is delineating these PVA interactions, ideally with adequate specificity to permit more explicitly computational approaches. We believe that an explicitly integrative valuation framework represents a step in this direction, and holds out the possibility of better coordinating hitherto unconnected research literatures, while simultaneously deepening our understanding of each one.

References

Anderson, J. R. (1983). A spreading activation theory of memory. *Journal of Verbal Learning and Verbal Behavior, 22*(3), 261–295.

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in Cognitive Sciences, 8*(4), 170–177.

Atlas, L. Y., Bolger, N., Lindquist, M. A., & Wager, T. D. (2010). Brain mediators of predictive cue effects on perceived pain. *Journal of Neuroscience, 30*(39), 12964–12977.

Badre, D., & Wagner, A. D. (2007). Left ventrolateral prefrontal cortex and the cognitive control of memory. *Neuropsychologia, 45*(13), 2883–2901.

Barrett, L. F. (2006). Solving the emotion paradox: Categorization and the experience of emotion. *Personality and Social Psychology Review, 10*(1), 20–46.

Barrett, L. F., Mesquita, B., Ochsner, K. N., & Gross, J. J. (2007). The experience of emotion. *Annual Review of Psychology, 58*, 373–403.

Barrett, L. F., Ochsner, K. N., & Gross, J. J. (2007). Automaticity and emotion. In J. A. Bargh (Ed.), *Social Psychology and the Unconscious* (pp. 173–218). New York: Psychology Press.

Bishop, S. J. (2007). Neurocognitive mechanisms of anxiety: An integrative account. *Trends in Cognitive Sciences, 11*(7), 307–316.

Bouton, M. E. (2004). Context and behavioral processes in extinction. *Learning and Memory, 11*(5), 485–494.

Buhle, J. T., Kober, H., Ochsner, K. N., Mende-Siedlecki, P., Weber, J., Hughes, B. L., et al. (2012). Common representation of pain and negative emotion in the midbrain periaqueductal gray. *Social Cognitive and Affective Neuroscience*. [E-publication ahead of print]

Butler, E. A., Egloff, B., Wilhelm, F. H., Smith, N. C., Erickson, E. A., & Gross, J. J. (2003). The social consequences of expressive suppression. *Emotion, 3*(1), 48–67.

Cato, M. A., Crosson, B., Gokcay, D., Soltysik, D., Wierenga, C., Gopinath, K., et al. (2004). Processing words with emotional connotation: An fMRI study of time course and laterality in rostral frontal and retrosplenial cortices. *Journal of Cognitive Neuroscience, 16*(2), 167–177.

Corcoran, K. A., & Quirk, G. J. (2007). Recalling safety: Cooperative functions of the ventromedial prefrontal cortex and the hippo-

campus in extinction. *CNS Spectrums, 12*(3), 200–206.

Craig, A. D. (2003). Interoception: The sense of the physiological condition of the body. *Current Opinion in Neurobiology, 13*(4), 500–505.

Craig, A. D. (2009). How do you feel—now?: The anterior insula and human awareness. *Nature Reviews Neuroscience, 10*(1), 59–70.

Critchley, H. D. (2005). Neural mechanisms of autonomic, affective, and cognitive integration. *Journal of Comparative Neurology, 493*(1), 154–166.

Cunningham, W. A., Raye, C. L., & Johnson, M. K. (2004). Implicit and explicit evaluation: fMRI correlates of valence, emotional intensity, and control in the processing of attitudes. *Journal of Cognitive Neuroscience, 16*(10), 1717–1729.

Cunningham, W. A., & Zelazo, P. D. (2007). Attitudes and evaluations: A social cognitive neuroscience perspective. *Trends in Cognitive Sciences, 11*(3), 97–104.

Damasio, A., Damasio, H., & Tranel, D. (2013). Persistence of feelings and sentience after bilateral damage of the insula. *Cerebral Cortex, 23*(4), 833–846.

Davachi, L. (2006). Item, context and relational episodic encoding in humans. *Current Opinion in Neurobiology, 16*(6), 693–700.

Davis, J. I., Gross, J. J., & Ochsner, K. N. (2011). Psychological distance and emotional experience: What you see is what you get. *Emotion, 11*(2), 438–444.

Delgado, M. R., Gillis, M. M., & Phelps, E. A. (2008). Regulating the expectation of reward via cognitive strategies. *Nature Neuroscience, 11*(8), 880–881.

Delgado, M. R., Jou, R. L., LeDoux, J. E., & Phelps, E. A. (2009). Avoiding negative outcomes: Tracking the mechanisms of avoidance learning in humans during fear conditioning. *Frontiers in Behavioral Neuroscience, 3*, 33.

Delgado, M. R., Nearing, K. I., Ledoux, J. E., & Phelps, E. A. (2008). Neural circuitry underlying the regulation of conditioned fear and its relation to extinction. *Neuron, 59*(5), 829–838.

Delgado, M. R., Olsson, A., & Phelps, E. A. (2006). Extending animal models of fear conditioning to humans. *Biological Psychology, 73*(1), 39–48.

Denny, B. T., Kober, H., Wager, T. D., & Ochsner, K. N. (2012). A meta-analysis of functional neuroimaging studies of self- and other judgments reveals a spatial gradient for mentalizing in medial prefrontal cortex. *Journal of Cognitive Neuroscience, 24*(8), 1742–1752.

Desimone, R., & Duncan, J. (1995). Neural mechanisms of selective visual attention. *Annual Review of Neuroscience, 18*, 193–222.

Dum, R. P., Levinthal, D. J., & Strick, P. L. (2009). The spinothalamic system targets motor and sensory areas in the cerebral cortex of monkeys. *Journal of Neuroscience, 29*(45), 14223–14235.

Egner, T., Etkin, A., Gale, S., & Hirsch, J. (2008). Dissociable neural systems resolve conflict from emotional versus nonemotional distracters. *Cerebral Cortex, 18*(6), 1475–1484.

Etkin, A., Egner, T., Peraza, D. M., Kandel, E. R., & Hirsch, J. (2006). Resolving emotional conflict: A role for the rostral anterior cingulate cortex in modulating activity in the amygdala. *Neuron, 51*(6), 871–882.

Everitt, B. J., Parkinson, J. A., Olmstead, M. C., Arroyo, M., Robledo, P., & Robbins, T. W. (1999). Associative processes in addiction and reward: The role of amygdala–ventral striatal subsystems. *Annals of the New York Academy of Sciences, 877*, 412–438.

Ferstl, E. C., & von Cramon, D. Y. (2002). What does the frontomedian cortex contribute to language processing: coherence or theory of mind? *NeuroImage, 17*(3), 1599–1612.

Figner, B., Knoch, D., Johnson, E. J., Krosch, A. R., Lisanby, S. H., Fehr, E., et al. (2011). Lateral prefrontal cortex and self-control in intertemporal choice. *Nature Neuroscience, 13*(5), 538–539.

Frankenstein, U. N., Richter, W., McIntyre, M. C., & Remy, F. (2001). Distraction modulates anterior cingulate gyrus activations during the cold pressor test. *NeuroImage, 14*(4), 827–836.

Gallagher, H. L., & Frith, C. D. (2003). Functional imaging of "theory of mind." *Trends in Cognitive Sciences, 7*(2), 77–83.

Gianotti, L. R., Knoch, D., Faber, P. L., Lehmann, D., Pascual-Marqui, R. D., Diezi, C., et al. (2009). Tonic activity level in the right prefrontal cortex predicts individuals' risk taking. *Psychological Science, 20*(1), 33–38.

Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The neural bases of emotion regulation: Reappraisal and suppression of negative emotion. *Biological Psychiatry, 63*(6), 577–586.

Gray, M. A., Beacher, F. D., Minati, L., Nagai,

Y., Kemp, A. H., Harrison, N. A., et al. (2012). Emotional appraisal is influenced by cardiac afferent information. *Emotion, 12*(1), 180–191.

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*(1), 224–237.

Gross, J. J. (2007). *The handbook of emotion regulation*. New York: Guilford Press.

Hamann, S. B., Ely, T. D., Hoffman, J. M., & Kilts, C. D. (2002). Ecstasy and agony: Activation of the human amygdala in positive and negative emotion. *Psychological Science, 13*(2), 135–141.

Hare, T. A., Camerer, C. F., & Rangel, A. (2009). Self-control in decision-making involves modulation of the vmPFC valuation system. *Science, 324*(5927), 646–648.

Harrison, N. A., Gray, M. A., Gianaros, P. J., & Critchley, H. D. (2010). The embodiment of emotional feelings in the brain. *Journal of Neuroscience, 30*(38), 12878–12884.

Hartley, C. A., & Phelps, E. A. (2010). Changing fear: The neurocircuitry of emotion regulation. *Neuropsychopharmacology, 35*(1), 136–146.

Holland, P. C., & Gallagher, M. (2004). Amygdala–frontal interactions and reward expectancy. *Current Opinion in Neurobiology, 14*(2), 148–155.

Johnstone, T., van Reekum, C. M., Urry, H. L., Kalin, N. H., & Davidson, R. J. (2007). Failure to regulate: Counterproductive recruitment of top-down prefrontal–subcortical circuitry in major depression. *Journal of Neuroscience, 27*(33), 8877–8884.

Kalisch, R. (2009). The functional neuroanatomy of reappraisal: Time matters. *Neuroscience and Biobehavioral Reviews, 33*(8), 1215–1226.

Kalisch, R., Wiech, K., Critchley, H. D., & Dolan, R. J. (2006). Levels of appraisal: A medial prefrontal role in high-level appraisal of emotional material. *NeuroImage, 30*(4), 1458–1466.

Keay, K. A., & Bandler, R. (2001). Parallel circuits mediating distinct emotional coping reactions to different types of stress. *Neuroscience and Biobehavioral Reviews, 25*(7–8), 669–678.

Knoch, D., Nitsche, M. A., Fischbacher, U., Eisenegger, C., Pascual-Leone, A., & Fehr, E. (2008). Studying the neurobiology of social interaction with transcranial direct current stimulation—the example of punishing unfairness. *Cerebral Cortex, 18*(9), 1987–1990.

Knoch, D., Pascual-Leone, A., Meyer, K., Treyer, V., & Fehr, E. (2006). Diminishing reciprocal fairness by disrupting the right prefrontal cortex. *Science, 314*(5800), 829–832.

Kober, H., Barrett, L. F., Joseph, J., Bliss-Moreau, E., Lindquist, K., & Wager, T. D. (2008). Functional grouping and cortical–subcortical interactions in emotion: A meta-analysis of neuroimaging studies. *NeuroImage, 42*(2), 998–1031.

Kober, H., Mende-Siedlecki, P., Kross, E. F., Weber, J., Mischel, W., Hart, C. L., et al. (2010). Prefrontal–striatal pathway underlies cognitive regulation of craving. *Proceedings of the National Academy of Sciences USA, 107*(33), 14811–14816.

Kravitz, D. J., Saleem, K. S., Baker, C. I., & Mishkin, M. (2011). A new neural framework for visuospatial processing. *Nature Reviews Neuroscience, 12*(4), 217–230.

Kurth, F., Zilles, K., Fox, P. T., Laird, A. R., & Eickhoff, S. B. (2010). A link between the systems: Functional differentiation and integration within the human insula revealed by meta-analysis. *Brain Structure and Function, 214*(5–6), 519–534.

Lane, R. D., Reiman, E. M., Axelrod, B., Yun, L. S., Holmes, A., & Schwartz, G. E. (1998). Neural correlates of levels of emotional awareness: Evidence of an interaction between emotion and attention in the anterior cingulate cortex. *Journal of Cognitive Neuroscience, 10*(4), 525–535.

LeDoux, J. E. (1993). Emotional memory: In search of systems and synapses. *Annals of the New York Academy of Sciences, 702*, 149–157.

LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience, 23*, 155–184.

LeDoux, J. E., & Gorman, J. M. (2001). A call to action: Overcoming anxiety through active coping. *American Journal of Psychiatry, 158*(12), 1953–1955.

Levenson, R. W. (1999). The intrapersonal functions of emotion [Special issue]. *Cognition and Emotion, 13*(5), 481–504.

Leventhal, H. (1984). A perceptual–motor theory of emotion. *Advances in Experimental Social Psychology, 17*, 117–182.

Levy, D. J., & Glimcher, P. W. (2011). Comparing apples and oranges: Using reward-specific

and reward-general subjective value representation in the brain. *Journal of Neuroscience, 31*(41), 14693–14707.

Lieberman, M. D., Eisenberger, N. I., Crockett, M. J., Tom, S. M., Pfeifer, J. H., & Way, B. M. (2007). Putting feelings into words: Affect labeling disrupts amygdala activity in response to affective stimuli. *Psychological Science, 18*(5), 421–428.

Lieberman, M. D., Jarcho, J. M., Berman, S., Naliboff, B. D., Suyenobu, B. Y., Mandelkern, M., et al. (2004). The neural correlates of placebo effects: A disruption account. *NeuroImage, 22*(1), 447–455.

Lieberman, M. D., Jarcho, J. M., & Satpute, A. B. (2004). Evidence-based and intuition-based self-knowledge: An fMRI study. *Journal of Personality and Social Psychology, 87*(4), 421–435.

Lieberman, M. D., Ochsner, K. N., Gilbert, D. T., & Schacter, D. L. (2001). Do amnesics exhibit cognitive dissonance reduction?: The role of explicit memory and attention in attitude change. *Psychological Science, 12*(2), 135–140.

Lindquist, K. A., & Barrett, L. F. (2008). Constructing emotion: The experience of fear as a conceptual act. *Psychological Science, 19*(9), 898–903.

Lindquist, K. A., Wager, T. D., Kober, H., Bliss-Moreau, E., & Barrett, L. F. (2012). The brain basis of emotion: A meta-analytic review. *Behavioral and Brain Sciences, 35*(3), 121–143.

Maas, W. (2000). On the computational power of winner-take-all. *Neural Computation, 12*(11), 2519–2535.

Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. H., & Gross, J. J. (2005). The tie that binds?: Coherence among emotion experience, behavior, and physiology. *Emotion, 5*(2), 175–190.

McClure, S. M., Ericson, K. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. (2007). Time discounting for primary rewards. *Journal of Neuroscience, 27*(21), 5796–5804.

McClure, S. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. (2004). Separate neural systems value immediate and delayed monetary rewards. *Science, 306*(5695), 503–507.

McRae, K., Hughes, B., Chopra, S., Gabrieli, J. D., Gross, J. J., & Ochsner, K. N. (2010). The neural bases of distraction and reappraisal. *Journal of Cognitive Neuroscience, 22*(2), 248–262.

Miller, E. K. (2000). The prefrontal cortex and cognitive control. *Nature Reviews Neuroscience, 1*(1), 59–65.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*, 167–202.

Mischel, W., Shoda, Y., & Rodriguez, M. L. (1989). Delay of gratification in children. *Science, 244*(4907), 933–938.

Mitchell, J. P. (2009). Inferences about mental states. *Philosophical Transactions of the Royal Society of London B: Biological Sciences, 364*(1521), 1309–1316.

Mobbs, D., Hassabis, D., Seymour, B., Marchant, J. L., Weiskopf, N., Dolan, R. J., et al. (2009). Choking on the money: Reward-based performance decrements are associated with midbrain activity. *Psychological Science, 20*(8), 955–962.

Mobbs, D., Petrovic, P., Marchant, J. L., Hassabis, D., Weiskopf, N., Seymour, B., et al. (2007). When fear is near: Threat imminence elicits prefrontal–periaqueductal gray shifts in humans. *Science, 317*(5841), 1079–1083.

Muhlberger, A., Neumann, R., Wieser, M. J., & Pauli, P. (2008). The impact of changes in spatial distance on emotional responses. *Emotion, 8*(2), 192–198.

Murray, E. A., O'Doherty, J. P., & Schoenbaum, G. (2007). What we know and do not know about the functions of the orbitofrontal cortex after 20 years of cross-species studies. *Journal of Neuroscience, 27*(31), 8166–8169.

Neely, J. H. (1991). Semantic priming effects in visual word recognition: A selective review of current findings and theories. In D. Besner & G. Humphreys (Eds.), *Basic processes in reading: Visual word recognition* (pp. 264–336). Hillsdale, NJ: Erlbaum.

Ochsner, K. N., & Barrett, L. F. (2001). A multiprocess perspective on the neuroscience of emotion. In T. J. Mayne & G. A. Bonanno (Eds.), *Emotions: Currrent issues and future directions* (pp. 38–81). New York: Guilford Press.

Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9*(5), 242–249.

Ochsner, K. N., & Gross, J. J. (2008). Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. *Currents Directions in Psychological Science, 17*(1), 153–158.

Ochsner, K. N., Hughes, B., Robertson, E. R.,

Cooper, J. C., & Gabrieli, J. D. (2009). Neural systems supporting the control of affective and cognitive conflicts. *Journal of Cognitive Neuroscience, 21*(9), 1842–1855.

Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D. E., et al. (2004). For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *NeuroImage, 23*(2), 483–499.

Ochsner, K. N., Ray, R. R., Hughes, B., McRae, K., Cooper, J. C., Weber, J., et al. (2009). Bottom-up and top-down processes in emotion generation: Common and distinct neural mechanisms. *Psychological Science, 20*(11), 1322–1331.

Ochsner, K. N., Silvers, J. A., & Buhle, J. T. (2012). Functional imaging studies of emotion regulation: A synthetic review and evolving model of the cognitive control of emotion. *Annals of the New York Academy of Sciences, 1251*, E1–E24.

Olsson, A., & Ochsner, K. N. (2008). The role of social cognition in emotion. *Trends in Cognitive Sciences, 12*(2), 65–71.

Ongur, D., Ferry, A. T., & Price, J. L. (2003). Architectonic subdivision of the human orbital and medial prefrontal cortex. *Journal of Comparative Neurology, 460*(3), 425–449.

Ortony, A., Clore, G. L., & Collins, A. (1988). *The cognitive structure of emotions*. New York: Cambridge University Press.

Packard, M. G. (2009). Anxiety, cognition, and habit: A multiple memory systems perspective. *Brain Research, 1293*, 121–128.

Pessoa, L. (2008). On the relationship between emotion and cognition. *Nature Reviews Neuroscience, 9*(2), 148–158.

Pessoa, L. (2009). How do emotion and motivation direct executive control? *Trends in Cognitive Sciences, 13*(4), 160–166.

Pessoa, L., Kastner, S., & Ungerleider, L. G. (2003). Neuroimaging studies of attention: From modulation of sensory processing to top-down control. *Journal of Neuroscience, 23*(10), 3990–3998.

Phelps, E. A. (2006). Emotion and cognition: Insights from studies of the human amygdala. *Annual Review of Psychology, 57*, 27–53.

Phillips, M. L., Ladouceur, C. D., & Drevets, W. C. (2008). A neural model of voluntary and automatic emotion regulation: Implications for understanding the pathophysiology and neurodevelopment of bipolar disorder. *Molecular Psychiatry, 13*(9), 829, 833–857.

Ploghaus, A., Becerra, L., Borras, C., & Borsook, D. (2003). Neural circuitry underlying pain modulation: Expectation, hypnosis, placebo. *Trends in Cognitive Sciences, 7*(5), 197–200.

Polk, T. A., Drake, R. M., Jonides, J. J., Smith, M. R., & Smith, E. E. (2008). Attention enhances the neural processing of relevant features and suppresses the processing of irrelevant features in humans: A functional magnetic resonance imaging study of the Stroop task. *Journal of Neuroscience, 28*(51), 13786–13792.

Price, J. L. (1999). Prefrontal cortical networks related to visceral function and mood. *Annals of the New York Academy of Sciences, 877*, 383–396.

Quirk, G. J., & Beer, J. S. (2006). Prefrontal involvement in the regulation of emotion: Convergence of rat and human studies. *Curentr Opinion in Neurobiology, 16*(6), 723–727.

Rangel, A., Camerer, C., & Montague, P. R. (2008). A framework for studying the neurobiology of value-based decision making. *Nature Reviews Neuroscience, 9*(7), 545–556.

Richards, J. M., & Gross, J. J. (2000). Emotion regulation and memory: The cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology, 79*(3), 410–424.

Rolls, E. T. (1999). *The brain and emotion*. Oxford, UK: Oxford University Press.

Russell, J. A., & Barrett, L. F. (1999). Core affect, prototypical emotional episodes, and other things called emotion: Dissecting the elephant. *Journal of Personality and Social Psychology, 76*(5), 805–819.

Saxe, R. (2006). Uniquely human social cognition. *Current Opinion in Neurobiology, 16*(2), 235–239.

Scherer, K. R. (2001). Appraisal considered as a process of multilevel sequential checking. In K. R. Scherer & A. Schorr (Eds.), *Appraisal processes in emotion: Theory, methods, research* (pp. 92–120). New York: Oxford University Press.

Scherer, K. R., Schorr, A., & Johnstone, T. (Eds.). (2001). *Appraisal processes in emotion: Theory, methods, research*. New York: Oxford University Press.

Schoenbaum, G., Saddoris, M. P., & Stalnaker, T. A. (2007). Reconciling the roles of orbitofrontal cortex in reversal learning and the encoding of outcome expectancies. *Annals of the New York Academy of Sciences, 1121*, 320–335.

Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science, 275*(5306), 1593–1599.

Sharot, T., De Martino, B., & Dolan, R. J. (2009). How choice reveals and shapes expected hedonic outcome. *Journal of Neuroscience, 29*(12), 3760–3765.

Sharot, T., Shiner, T., & Dolan, R. J. (2010). Experience and choice shape expected aversive outcomes. *Journal of Neuroscience, 30*(27), 9209–9215.

Thompson-Schill, S. L., Bedny, M., & Goldberg, R. F. (2005). The frontal lobes and the regulation of mental activity. *Current Opinion in Neurobiology, 15*(2), 219–224.

Tracey, I., Ploghaus, A., Gati, J. S., Clare, S., Smith, S., Menon, R. S., et al. (2002). Imaging attentional modulation of pain in the periaqueductal gray in humans. *Journal of Neuroscience, 22*(7), 2748–2752.

Urry, H. L., van Reekum, C. M., Johnstone, T., Kalin, N. H., Thurow, M. E., Schaefer, H. S., et al. (2006). Amygdala and ventromedial prefrontal cortex are inversely coupled during regulation of negative affect and predict the diurnal pattern of cortisol secretion among older adults. *Journal of Neuroscience, 26*(16), 4415–4425.

van Veen, V., Krug, M. K., Schooler, J. W., & Carter, C. S. (2009). Neural activity predicts attitude change in cognitive dissonance. *Nature Neuroscience, 12*(11), 1469–1474.

Wager, T. D. (2005). The neural bases of placebo effects in pain. *Current Directions in Psychological Science, 14*(4), 175–179.

Wager, T. D., Atlas, L. Y., Leotti, L. A., & Rilling, J. K. (2011). Predicting individual differences in placebo analgesia: Contributions of brain activity during anticipation and pain experience. *Journal of Neuroscience, 31*(2), 439–452.

Wager, T. D., Barrett, L. F., Bliss-Moreau, E., Lindquist, K., Duncan, S., Kober, H., et al. (2008). The neuroimaging of emotion. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *The handbook of emotion* (3rd ed., pp. 249–271). New York: Guilford Press.

Wager, T. D., Davidson, M. L., Hughes, B. L., Lindquist, M. A., & Ochsner, K. N. (2008). Prefrontal–subcortical pathways mediating successful emotion regulation. *Neuron, 59*(6), 1037–1050.

Wager, T. D., Jonides, J., & Reading, S. (2004). Neuroimaging studies of shifting attention: A meta-analysis. *NeuroImage, 22*(4), 1679–1693.

Wager, T. D., & Smith, E. E. (2003). Neuroimaging studies of working memory: A meta-analysis. *Cognitive, Affective, and Behavioral Neuroscience, 3*(4), 255–274.

Williams, L. E., & Bargh, J. A. (2008). Keeping one's distance: The influence of spatial distance cues on affect and evaluation. *Psychological Science, 19*(3), 302–308.

Willis, W. D., & Westlund, K. N. (1997). Neuroanatomy of the pain system and of the pathways that modulate pain. *Journal of Clinical Neurophysiology, 14*(1), 2–31.

Wunderlich, K., Rangel, A., & O'Doherty, J. P. (2009). Neural computations underlying action-based decision making in the human brain. *Proceedings of the National Academy of Sciences USA, 106*(40), 17199–17204.

Young, L., Camprodon, J. A., Hauser, M., Pascual-Leone, A., & Saxe, R. (2010). Disruption of the right temporoparietal junction with transcranial magnetic stimulation reduces the role of beliefs in moral judgments. *Proceedings of the National Academy of Sciences USA, 107*(15), 6753–6758.

Zaki, J., Davis, J. I., & Ochsner, K. N. (2012). Overlapping activity in anterior insula during interoception and emotional experience. *NeuroImage, 62*(1), 493–499.

Zysset, S., Huber, O., Ferstl, E., & von Cramon, D. Y. (2002). The anterior frontomedian cortex and evaluative judgment: An fMRI study. *NeuroImage, 15*(4), 983–991.

Zysset, S., Huber, O., Samson, A., Ferstl, E. C., & von Cramon, D. Y. (2003). Functional specialization within the anterior medial prefrontal cortex: A functional magnetic resonance imaging study with human subjects. *Neuroscience Letters, 335*(3), 183–186.

CHAPTER 3

Temporal Dynamics of Emotion Regulation

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Gross and colleagues have framed emotion generation and regulation as a recursive process involving attention and appraisal, whereby an emotional response unfolds over time as a function of both how attention is allocated to an emotion-eliciting stimulus or situation and the interpretation of (or meaning attributed to) the stimulus or situation (see Gross, this volume). Based on this model, both attentional deployment and reappraisal are increasingly studied strategies for emotion regulation—and both strategies are effective means of altering emotion.

Functional magnetic resonance imaging (fMRI) measures have been used in the context of emotion regulation studies, providing a portrait of neural regions that are more active during emotion regulation than control conditions—and these data suggest that areas of the prefrontal cortex become more active, and amygdala activity is decreased, when emotion is down-regulated (see Ochsner & Gross, this volume). Though we are coming to know the circuitry of emotion regulation, this is a static portrait: a fine-grained but frozen map representing "more activation here" and "less activation there." To be sure, more advanced analytic techniques can augment traditional analyses of fMRI data to indicate the temporal sequencing of these effects: Presumably, increases in frontal activation precede decreases in amygdala activity.

This chapter focuses on a different neuroimaging method that complements the excellent spatial resolution of fMRI: We review research that uses neural activity in the electroencephalogram (EEG) to study mechanisms and the time course of emotion regulation. We believe that investigations of emotion regulation benefit enormously from measures that are temporally precise and can track processes unfolding—and changing—over time. Much of our work has focused on event-related potentials (ERPs) derived from the EEG. ERPs reflect the near-instantaneous activity of underlying neuronal populations and are well-suited for tracking rapidly changing brain activity. For instance, ERPs can index emotional reactivity and its regulation from one second to the next—and can be used to separately quantify reactivity and regulation. Moreover, ERPs are relatively inexpensive and well-tolerated neural measures. In this chapter, we focus mainly on the *late positive potential* (LPP), an ERP component that reflects the flexible and dynamic deployment of motivated attention, and as such represents an ideal vehicle for investigating iterative and interactive processes as they unfold over time.

ERPs and the LPP

What Is an ERP?

ERPs are a direct measure of neural activity. Scalp-recorded ERPs reflect the summed activity of excitatory and inhibitory postsynaptic potentials (PSPs) generated by large populations of pyramidal cortical neurons aligned perpendicular to the cortical surface. Because these neurons are oriented in the same direction, simultaneously occurring or closely occurring signals typically do not cancel each other out; moreover, because PSPs tend to be rather sustained in nature (compared to, for example, action potentials), it is possible for this activity to summate and propagate to the surface of the scalp (Luck, 2005). This summated activity is what is recorded in the ongoing EEG (see Figure 3.1). ERPs then reflect the activity of the EEG, time-locked to specific events and averaged across many trials to increase signal and reduce noise. In the studies we discuss, the event of interest is typically the presentation of a visual stimulus, frequently images from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008), a standardized database containing hundreds of pictures that have been normed on ratings of hedonic valence and arousal. Neural activity elicited by emotional compared to neutral pictures, and as a function of emotion regulation instructions, can then be averaged and compared within and between subjects.

The LPP

The LPP is a sustained positive-going ERP with a central–parietal scalp distribution; the amplitude of the LPP is modulated by emotional content, becoming more positive as early as 200 milliseconds following the presentation of both pleasant and unpleasant compared to neutral stimuli (see Figure 3.1; Foti, Hajcak, & Dien, 2009; Hajcak & Olvet, 2008; Hajcak, Weinberg, MacNamara, & Foti, 2011). The LPP is larger

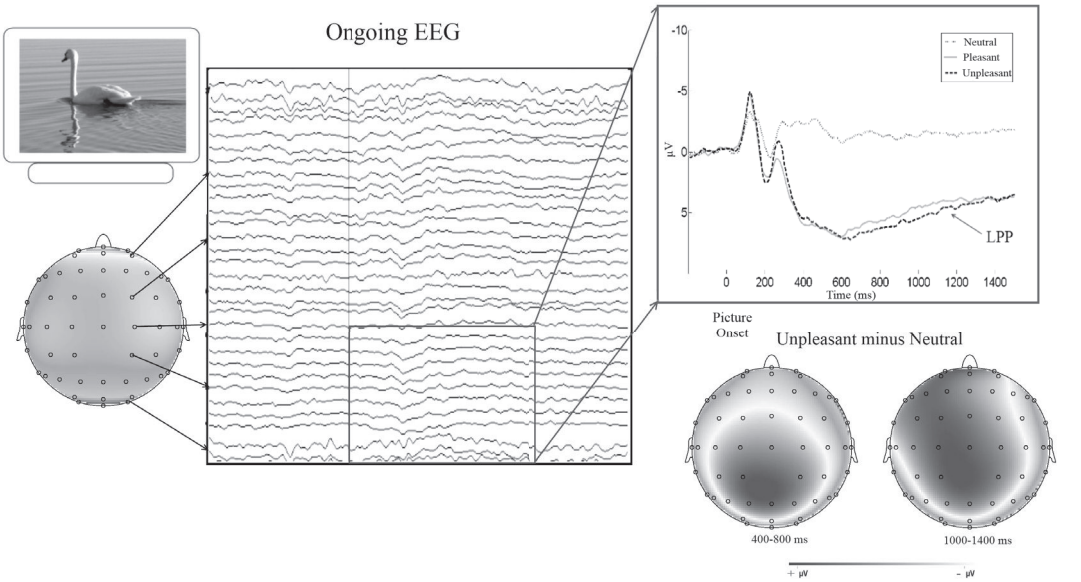

FIGURE 3.1. A participant views images as the ongoing electroencephalogram (EEG) is recorded via sensors affixed to the scalp (left). Event-related potentials (ERPs) reflect the activity of the EEG time-locked to an event of interest, in this case, the onset of an image. On the right, the LPP is presented as a positive-going deflection in the waveform, which is enhanced for both pleasant and unpleasant images compared to neutral (data from Weinberg & Hajcak, 2010). Additionally, while the early portion of the LPP (400–800 milliseconds) has a parietal distribution—represented here by the positive *difference* between unpleasant and neutral images, as indicated by the dark area on the scalp—the later LPP (after 1,000 milliseconds) has a broader and more frontal distribution.

following the presentation of emotional images (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Foti et al., 2009), words (Kissler, Herbert, Winkler, & Junghofer, 2009; Tacikowski & Nowicka, 2010), and even hand gestures (Flaisch, Häcker, Renner, & Schupp, 2011).

It has been argued that the LPP reflects sustained attention to visual stimuli (Hajcak, MacNamara, & Olvet, 2010; Hajcak & Olvet, 2008; Hajcak et al., 2011; Weinberg & Hajcak, 2011b). Furthermore, there may be functional differences between early compared to late portions of the LPP, highlighting the utility of this component in tracking multiple processes over time. Relatively early portions of the LPP (i.e., 300–600 milliseconds) appear to reflect more obligatory processing of emotional stimuli, whereas later time windows (i.e., 600 milliseconds and beyond) might reflect more sustained and elaborative engagement with stimuli (Olofsson, Nordin, Sequeira, & Polich, 2008; Weinberg & Hajcak, 2011b; Weinberg, Hilgard, Bartholow, & Hajcak, 2012). For instance, in one study, larger LPPs in response to task-irrelevant pictures predicted greater behavioral interference—in both within- and between-subjects analyses (Weinberg & Hajcak, 2011b). Moreover, though the early LPP was modulated by emotional stimuli, it was the later portion of the LPP (i.e., just preceding target onset) that uniquely predicted behavioral interference: Only increased sustained attention just prior to target presentation related to slower reaction times.

During passive picture-viewing paradigms, the emotional modulation of the LPP is sustained throughout stimulus presentation (i.e., up to several seconds; Hajcak et al., 2010), and can even be observed for as long as 1,000 milliseconds after stimulus offset. These data are consistent with the notion that emotional stimuli not only capture but also hold attention (Vuilleumier, 2005)—and we have suggested that the LPP is sensitive to the continued allocation of attention to emotional stimuli. Consistent with this view, it is possible to manipulate the *duration* of the LPP by instructions that encourage maintainance of previously viewed images in working memory, again suggesting that this later component may reflect more volitional engagement with emotional stimuli (Hajcak et al., 2010; Thiruchselvam, Hajcak, & Gross, 2012).

The modulation of the LPP is thought to reflect the motivational salience of the stimulus being viewed. In the case of emotional stimuli, salience is thought to be determined by the content of the stimuli—and how relevant the content is to basic biological imperatives (e.g., survival themes that include defense and reproduction). Indeed, evidence suggests that the LPP is most enhanced by image content more directly related to these imperatives, such as images of mutilation and gore, or erotic images (Weinberg & Hajcak, 2010). Additionally, food deprivation compared to satiated states enhances the magnitude of the LPP elicited by images of food—a motivationally salient category of images when one is hungry (Stockburger, Schmälzle, Flaisch, Bublatzky, & Schupp, 2009). The LPP is also enhanced by personally relevant stimuli (e.g., photographs of relatives, or one's own name and face; Grasso & Simons, 2010; Tacikowski & Nowicka, 2010) compared to even very familiar famous faces.

There is also evidence that extrinsic manipulations of salience can impact the LPP. It is possible to designate specific stimuli as "targets" by asking participants to count or otherwise respond to them when they are presented among other nontarget stimuli. The LPP is enhanced for arbitrarily designated target stimuli compared to nontarget stimuli (Ferrari, Bradley, Codispoti, & Lang, 2010; Weinberg et al., 2012), even when the same images serve as both targets and nontargets (Weinberg et al., 2012). The LPP is largest when emotional images are *also* targets, compared to both neutral targets and emotional nontargets (Ferrari et al., 2010; Weinberg et al., 2012). This suggests that intrinsic and extrinsic manipulations of salience are additive in their impact on the magnitude of the LPP.

Work that combines EEG and fMRI suggests that the LPP reflects widespread and concurrent activity across the visual system (Sabatinelli, Keil, Frank, & Lang, 2013), including the visual cortex (Bradley et al., 2003; Keil et al., 2002) as well as occipital, parietal, and inferotemporal regions of the brain (Sabatinelli, Lang, Keil, & Bradley, 2007). Rather than the activation of a single anatomical node, it is possible that the

LPP reflects fairly widespread neuromodulatory activity (de Rover et al., 2012; Hajcak et al., 2010), or communication among diverse brain regions. This latter possibility is supported by a recent study indicating that the magnetic equivalent of the LPP was generated in occipitoparietal and prefrontal cortices—and that the emotional modulation of the LPP might index the coordination of frontoparietal attention networks (Moratti, Saugar, & Strange, 2011).

The involvement of frontal areas in the generation of the LPP is consistent with studies indicating that both physical stimulation (Hajcak, Anderson, et al., 2010) and functional activation (based on working memory load; MacNamara, Ferri, & Hajcak, 2011) of the dorsolateral prefrontal cortex reduces the amplitude of the LPP. Moreover, frontal involvement in the LPP is also consistent with our observation that the scalp distribution of the emotional modulation of the LPP during passive picture viewing becomes evident at central and even frontal recording sites after approximately 1,000 milliseconds (see Figure 3.1; Foti et al., 2009; MacNamara, Foti, & Hajcak, 2009).

A substantial literature indicates that the LPP is a robust measure of neural activity associated with emotional processing. In contrast to the majority of peripheral and central measures that are sensitive to emotional stimuli, emotional modulation of the LPP is highly stable. Whereas skin conductance, heart rate, facial muscle activity, and neural activation indexed by fMRI habituate over repeated presentations of stimuli (Codispoti & De Cesarei, 2007; Codispoti, Ferrari, & Bradley, 2006; Phan, Liberzon, Welsh, Britton, & Taylor, 2003), emotional modulation of the LPP does not (Codispoti et al., 2006; Codispoti, Ferrari, & Bradley, 2007; Olofsson & Polich, 2007).

The LPP and Individual Differences

The LPP has proven to be an effective measure of attentional allocation to emotional stimuli across multiple populations. For example, enhancement of LPP amplitude by emotional content appears cross-culturally (Hot, Saito, Mandai, Kobayashi, & Sequeira, 2006; Yen, Chen, & Liu, 2010). The LPP has also been applied to the study of emotional processing across the lifespan; affective modulation of the LPP has been observed in both very young children (Dennis & Hajcak, 2009; Kujawa, Hajcak, Torpey, Kim, & Klein, 2012) and in adults as old as 81 (Kisley, Wood, & Burrows, 2007; Langeslag & Van Strien, 2010). Furthermore, the LPP has been used to test theories of aging, which posit that younger compared to older participants are more reactive to unpleasant stimuli, and less reactive to pleasant stimuli (Kisley et al., 2007; Langeslag & Van Strien, 2010).

Additionally, the LPP has been used effectively to characterize the abnormal processing of affective stimuli in anxiety, mood, and substance use disorders (e.g., Dunning et al., 2011; Foti, Olvet, Klein, & Hajcak, 2010; Weinberg & Hajcak, 2011a). For instance, consistent with the notion that depression is characterized by motivational disengagement from the environment, depressed individuals tend to show decreased emotional modulation of the LPP compared to healthy controls (Foti et al., 2010). Likewise, while cocaine use disorders are associated with a decreased LPP in response to normative emotional stimuli, individuals with this diagnosis also show an increased LPP in response to cocaine-related images compared to healthy controls (Dunning et al., 2011). The relationship of the LPP to anxiety appears to be somewhat context-dependent. For instance, anxiety has been associated with an increased LPP when pictures are task-irrelevant (MacNamara, Ferri, et al., 2011), suggesting deficits in disengaging attention from emotional stimuli. However, there is also evidence from passive-viewing tasks—in which the pictures are task-relevant—that anxiety is instead characterized by initial vigilance for threat images, as indicated by enhancement of early ERP components, followed by a failure to engage in sustained processing of these images, as indicated by an attenuation of the LPP (Weinberg & Hajcak, 2011a). These data indicate that the LPP is a useful tool for quantifying abnormalities in the temporal dynamics of emotional processing across different forms of psychopathology. Below, we review findings that have extended this literature to emotion regulation, focusing in particular on attentional deployment and reappraisal.

The LPP and Emotion Regulation

Directed Attention

An emotional response to a stimulus unfolds over time as a function of multiple processes, including the allocation of attention to the content of the stimulus, as well as the meaning that is assigned to that stimulus (Sheppes & Gross, 2011). With regard to emotion regulation, distraction and other attentional deployment strategies alter the emotional response by directing attention away from affective content. Spatial attention, in particular, plays a large role in the electrocortical response to emotional images (Eimer, Holmes, & McGlone, 2003; Holmes, Vuilleumier, & Eimer, 2003; Keil, Moratti, Sabatinelli, Bradley, & Lang, 2005) and, consequently, emotion regulation processes. Indeed, a growing body of work demonstrates that directing attention, either implicitly or explicitly, away from emotionally salient features of a stimulus is an effective method of down-regulating emotional response.

Online Regulation

Attempts to regulate an emotional response after a stimulus has been encountered are considered "online" regulation. Recent work has examined whether the amplitude of the LPP can be manipulated by explicitly directing participants' visual attention to various areas *within* unpleasant images. This approach is considered online regulation insofar as visual attention is manipulated during the generation of the emotional response. In one study, participants were shown an unpleasant picture for 6 seconds; after passively viewing the image for 3 seconds, participants' attention was then directed to either an arousing or nonarousing area of the picture for the last 3 seconds of each trial. As indicated in Figure 3.2, directing attention to nonarousing com-

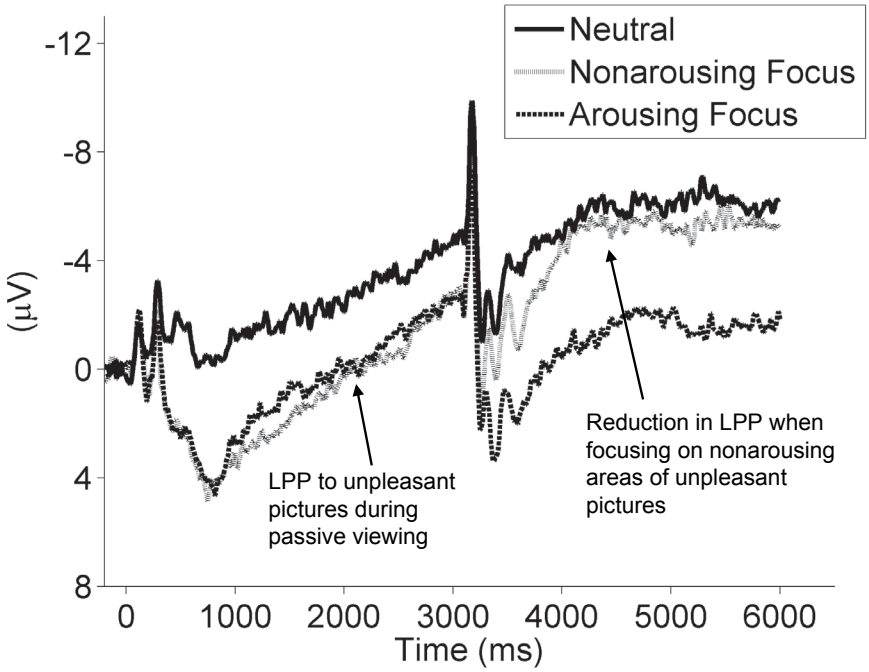

FIGURE 3.2. Grand averaged ERPs at electrode site Pz elicited by neutral pictures (solid black line; always associated with an instruction to focus on a nonarousing area of the image) and unpleasant pictures (associated with an instruction to attend to either a nonarousing [dotted line] or arousing [dashed line area of the image]). Picture onset occurred at 0 milliseconds and the instruction tone occurred at 3,000 milliseconds. The figure is based on data collected by Hajcak, Dunning, and Foti (2009).

pared to highly arousing areas of unpleasant images resulted in a decreased LPP (Dunning & Hajcak, 2009). This finding was subsequently replicated with a focus on the time course of LPP modulation: Sequential significance testing indicated that reduction of the LPP occurred 620 milliseconds after signaling participants to attend to nonarousing areas of unpleasant images (Hajcak, Dunning, & Foti, 2009).

Directed attention also appears to impact the emotional response to representations in working memory during a post-stimulus period. Thiruchselvam and colleagues (2012) had participants view unpleasant and neutral pictures, then maintain representations of each image in working memory after picture offset. Participants were then instructed to focus on arousing or nonarousing aspects of their mental representation. Results showed that focusing on nonarousing compared to arousing aspects of pictures held in working memory was related to a reduction in both the LPP and self-reported ratings of unpleasantness. These results suggest that the emotional response can be modulated, and indexed by the LPP, even after images have been fully encoded in working memory.

Convergence of Directed Attention Findings across Neural Measures

This same pattern of LPP modulation via directed spatial attention has also been replicated and extended to steady-state visual evoked potentials (ssVEP; Hajcak, MacNamara, Foti, Ferri, & Keil, 2013). The ssVEP can be used to index visual processing of stimuli flickering at a particular frequency. It is not an ERP component, but rather a measure of the degree to which the EEG signal over occipital cortex is being driven at the flicker frequency. For instance, suppose two faces are simultaneously presented to the left and right of fixation, flickered at 8 and 18 Hz, respectively. The ssVEP would be characterized by increased activity at both frequencies, which is typically maximal at occipital electrodes. Furthermore, if participants were instructed to attend to the left face, power at 8 Hz in the ssVEP would be increased (Hillyard et al., 1997). Related to this chapter, the ssVEP is larger for emotional compared to neutral flickering stimuli, and modulation of this activity is linked to facilitated processing in the visual cortex (Keil et al., 2003). In a study combining the LPP and ssVEP within a single paradigm, directing attention to nonarousing areas of unpleasant pictures reduced both of these electrocortical indices, indicating that each neural measure tracks the effective down-regulation of the emotional response via attentional deployment (Hajcak et al., 2013). Moreover, modulations of the LPP and ssVEP were unrelated, suggesting that these measures provide distinct sources of information regarding emotional processing.

Cognitive Reappraisal

Unlike distraction and other attentional deployment techniques, where attention is directed away from the emotional content of a stimulus, reappraisal involves attending directly to emotional content and altering the emotional response by reinterpreting the *meaning* of the stimulus (Sheppes & Gross, 2011). In this way, emotion regulation is achieved while engaging directly with emotionally arousing aspects of stimuli, an approach that has been shown to be effective for reducing both subjective and peripheral physiological indicators of emotional arousal (Urry, 2010). Of interest is how LPP amplitude may complement these data as a neurobiological index of effective emotion regulation, clarifying the impact of cognitive reappraisal on the time course of emotional processing.

According to the process-specific timing hypothesis, online emotion regulation likely requires significant effort (i.e., cognitive resources) in order to modify existing and incoming emotional information—and that effort would be directly proportional to the strength and intensity of the emotion being experienced (Sheppes & Gross, 2011). On the other hand, several studies have presented emotion regulation instructions to participants *prior to* the presentation of emotional stimuli. In this way, regulation goals might be established while emotional intensity is low, in anticipation of the emotional response to come. Anticipatory regulation ought to impact the initial processing of an affective stimulus, indicated by the

affective modulation of the LPP elicited by the first presentation of the stimulus.

In one of the first investigations of cognitive reappraisal using the LPP, Hajcak and Nieuwenhuis (2006) had participants view a series of unpleasant IAPS images for 1 second; an instruction to "reinterpret" (i.e., reappraise the picture in order to reduce one's negative response) or "attend" (i.e., focus on one's natural feelings about the picture) was then presented for 4,500 milliseconds, after which the same picture was again presented for 2,000 milliseconds. Compared to unpleasant pictures presented after the attend instructions, those presented after reappraisal instructions were associated with a reduced LPP, beginning 200 milliseconds after picture onset—an effect that lasted for the duration of picture presentation. Furthermore, greater reduction in the LPP was related to greater reduction in self-reported arousal ratings of the images. This study was the first to demonstrate that the magnitude of the LPP could be modulated by cognitive reappraisal, down-regulating the LPP by reinterpreting the meaning of emotional stimuli.

Anticipatory Regulation

In the Hajcak and Nieuwenhuis (2006) study, regulation instructions were presented after the first encounter with the emotional stimulus, indicating that the LPP is sensitive to cognitive reappraisal when it is done online. Separate from this study, and consistent with the notion of anticipatory regulation, experimental manipulations that vary how participants attend to affective stimuli during the initial presentation have also been shown to modulate the LPP. For example, Hajcak, Moser, and Simons (2006) had participants view pleasant and unpleasant pictures, and either categorize the pictures affectively (i.e., indicating whether the picture was pleasant or unpleasant) or nonaffectively (i.e., indicating how many people were present in the picture). Results indicated that the LPP elicited by both pleasant and unpleasant pictures was reduced when participants evaluated images—and, presumably, interpreted the meaning of the images—along a nonaffective compared to an affective dimension.

In three additional studies, the impact of cognitive reappraisal within an anticipatory emotion regulation context was examined, testing whether alteration of the manner in which participants appraise stimuli during the initial presentation would modulate LPP amplitude. Participants were instructed to use effortful cognitive reappraisal of emotional scenes (Krompinger, Moser, & Simons, 2008; Moser, Hajcak, Bukay, & Simons, 2006) and angry faces (Blechert, Sheppes, Di Tella, Williams, & Gross, 2012). Prior to viewing each stimulus, participants were instructed to decrease the intensity of their emotional response by reinterpreting the image. Compared to a passive viewing condition, the LPP elicited by pleasant and unpleasant images was blunted for the reappraisal condition at an early latency range, beginning approximately 300–600 milliseconds following picture presentation. Together, these lines of research demonstrate that LPP amplitude tracks the successful down-regulation of an emotional response using cognitive reappraisal, whether reappraisal is enacted online or in anticipation of an affective stimulus.

Building on these lines of research, Thiruchselvam, Blechert, Sheppes, Rydstrom, and Gross (2011) compared the effects of different anticipatory regulation strategies on LPP amplitude at both the initial presentation of stimuli and a later reexposure. Whereas the aforementioned studies compared reappraisal to a passive viewing condition, here the impact of reappraisal on the temporal dynamics of LPP amplitude was also contrasted with the impact of distraction instructions. Reappraisal was achieved by instructing participants to reinterpret affective stimuli in a more neutral manner. Distraction, on the other hand, was achieved by asking participants to generate neutral thoughts unrelated to the stimulus, such as imagining complex geometric patterns. During a baseline session, the LPP in response to unpleasant images was recorded under conditions of passive viewing, effortful reappraisal, and distraction; 30 minutes later, participants passively viewed the same images with no regulation instructions. Effortful reappraisal, compared to passive viewing, yielded a blunted LPP only within a relatively later latency range (i.e., 1,500–

1,700 milliseconds). The effect of distraction was stronger and apparent earlier, with a significant and sustained reduction in LPP amplitude as early as 300 milliseconds. A very different pattern emerged during the reexposure session: Images that had previously been processed under the reappraisal condition again yielded a blunted LPP compared to the passive viewing condition, from 800 to 1,400 milliseconds. Images that had previously been processed under the distraction condition, however, yielded a sustained *increase* in LPP amplitude compared to the passive viewing condition, beginning at approximately 1,200 milliseconds. This study highlights the fact that emotion regulation strategies likely have a differential impact on initial compared to subsequent encounters with emotional stimuli. Unlike reappraisal, distraction may only be an effective emotion regulation strategy in the short-term, reducing LPP amplitude during the initial stimulus exposure but resulting in an enhanced LPP during reexposure.

Preappraisal

The previous studies indicate that LPP amplitude is sensitive to the implementation of anticipatory emotion regulation through cognitive reappraisal, but they do not fully address whether the down-regulation of the LPP is due to a shift in stimulus appraisal/meaning per se. An alternate explanation is that the reduction in LPP amplitude is a result of reappraisal being more difficult and more cognitively taxing than passive viewing, an important possibility to rule out in light of evidence that cognitive load results in a reduced LPP (MacNamara, Ferri, et al., 2011). To pursue this issue further, several studies have used a guided version of cognitive reappraisal, in which images are preceded by verbal descriptions that frame the upcoming stimuli in either a more negative or more neutral manner (Foti & Hajcak, 2008; MacNamara et al., 2009). In other words, reappraisal frames were provided to participants, a manipulation that might be best described as *preappraisal*. For example, an unpleasant image of a man pointing a gun at his head might be preceded by either "This man is about to commit suicide" or "This man ends up not committing suicide"; the latter description is designed to down-regulate the initial emotional response elicited by the image. On each trial, the description precedes the presentation of the image, thereby shaping the first appraisal of the stimulus during the initial iteration of the emotion-generative cycle. Guiding the initial stimulus appraisal with verbal descriptions removes the potential confound of task difficulty, such that each condition simply involves listening to descriptions and then viewing pictures.

In an initial study using this paradigm with an adult sample, modulation of LPP amplitude by description type was evident as early as 400 milliseconds following picture presentation, with neutral descriptions reducing the subsequent LPP elicited by unpleasant images (Foti & Hajcak, 2008). Compared to neutral images—which in this study were always preceded by a neutral description—the LPP in response to unpleasant images preceded by unpleasant descriptions was increased throughout stimulus presentation, from 400 to 3,000 milliseconds. The LPP in response to unpleasant images was increased only from 400 to 1,000 milliseconds when preceded by neutral descriptions, and to a lesser extent than when preceded by negative descriptions. This temporal pattern suggests that unpleasant images preappraised by a neutral description were associated with reduced early, obligatory attentional capture associated with emotion generation; moreover, the sustained, elaborative processing that is typically observed for highly arousing affective stimuli was nearly absent. Unpleasant images preceded by neutral descriptions were also rated as less unpleasant and less emotionally arousing than those preceded by negative descriptions, providing further evidence that manipulating the initial appraisal of the stimuli effectively regulated the subsequent emotional response.

Because neutral images were always preceded by neutral descriptions in this initial study, an important alternative explanation was that the reduced LPP observed for neutrally described unpleasant images was due to the mismatch between the valence of the description and the image. To address this confound in a follow-up study on preappraisal, description and image type were fully crossed: Neutral and negative descriptions preceded both neutral and unpleasant images (MacNamara et al., 2009). For

instance, a neutral image of a drill might be described as "This drill is used by a repairman" or "This drill was used in a grisly murder," with the latter description designed to up-regulate the emotional response elicited by the image. As before, the LPP was reduced in response to unpleasant images preceded by neutral descriptions compared to those preceded by negative descriptions. Importantly, though, the LPP was increased in response to neutral pictures that were preceded by negative compared to neutral descriptions. Framing neutral images in a more negative manner elicited affective modulation of the LPP that would not normally be observed for low-arousal, neutral stimuli. These data demonstrate that the LPP tracks the emotionality of the preappraisal and not the match between description and image. In fact, at late latencies (>1,500 milliseconds), affective modulation of LPP amplitude was apparent *only* for images preceded by negative descriptions, regardless of whether the image itself was unpleasant or neutral. Thus, the later portion of the LPP was only sensitive to meaning imbued by the preappraisals.

Altering the context in which stimuli are initially appraised has a robust impact on LPP amplitude during the first viewing of a stimulus. A further question is how modifying the initial appraisal may influence subsequent iterations of the emotion-generative process when the stimuli are encountered again. To examine this, MacNamara, Ochsner, and Hajcak (2011) first presented images paired with negative or neutral descriptions, then conducted a second viewing session 30 minutes later without the preceding descriptions. During the reexposure session, images that had previously been preappraised in a more neutral manner elicited a blunted LPP in a middle latency range (i.e., 700–1,400 milliseconds), and the images were rated as being less unpleasant and less emotionally arousing. This is a less protracted effect of preappraisal on LPP than observed previously (Foti & Hajcak, 2008; MacNamara et al., 2009), suggesting that preappraisal effects may diminish over time. However, modifying the initial appraisal of an affective stimulus—whether by effortful reappraisal (Thiruchselvam et al., 2011) or preappraisal (MacNamara, Ochsner, et al., 2011)—appears to yield down-regulation of the LPP upon reexposure.

Although the aforementioned studies used paradigms in which the stimuli were task-relevant and the primary focus of attention, modifying the initial stimulus appraisal also appears to impact the LPP elicited by *task-irrelevant*, distracting images. For instance, during a task in which participants were required to judge the spatial orientation of geometric shapes, mutilation and neutral scenes were presented as distractors (Mocaiber et al., 2010). These images were described beforehand as being either real or fictitious, thereby shaping the initial appraisal. In the "real" context, unpleasant images elicited an increased LPP compared to neutral images in an early latency range (i.e., 300–600 milliseconds) and were associated with significant reaction time slowing, indicating attentional capture that interfered with performance on the primary task. In the "fictitious" context, however, there was no affective modulation of the LPP and no reaction time slowing, indicating that the distractor images were less salient and produced less behavioral interference. This study is consistent with other research linking the LPP to attentional capture and task interference (Weinberg & Hajcak, 2011b), and it demonstrates that the LPP elicited by task-irrelevant stimuli can be modulated by preappraisal.

Convergence of Cognitive Reappraisal Findings across Neural Measures

The work reviewed thus far focused on identifying the consequences of regulation on the emotion-generative process, as indexed by electrocortical measures. In other words, electrocortical measures were used to index the downstream impact of emotion regulation on neural activity. In addition to this, it may be possible to measure neural activation responsible for these regulation effects. For instance, Parvaz, MacNamara, Goldstein, and Hajcak (2012) examined the LPP and alpha bandwidth power while participants either passively viewed or reappraised unpleasant pictures. Consistent with previous work, the amplitude of the LPP was reduced when reappraising unpleasant pictures. However, reappraisal was also associated with increased left prefrontal cortex activation, as evidenced by reduced left frontal alpha band power (alpha power

is inversely related to brain activity). LPP amplitude and alpha power were not correlated in this study, though, suggesting that both of these measures contributed unique information about cognitive reappraisal processes. These data are consistent with fMRI research indicating that reappraisal is associated with increased activity in the ventromedial and lateral prefrontal cortex (Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007; Ochsner et al., 2004; Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008), and suggests that EEG can be used not only to index the downstream consequences of emotion regulation but also the neural activity that may be responsible for regulatory effects.

Clinical Applications

Researchers have begun to apply the LPP to the study of emotion regulation in clinical contexts. For example, the effects of preappraisal have been studied in children, with the purpose of identifying risk factors that might aid in detection and treatment of emotion regulation difficulties early in life. In one study of children ages 7–10, the LPP was reduced for unpleasant images preceded by a neutral description, compared to unpleasant images preceded by a negative description (Dennis & Hajcak, 2009). This modulation of the LPP by description type was associated with reduced symptoms of depression and anxiety, suggesting that modulation of LPP amplitude may serve as an early marker for emotion regulation difficulties or mood disruptions. In younger children ages 5–7, however, preappraisal does not appear to modulate LPP amplitude (Decicco, Solomon, & Dennis, 2012; Dennis & Hajcak, 2009), suggesting important developmental differences that warrant further investigation.

The LPP has also been used to track emotion regulation processes related to the treatment of psychological disorders. For example, individuals with phobias are characterized by an attentional pattern of vigilance followed by avoidance: They rapidly detect and attend to phobic objects initially but quickly direct attention away from the phobic object. This is a maladaptive emotion regulation strategy that prevents habituation and reinforces the anxiety response over time through active attentional avoidance. In one study of individuals with a spider phobia, the LPP elicited by spider stimuli was increased compared to other negative stimuli during an early time window (340–770 milliseconds) but not a later time window (800–1,500 milliseconds; Leutgeb, Schafer, & Schienle, 2009)—consistent with an attentional pattern of vigilance followed by avoidance. Participants were then treated with exposure therapy in which they were trained to approach and engage with a spider until their anxiety diminished, rather than avoid the spider. Compared to a wait-list control group, individuals in the exposure therapy condition exhibited an increased and sustained LPP in response to spider stimuli from 800 to 1,500 milliseconds at a follow-up testing session. This effect of treatment on LPP amplitude was specific to spider stimuli and was not observed for other unpleasant stimuli. These data suggest that successful treatment led to increased attention and engagement with the phobic object.

Other researchers have used the LPP to clarify patterns of emotion regulation deficits associated with the treatment of substance use disorders. Attentional biases toward drug-related stimuli play a significant role in drug abuse and addiction (Field & Cox, 2008), and several studies have found that larger LPPs are elicited by drug-related compared to neutral pictures in alcoholics (Herrmann et al., 2000; Namkoong, Lee, Lee, Lee, & An, 2004), cocaine-addicted individuals (Dunning et al., 2011; Franken et al., 2008), and smokers (Littel & Franken, 2007, 2011a). Furthermore, attentional bias toward drug cues and away from other, intrinsically pleasant stimuli has been related to risk for relapse. In one smoking cessation study, the LPP elicited by cigarette-related cues was increased among all smokers, but a subgroup of individuals also exhibited a blunted LPP to normative pleasant stimuli that was associated with higher rates of relapse over 6 months (Versace et al., 2012). This suggests that the LPP could serve as a biomarker for identifying smokers with a higher risk of relapse (Versace et al., 2012).

Given that drug craving has been shown to be modulated by cognitive regulation (Kober, Kross, Mischel, Hart, & Ochsner,

2010), Littel and Franken (2011b) sought to examine whether the LPP in response to smoking-related images could be modulated by emotion regulation strategies in smokers. Indeed, both reappraisal (i.e., viewing the scenes from an uninvolved, rational, and detached perspective) and distraction (i.e., identifying the dominant color in the scene) strategies reduced the amplitude of the LPP to smoking-related pictures; this effect was prevalent in both light and heavy smokers, suggesting that regulation of drug-related stimuli can be accomplished by smokers with differing levels of dependency (Littel & Franken, 2011b).

Future Research Directions

Although electrocortical activity has been used for decades to study cognitive processes that include attention, it has more recently been used to measure attention to emotional stimuli (Hajcak et al., 2011). We have argued that the LPP is modulated by sustained attention to motivationally salient stimuli, possibly indexing the coordinated increased activity of frontoparietal attention networks. However, the precise mechanisms of this sustained attention remain unclear. Although evidence suggests that the LPP reflects enhanced attention to emotional stimuli, some have suggested that the LPP might instead reflect the *suppressed* processing of competing stimuli (Brown, van Steenbergen, Band, de Rover, & Nieuwenhuis, 2012; MacNamara, Ferri, et al., 2011; Mocaiber et al., 2010; Weinberg & Hajcak, 2011b). Future work using fMRI might help to clarify whether the LPP indexes facilitated attention to emotional stimuli. For instance, given that the LPP is modulated by both attentional deployment and cognitive reappraisal, it would be informative to know what neural circuits are similarly increased or decreased during these emotion regulation strategies. Moreover, it is imperative to combine EEG and fMRI technologies in future research. What neural regions and networks support the emotional modulation of the LPP and its reduction through reappraisal and attentional deployment? Do fMRI and EEG provide complementary or redundant information within and between subjects? In our view, these are crucial and exciting questions that can be answered in the years ahead.

Initial research indicated that the LPP is reduced via reappraisal, and subsequent studies confirm that changes in stimulus meaning are sufficient to modulate the LPP. Moreover, meaning-based changes that characterize reappraisal appear to alter the LPP elicited by subsequent encounters with emotional stimuli—and more so than other emotion regulation strategies (i.e., distraction). Both the LPP and ssVEPs are modulated by attentional deployment: When participants focus on less arousing aspects of emotional stimuli, both metrics of neural activity are down-regulated—and these effects become evident within about 200 milliseconds. Because of this temporal resolution, electrocortical indices of emotion and its regulation can be used to index the iterative and recursive processes that determine an emotional response. Recent work also highlights the possibility that frontal activation in reappraisal can be indexed using time-frequency decompositions of EEG data (Parvaz et al., 2012).

Electrocortical measures of emotion and its regulation have been fruitful, yet there are many open avenues for further exploration, particularly with regard to the study of individual differences in emotion regulation. What emotion strategies work best for whom? In what ways does emotion regulation go awry across psychological disorders? There is an emerging clinical literature utilizing the LPP to shed light on the temporal dynamics of information processing abnormalities associated with psychopathology. As discussed elsewhere in this volume (Parts VII and VIII, Chapters 24–32), research on emotion regulation has important implications for the treatment of a range of psychological disorders, and bringing the LPP to bear on this topic may be useful for both quantifying impairment in emotion regulation and clarifying mechanisms of change over the course of treatment. As a neurobiological complement to self-report and behavioral indices of emotion regulation difficulties, LPP data may provide unique information regarding the time course of abnormal emotional information processing within a specific patient population, which may then become a target for subsequent intervention.

As evidenced by the chapters in this volume, emotion regulation is a topic that figures heavily in both developmental and clinical literatures. Although electrocortical activity has been relatively underutilized in these areas, EEG-based measures of neural function might be particularly suitable for emotion regulation studies in children and clinical populations. The few and emerging studies in these areas suggest a large and positive potential.

References

Blechert, J., Sheppes, G., Di Tella, C., Williams, H., & Gross, J. J. (2012). See what you think: Reappraisal modulates behavioral and neural responses to social stimuli. *Psychological Science, 23*(4), 346–353.

Bradley, M., Sabatinelli, D., Lang, P., Fitzsimmons, J., King, W., & Desai, P. (2003). Activation of the visual cortex in motivated attention. *Behavioral Neuroscience, 117*(2), 369–380.

Brown, S. B. R. E., van Steenbergen, H., Band, G. P. H., de Rover, M., & Nieuwenhuis, S. (2012). Functional significance of the emotion-related late positive potential. *Frontiers in Human Neuroscience, 6*. [E-publication ahead of print]

Codispoti, M., & De Cesarei, A. (2007). Arousal and attention: Picture size and emotional reactions. *Psychophysiology, 44*(5), 680–686.

Codispoti, M., Ferrari, V., & Bradley, M. (2006). Repetitive picture processing: Autonomic and cortical correlates. *Brain Research, 1068*(1), 213–220.

Codispoti, M., Ferrari, V., & Bradley, M. (2007). Repetition and event-related potentials: Distinguishing early and late processes in affective picture perception. *Journal of Cognitive Neuroscience, 19*(4), 577–586.

Codispoti, M., Mazzetti, M., & Bradley, M. (2009). Unmasking emotion: Exposure duration and emotional engagement. *Psychophysiology, 46*(4), 731–738.

Cuthbert, B., Schupp, H., Bradley, M., Birbaumer, N., & Lang, P. (2000). Brain potentials in affective picture processing: Covariation with autonomic arousal and affective report. *Biological Psychology, 52*(2), 95–111.

de Rover, M., Brown, S., Boot, N., Hajcak, G., van Noorden, M., van der Wee, N., & Nieuwenhuis, S. (2012). Beta receptor–mediated modulation of the late positive potential in humans. *Psychopharmacology, 219*(4), 971–979.

Decicco, J. M., Solomon, B., & Dennis, T. A. (2012). Neural Correlates of Cognitive Reappraisal in Children: An ERP study. *Developmental Cognitive Neuroscience, 2*(1), 79–80.

Dennis, T., & Hajcak, G. (2009). The late positive potential: A neurophysiological marker for emotion regulation in children. *Journal of Child Psychology and Psychiatry, 50*(11), 1373–1383.

Dunning, J. P., & Hajcak, G. (2009). See no evil: Directing visual attention within unpleasant images modulates the electrocortical response. *Psychophysiology, 46*(1), 28–33.

Dunning, J. P., Parvaz, M. A., Hajcak, G., Maloney, T., Alia-Klein, N., Woicik, P. A., et al. (2011). Motivated attention to cocaine and emotional cues in abstinent and current cocaine users—An ERP study. *European Journal of Neuroscience, 33*, 1716–1723.

Eimer, M., Holmes, A., & McGlone, F. (2003). The role of spatial attention in the processing of facial expression: An ERP study of rapid brain responses to six basic emotions. *Cognitive, Affective, and Behavioral Neuroscience, 3*(2), 97–110.

Ferrari, V., Bradley, M., Codispoti, M., & Lang, P. (2010). Detecting novelty and significance. *Journal of Cognitive Neuroscience, 22*(2), 404–411.

Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: A review of its development, causes, and consequences. *Drug and Alcohol Dependence, 97*(1–2), 1–20.

Flaisch, T., Häcker, F., Renner, B., & Schupp, H. (2011). Emotion and the processing of symbolic gestures: An event-related brain potential study. *Social Cognitive and Affective Neuroscience, 6*(1), 109–118.

Foti, D., & Hajcak, G. (2008). Deconstructing reappraisal: Descriptions preceding arousing pictures modulate the subsequent neural response. *Journal of Cognitive Neuroscience, 20*(6), 977–988.

Foti, D., Hajcak, G., & Dien, J. (2009). Differentiating neural responses to emotional pictures: Evidence from temporal–spatial PCA. *Psychophysiology, 46*, 521–530.

Foti, D., Olvet, D., Klein, D., & Hajcak, G. (2010). Reduced electrocortical response to threatening faces in major depressive disorder. *Depression and Anxiety, 27*(9), 813–820.

Franken, I. H. A., Dietvorst, R. C., Hesselmans, M., Franzek, E. J., Van De Wetering, B. J. M.,

& Van Strien, J. W. (2008). Cocaine craving is associated with electrophysiological brain responses to cocaine-related stimuli. *Addiction Biology, 13*, 386–392.

Grasso, D. J., & Simons, R. F. (2011). Perceived parental support predicts enhanced late positive event-related brain potentials to parent faces. *Biological Psychology, 86*(1), 26–30.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Hajcak, G., Anderson, B., Arana, A., Borckardt, J., Takacs, I., George, M., et al. (2010). Dorsolateral prefrontal cortex stimulation modulates electrocortical measures of visual attention: Evidence from direct bilateral epidural cortical stimulation in treatment-resistant mood disorder. *Neuroscience, 170*(1), 281–288.

Hajcak, G., Dunning, J. P., & Foti, D. (2009). Motivated and controlled attention to emotion: Time-course of the late positive potential. *Clinical Neurophysiology, 120*, 505–510.

Hajcak, G., MacNamara, A., Foti, D., Ferri, J., & Keil, A. (2013). The dynamic allocation of attention to emotion: Simultaneous and independent evidence from the late positive potential and steady state visual evoked potentials. *Biological Psychology, 92*(3), 447–455.

Hajcak, G., MacNamara, A., & Olvet, D. M. (2010). Event-related potentials, emotion, and emotion regulation: An integrative review. *Developmental Neuropsychology, 35*(2), 129–155.

Hajcak, G., Moser, J. S., & Simons, R. F. (2006). Attending to affect: Appraisal strategies modulate the electrocortical response to arousing pictures. *Emotion, 6*(3), 517–522.

Hajcak, G., & Nieuwenhuis, S. (2006). Reappraisal modulates the electrocortical response to unpleasant pictures. *Cognitive, Affective, and Behavioral Neuroscience, 6*, 291–297.

Hajcak, G., & Olvet, D. M. (2008). The persistence of attention to emotion: Brain potentials during and after picture presentation. *Emotion, 8*(2), 250–255.

Hajcak, G., Weinberg, A., MacNamara, A., & Foti, D. (2011). ERPs and the study of emotion. In S. Luck & E. Kappenman (Eds.), *The Oxford handbook of event-related potential components* (pp. 441–474). New York: Oxford University Press.

Herrmann, M. J., Weijers, H., Wiesbeck, G. A., Aranda, D., Böning, J., & Fallgatter, A. J. (2000). Event-related potentials and cue-reactivity in alcoholism. *Alcoholism: Clinical and Experimental Research, 24*(11), 1724–1729.

Hillyard, S. A., Hinrichs, H., Tempelmann, C., Morgan, S. T., Hansen, J. C., Scheich, H., et al. (1997). Combining steady-state visual evoked potentials and fMRI to localize brain activity during selective attention. *Human Brain Mapping, 5*(4), 287–292.

Holmes, A., Vuilleumier, P., & Eimer, M. (2003). The processing of emotional facial expression is gated by spatial attention: Evidence from event-related brain potentials. *Cognitive Brain Research, 16*(2), 174–184.

Hot, P., Saito, Y., Mandai, O., Kobayashi, T., & Sequeira, H. (2006). An ERP investigation of emotional processing in European and Japanese individuals. *Brain Research, 1122*(1), 171–178.

Johnstone, T., van Reekum, C. M., Urry, H. L., Kalin, N. H., & Davidson, R. J. (2007). Failure to regulate: Counterproductive recruitment of top-down prefrontal–subcortical circuitry in major depression. *Journal of Neuroscience, 27*(33), 8877–8884.

Keil, A., Bradley, M., Hauk, O., Rockstroh, B., Elbert, T., & Lang, P. (2002). Large-scale neural correlates of affective picture processing. *Psychophysiology, 39*(5), 641–649.

Keil, A., Gruber, T., Muller, M., Moratti, S., Stolarova, M., Bradley, M., & Lang, P. (2003). Early modulation of visual perception by emotional arousal: Evidence from steady-state visual evoked brain potentials. *Cognitive, Affective, and Behavioral Neuroscience, 3*(3), 195–206.

Keil, A., Moratti, S., Sabatinelli, D., Bradley, M., & Lang, P. (2005). Additive effects of emotional content and spatial selective attention on electrocortical facilitation. *Cerebral Cortex, 15*(8), 1187–1197.

Kisley, M. A., Wood, S., & Burrows, C. L. (2007). Looking at the sunny side of life. *Psychological Science, 18*(9), 838–843.

Kissler, J., Herbert, C., Winkler, I., & Junghofer, M. (2009). Emotion and attention in visual word processing—An ERP study. *Biological Psychology, 80*(1), 75–83.

Kober, H., Kross, E. F., Mischel, W., Hart, C. L., & Ochsner, K. N. (2010). Regulation of craving by cognitive strategies in cigarette smokers. *Drug and Alcohol Dependence, 106*(1), 52–55.

Krompinger, J., Moser, J., & Simons, R. (2008).

Modulations of the electrophysiological response to pleasant stimuli by cognitive reappraisal. *Emotion, 8*(1), 132–137.

Kujawa, A., Hajcak, G., Torpey, D., Kim, J., & Klein, D. (2012). Electrocortical reactivity to emotional faces in young children and associations with maternal and paternal depression. *Journal of Child Psychology and Psychiatry, 53*, 207–215.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2008). *International Affective Picture System (IAPS): Affective ratings of pictures and instructional manual* (Technical Report A-8). Gainesville: University of Florida.

Langeslag, S. J. E., & Van Strien, J. W. (2010). Comparable modulation of the late positive potential by emotion regulation in younger and older adults. *Journal of Psychophysiology, 24*(3), 186–197.

Leutgeb, V., Schafer, A., & Schienle, A. (2009). An event-related potential study on exposure therapy for patients suffering from spider phobia. *Biological Psychology, 82*(3), 293–300.

Littel, M., & Franken, I. H. A. (2007). The effects of prolonged abstinence on the processing of smoking cues: An ERP study among smokers, ex-smokers and never-smokers. *Journal of Psychopharmacology, 21*(8), 873–882.

Littel, M., & Franken, I. H. A. (2011a). Implicit and explicit selective attention to smoking cues in smokers indexed by brain potentials. *Journal of Psychopharmacology, 25*(4), 503–513.

Littel, M., & Franken, I. H. A. (2011b). Intentional modulation of the late positive potential in response to smoking cues by cognitive strategies in smokers. *PloS ONE, 6*(11), e27519.

Luck, S. (2005). *An introduction to the event-related potential technique*. Cambridge, MA: MIT Press.

MacNamara, A., Ferri, J., & Hajcak, G. (2011). Working memory load reduces the late positive potential and this effect is attenuated with increasing anxiety. *Cognitive, Affective, and Behavioral Neuroscience, 11*(3), 321–331.

MacNamara, A., Foti, D., & Hajcak, G. (2009). Tell me about it: Neural activity elicited by emotional stimuli and preceding descriptions. *Emotion, 9*(4), 531–543.

MacNamara, A., Ochsner, K. N., & Hajcak, G. (2011). Previously reappraised: The lasting effect of description type on picture-elicited electrocortical activity. *Social Cognitive and Affective Neuroscience, 6*(3), 348–358.

Mocaiber, I., Pereira, M. G., Erthal, F. S., Machado-Pinheiro, W., David, I. A., Cagy, M., et al. (2010). Fact or fiction?: An event-related potential study of implicit emotion regulation. *Neuroscience Letters, 476*(2), 84–88.

Moratti, S., Saugar, C., & Strange, B. A. (2011). Prefrontal–occipitoparietal coupling underlies late latency human neuronal responses to emotion. *Journal of Neuroscience, 31*(47), 17278–17286.

Moser, J., Hajcak, G., Bukay, E., & Simons, R. (2006). Intentional modulation of emotional responding to unpleasant pictures: An ERP study. *Psychophysiology, 43*(3), 292–296.

Namkoong, K., Lee, E., Lee, C. H., Lee, B. O., & An, S. K. (2004). Increased P3 amplitudes induced by alcohol-related pictures in patients with alcohol dependence. *Alcoholism: Clinical and Experimental Research, 28*(9), 1317–1323.

Ochsner, K. N., & Gross, J. J. (this volume, 2014). The neural bases of emotion and emotion regulation: A valuation perspective. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp.23–42). New York: Guilford Press.

Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D. E., et al. (2004). For better or for worse: Neural systems supporting the cognitive down-and up-regulation of negative emotion. *NeuroImage, 23*(2), 483–499.

Olofsson, J., Nordin, S., Sequeira, H., & Polich, J. (2008). Affective picture processing: An integrative review of ERP findings. *Biological Psychology, 77*(3), 247–265.

Olofsson, J., & Polich, J. (2007). Affective visual event-related potentials: Arousal, repetition, and time-on-task. *Biological Psychology, 75*(1), 101–108.

Parvaz, M., MacNamara, A., Goldstein, R., & Hajcak, G. (2012). Event-related frontal alpha as a marker of lateral prefrontal cortex activation during cognitive reappraisal. *Cognitive, Affective, and Behavioral Neuroscience, 12*(4), 730–740.

Phan, K., Liberzon, I., Welsh, R., Britton, J., & Taylor, S. (2003). Habituation of rostral anterior cingulate cortex to repeated emotionally salient pictures. *Neuropsychopharmacology, 28*(7), 1344–1350.

Sabatinelli, D., Keil, A., Frank, D., & Lang, P. (2013). Emotional perception: Correspon-

dence of early and late event-related potentials with cortical and subcortical functional MRI. *Biological Psychology, 92*(3), 513–519.

Sabatinelli, D., Lang, P., Keil, A., & Bradley, M. (2007). Emotional perception: Correlation of functional MRI and event-related potentials. *Cerebral Cortex, 17*(5), 1085–1091.

Sheppes, G., & Gross, J. J. (2011). Is timing everything?: Temporal considerations in emotion regulation. *Personality and Social Psychology Review, 15*(4), 319–331.

Stockburger, J., Schmälzle, R., Flaisch, T., Bublatzky, F., & Schupp, H. T. (2009). The impact of hunger on food cue processing: An event-related brain potential study. *NeuroImage, 47*(4), 1819–1829.

Tacikowski, P., & Nowicka, A. (2010). Allocation of attention to self-name and self-face: An ERP study. *Biological Psychology, 84*(2), 318–324.

Thiruchselvam, R., Blechert, J., Sheppes, G., Rydstrom, A., & Gross, J. J. (2011). The temporal dynamics of emotion regulation: An EEG study of distraction and reappraisal. *Biological Psychology, 87*(1), 84–92.

Thiruchselvam, R., Hajcak, G., & Gross, J. J. (2012). Looking inwards: Shifting attention within working memory representations alters emotional responses. *Psychological Science, 23*(12), 1461–1466.

Urry, H. L. (2010). Seeing, thinking, and feeling: Emotion-regulating effects of gaze-directed cognitive reappraisal. *Emotion, 10*(1), 125–135.

Versace, F., Lam, C. Y., Engelmann, J. M., Robinson, J. D., Minnix, J. A., Brown, V. L., et al. (2012). Beyond cue reactivity: Blunted brain responses to pleasant stimuli predict long-term smoking abstinence. *Addiction Biology, 17*(6), 991–1000.

Vuilleumier, P. (2005). How brains beware: Neural mechanisms of emotional attention. *Trends in Cognitive Sciences, 9*(12), 585–594.

Wager, T. D., Davidson, M. L., Hughes, B. L., Lindquist, M. A., & Ochsner, K. N. (2008). Prefrontal-subcortical pathways mediating successful emotion regulation. *Neuron, 59*(6), 1037–1050.

Weinberg, A., & Hajcak, G. (2010). Beyond good and evil: The time-course of neural activity elicited by specific picture content. *Emotion, 10*, 767–782.

Weinberg, A., & Hajcak, G. (2011a). Electrocortical evidence for vigilance-avoidance in Generalized Anxiety Disorder. *Psychophysiology, 48*, 842–851.

Weinberg, A., & Hajcak, G. (2011b). The Late Positive Potential predicts subsequent interference with target processing. *Journal of Cognitive Neuroscience, 23*, 2995–3007.

Weinberg, A., Hilgard, J., Bartholow, B., & Hajcak, G. (2012). Emotional targets: Evaluative categorization as a function of context and content. *International Journal of Psychophysiology, 84*, 149–154.

Yen, N. S., Chen, K. H., & Liu, E. H. (2010). Emotional modulation of the late positive potential (LPP) generalizes to Chinese individuals. *International Journal of Psychophysiology, 75*(3), 319–325.

CHAPTER 4

The Neural Basis of Emotion Dysregulation

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Humans have long had an ambivalent relationship with their emotions. Emotions have been celebrated as the inspiration for pinnacles of human art, literature, and music. Equally, however, emotions have often been seen as "evil spirits" that misguide us and tempt us to do things we should not—as "primal" instincts that need to be conquered by the rational human mind. Indeed, history is replete with fables of the terrible consequences of unchecked emotion, from the tragedies of Shakespeare and his recurring theme of "the fatalism of overmastering passion" (Corson, 1890, Preface) to the appalling crowd violence that can occur anywhere, from the streets of war-torn Baghdad to the neighborhoods of London.

The uncertainty with which we view our emotions reflects their function, both through our evolutionary past and in day-to-day life. Emotions allow us to respond to important environmental changes quickly and with a minimum of deliberation, and provide us with the drive to seek out things that are good for us and avoid things that are harmful. Emotion and mood[1] can be seen as arising from heuristic neural systems that act upon a vast quantity of incoming information and make speedy decisions, guiding behavior in a way that is robust against incomplete information or computational overload. Emotions are vital for quick, adaptive responses in the face of complexity that rules out a rational calculation of all our options but afford us far more flexibility than simple stimulus–response rules.

Emotion Regulation and Dysregulation

Emotions are not always "correct," based as they are on probabilistic systems that have evolved to ensure our survival across a wide range of circumstances. One of the functions emotions serve is to interrupt ongoing behavior when an encountered event or stimulus is highly personally significant and deserves our attention or requires us to prepare for action (Frijda, 1986; Gross, this volume). Yet we cannot afford to be constantly interrupted; emotions exist in a balance with other, ongoing cognitive, attentional, and behavioral processes. In order for us to benefit, emotions need to be appropriately managed depending on the specific contexts in which they occur. Given the importance of emotion regulation, it is unsurprising that neural systems have been identified that serve to manage emotions and moods in such a way that, on the balance, they contribute to, rather than interfere with, our daily lives (Ochsner & Gross, this volume).

In this chapter we consider how the dysfunction of these neural systems might underlie the inability to regulate emotions appropriately. The outcome of such a failure to regulate can range from a short-term inability to stop getting angry during a debate with a colleague, all the way to long-term psychopathology such as mood and anxiety disorders. We limit discussion to types of emotion dysregulation that are in some sense not normative. Thus, we do not discuss mechanisms that give rise to panic behavior when a bomb explodes, even though remaining calm might be the most adaptive thing to do in the context, since panic in such a situation would be considered quite normal. We also take a look at how knowledge of the neural systems involved in emotion regulation, and their dysfunction in psychopathology, is leading to novel, brain-focused clinical treatments.

Emotion regulation processes can be examined at multiple levels, from neurotransmitters and inhibitory interneurons to cortical and subcortical network feedback loops, to socially mediated regulation of moods and emotional behaviors. All of these types of regulation may be at least partially automatic and subconscious (Gyurak & Etkin, this volume), so limiting a discussion of emotion regulation to that which is "deliberate" or "conscious" is not particularly useful. Gross (this volume) makes the distinction between antecedent-focused and response-focused emotion regulation. Neurobiologically, this conceptual distinction can be elaborated by considering the neural circuits involved in detecting and appraising situations and contexts that call for certain emotional responses (e.g., threat detection, relevance detection, recognition of socially rewarding stimuli, evaluation of possible outcomes and their probabilities), as well as those responsible for generating the neural, physiological, and behavioral responses that make up emotions. Many of the brain processes involved in emotion regulation have thus been extensively studied in other domains of psychology and cognitive neuroscience, including (but not limited to) selective attention, cognitive control, working memory, and response inhibition. Effective emotion regulation involves the coordinated recruitment of these different neural mechanisms in specific contexts (Ochsner & Gross, this volume). One important question is the extent to which different manifestations of emotion dysregulation can be understood in terms of specific patterns of dysfunction in one or more of these neural systems.

Prefrontal Cortex and Emotion Regulation

Given the broad sweep of processes we just outlined, an exhaustive review of the role of different neural systems in emotion regulation and dysregulation would require an entire book. Instead, we focus here on research demonstrating the involvement of the prefrontal cortex (PFC) in the regulation of "bottom-up" emotion-generating neural circuits, which has provided the clearest empirical distinction between generation and regulation of emotions, and also potentially has the greatest implications for emotion regulation–based therapies for a variety of affective disorders (see Figure 4.1; also see Ochsner & Gross, this volume). Research on prefrontal cortical regulation of emotion has largely followed one of two approaches. Studies of cognitive reappraisal have focused on how the explicit cognitive reinterpretation of the emotional meaning or possible outcome of a stimulus can be used to modify the resulting emotional response. On a more automatic level, a range of studies has examined the more automatic engagement of prefrontal mechanisms that serve to regulate attention and the contents of working memory, and to shield these processes from emotional interference.

Cognitive Reappraisal

Based on cognitive appraisal theories of emotion (Scherer, Schorr, & Johnstone, 2001), the study of emotion regulation through reappraisal has formed the core of cognitive neuroscience research on emotion regulation (Gross, this volume). Reappraising the affective meaning of negative emotional stimuli can reduce the magnitude of felt negative emotion, as well as physiological indicators, such as potentiated eyeblink startle (Jackson, Malmstadt, Larson, & Davidson, 2000). Eyeblink startle increases in magnitude during presentation of negative pictures, relative to neutral pictures, an effect thought to

FIGURE 4.1. Depiction of prefrontal cortical and subcortical regions commonly linked to emotion regulation. dmPFC, dorsomedial PFC; dACC, dorsal anterior cingulate cortex; vmPFC, ventromedial PFC; NAc: nucleus accumbens; Amyg, amygdala; vlPFC, ventrolateral PFC; dlPFC, dorsolateral PFC.

be mediated by projections from the central nucleus of the amygdala to the brainstem (Davis, 2006). Evidence that reappraisal can directly influence this amygdala circuitry comes from consistent findings in positron emission tomographic (PET) and functional magnetic resonance imaging (fMRI) studies of healthy individuals showing reappraisal-dependent decreases in amygdala activation in response to negative stimuli. Increased dorsolateral and ventrolateral PFC activation is commonly measured in such reappraisal studies (Ochsner & Gross, this volume), though discrepancies such as the hemispheric lateralization of PFC activation still exist, with some studies finding predominantly right prefrontal activation and others finding left prefrontal activation.[2]

Spontaneous Regulation and Cognitive Control

In daily life we need to regulate our emotions continuously and automatically in order to remain focused on current thoughts and actions; we do not always have the luxury of being able to reappraise consciously the emotion-inducing events around us. A number of studies have used tasks in which regulating emotion is required but not explicitly instructed. For example, when cognitive tasks, such as those that tax working memory, are performed under an anxiety induction or in the presence of emotional distractors, a drop in task performance is observed. There is some evidence that this interference is reduced when the

cognitive load of the task increases, possibly through the automatic engagement of top-down control mechanisms (Blair et al., 2007; Clarke & Johnstone, 2013; Erthal et al., 2005; Van Dillen, Heslenfeld, & Koole, 2009). The specific subregions of the PFC that are involved in such automatic emotion regulation may depend on properties of the distractors, for example, the relative involvement of ventrolateral or dorsal PFC in regulating negative or positive distractors, respectively (Erk, Kleczar, & Walter, 2007). A number of the prefrontal regions implicated in either spontaneous or explicit emotion regulation, including the ventrolateral PFC, dorsolateral PFC, and the dorsal anterior cingulate, overlap with those commonly identified in studies of cognitive and attentional control (Mitchell, 2011). The dorsal anterior cingulate cortex (dACC) is posited to be involved in performance monitoring and detecting when control is necessary (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). Lateral regions of the PFC, including the ventrolateral and dorsolateral PFC, are involved in maintaining and manipulating information in working memory, and in implementing attentional, cognitive, or behavioral adjustments (Ridderinkhof et al., 2004).

Prefrontal–Subcortical Connectivity

There are few direct neural connections between dorsal and lateral PFC and the amygdala (Ghashghaei, Hilgetag, & Barbas, 2007), raising the question of how these lateral PFC brain regions might exert their top-down regulatory influence. One possibility is that the ventromedial PFC (vmPFC), which has neural connections with lateral PFC and the amygdala, might provide the missing link. Ventral parts of the medial PFC have been implicated in down-regulation of amygdala function in nonhuman and human studies of fear conditioning and extinction. Extinction of conditioned fear responses can be considered a form of emotion regulation, since the fear association itself is not destroyed (Bouton, 2004), but rather a new association indicating safety is created and inhibits or overcomes the original conditioned response in specific contexts. In rodent studies, the infralimbic cortex (homologous to the vmPFC in humans) plays an important role in the creation and maintenance and/or recall of extinction of conditioned fear responses (Milad & Quirk, 2002, 2012). Consistent with these studies, activation in the vmPFC correlates with recall of extinction in humans (Kalisch et al., 2006; Milad et al., 2007; Phelps, Delgado, Nearing, & LeDoux, 2004). The role of vmPFC–amygdala connectivity seems to be under genetic control, pointing to a role of dynorphins in human extinction learning (Bilkei-Gorzo et al., 2012). Thus, based on its role in fear conditioning and extinction paradigms, the vmPFC would appear to be a promising candidate as a region that can mediate more lateral PFC regulatory effects on the amygdala.

Evidence that this might be the case comes from studies of spontaneous regulation, as well as reappraisal-based down-regulation of emotional responses to aversive pictures. For example, Etkin, Egner, Peraza, Kandel, and Hirsch (2006) used a variant of an emotional Stroop task, in which participants responded to the words *fear* and *happy* superimposed on faces expressing either fear or happiness. In this task, there is a need to down-regulate automatic responses to the emotional facial expressions when they conflict with the word (e.g., the word *happy* superimposed on a fearful face). Their results indicated that while dorsal and lateral PFC detect the need for top-down regulation, rostral ACC (rACC) implements that regulation. Specifically, rACC activation was higher and amygdala activation was lower during the down-regulation of emotionally conflicting information, with a negative correlation between the two regions. Two studies of reappraisal-based emotion regulation, (Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007; Urry et al., 2006) found that individual differences in activation of the vmPFC during down-regulation correlated negatively with amygdala activation; the more activated the vmPFC during down-regulation, the less activated the amygdala. Furthermore, vmPFC activation was found statistically to mediate the association between dorsomedial PFC (Urry et al., 2006) ventrolateral PFC (Johnstone et al., 2007) or dorsolateral PFC (Erk et al., 2010) and amygdala activation during the down-regulation condition. It should be noted, however, that these studies did not find cat-

egorically increased activation of vmPFC during down-regulation.

One study (Delgado, Nearing, LeDoux, & Phelps, 2008) directly tested the connection between reappraisal-based emotion regulation and extinction. Participants were instructed simply to attend to a colored square that might give them a shock (the conditioned stimulus) or to down-regulate their conditioned response to the square by reappraising the meaning of the color (e.g., by thinking of contexts in which the color had a more calming influence, such as the sky for a blue square). Greater activation in the down-regulation relative to the attend condition was measured in dorsolateral PFC and vmPFC, with less activation in the amygdala. Prefrontal activation correlated negatively with skin conductance responses, providing evidence of its role in regulating emotional responses. Notably, the region of activation in vmPFC overlapped with that found in prior studies of extinction using the same stimuli.

In summary, evidence points to a prefrontal regulatory network in which dorsal and lateral regions of the PFC exert a top-down influence on subcortical structures involved in generating emotional responses. Some evidence suggests that this regulatory effect is via the same region of vmPFC involved in extinction, though whether this is the primary pathway, or other important regulatory pathways exist (Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008) is the topic of continuing investigation (e.g. see Clarke & Johnstone [2013] for an examination of the role of dACC in inhibition of amygdala output to PFC during spontaneous threat regulation). Identifying the exact pathways involved in effective emotion regulation will be vital in determining the extent to which the dysregulation of emotion can be explained in terms of a malfunction in the prefrontal emotion regulation network.

Emotion Dysregulation and Affective Disorders

Emotional disorders such as depression and anxiety are estimated by the World Health Organization to be the greatest cause of disability worldwide (Mathers, Lopez, & Murray, 2006). Efforts to understand the neurobiological causes of these conditions and potential avenues for treatment are therefore of enormous importance to the general population. Most brain research into mood and anxiety disorders has focused on the "bottom-up" determinants of emotions, with a focus on subcortical and early sensory processing that generates emotional responses and biases our behavior in a fairly automatic way. For example, much of the focus in anxiety disorders has been on attentional biases toward anxiety-relevant stimuli (Campbell-Sills, Ellard, & Barlow, this volume; MacLeod, Mathews, & Tata, 1986), generated either through low-level sensory cortex or subcortical structures such as the amygdala, known to play a role in early threat detection. In depression, an extensive literature exists on the role of the monoaminergic neurotransmitters serotonin, norepinephrine, and dopamine in subcortical and subcortical–cortical networks involved in mood, though for all that research, there have been no definitive breakthroughs in understanding the causes of depression.

Although negative emotions such as anxiety and sadness are a normal occurrence in response to adversity in all individuals, one feature that distinguishes those with mood and anxiety disorders is their inability to regulate certain negative emotions effectively when they arise. Indeed, it might well be argued that the defining feature of many mood and anxiety disorders is the extended duration of emotional episodes rather than their intensity (phobias and panic disorders are obvious exceptions, though even in these cases, feelings of panic and anxiety typically last well beyond the immediate trigger of the episode). Cognitive neuroscientists have argued that depression should not be equated with the "down" state itself, but rather with the tendency to "enter and get stuck in this state. Thus, the neurobiology of depression should be that of mood reaction and regulation rather than the mood state per se" (Holtzheimer & Mayberg, 2011, p. 1). Importantly, it is not just negative emotions that are dysregulated in mood disorders. "Anhedonia," a markedly reduced interest and pleasure in normally rewarding activities, is one of the three core features of major depression that may be partly explained by an inability to engage cognitively in rewarding situations and sus-

tain positive emotions, a different type of emotion regulation.

Although abnormalities in the neural circuitry supporting adaptive regulation of certain emotions may play a decisive role in determining vulnerability to mood disorders (Davidson, Pizzagalli, Nitschke, & Putnam, 2002), only recently have researchers explicitly examined this possibility. Below we discuss the findings separately for anxiety, which is symptomatic of a variety of disorders and phobias; the more general negative mood characteristic of depression; and the dysregulation of positive emotions in anhedonia.

Emotion Dysregulation and Anxiety

Substantial research has demonstrated that high-anxious individuals show greater attentional capture by threat-relevant information, with slowed reaction times and an increased number of errors when performing tasks such as the emotional Stroop or the dot-probe task, than low-anxious individuals (MacLeod et al., 1986). A number of models of anxiety-related attentional bias have proposed that an overly sensitive preattentive threat detection system is responsible for drawing attention toward threatening stimuli (see Cisler & Koster, 2010, for a review). In line with this, elevated amygdala activation in response to threat-relevant information is often measured in high-anxiety individuals, suggesting that attentional biases may have their roots in hyperactive or low-threshold threat detection at a subcortical level. Adults assessed as having an inhibited temperament as infants show heightened and sustained amygdala responses to novel social stimuli (Schwartz & Rauch, 2004; Schwartz, Wright, Shin, Kagan, & Rauch, 2003), suggesting that such amygdala hyperactivity might be stable across development. Heightened amygdala activation is not always found in high-anxiety individuals, however, in part because amygdala engagement may depend on the specific amygdala nucleus and properties of the threatening stimulus (Etkin et al., 2004). There are also other candidate subcortical regions that might underlie overly sensitive threat detection in anxious individuals, such as the bed nucleus of the stria terminalis (BNST), which plays an important role in sustained, as distinct from transient, forms of vigilance (Somerville et al., 2012; Walker, Toufexis, & Davis, 2003).

Nonetheless, it seems likely that anxiety disorders involve more than just hypersensitivity in bottom-up threat detection systems. There is now a large body of evidence that highly anxious individuals, in particular those with anxiety disorders, show deficits in prefrontal circuits that regulate attention to and responding to threatening stimuli. Over and above an attentional bias *toward* threatening information, highly anxious individuals have greater difficulty in *disengaging* from threat-relevant information when required. In Posner spatial cuing tasks (Posner, Snyder, & Davidson, 1980), the participant focuses on a central fixation, and a cue flashed up in one of two locations on either side of the fixation point is followed rapidly by a target in one of the two locations. When the cue is presented in a different location from the following target (invalid trials), response to the target is slowed. Highly anxious individuals show even greater slowing when the cue is threat-related than when it is of neutral valence, an effect that is reliable across multiple clinical anxiety disorders and high trait anxiety populations, but not in nonanxious individuals (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007). Results such as this indicate that weakened top-down attentional control, a core feature of spontaneous emotion regulation, might underpin some features of high trait anxiety and anxiety disorders (Cisler & Koster, 2010). Deficits in the performance of working memory tasks in the presence of sustained emotional distractors or anxiety induction in anxious individuals (Shackman et al., 2006) also point to weakened top-down control of emotion, potentially involving the prefrontal network for cognitive control and spontaneous emotion regulation discussed earlier.

Human brain imaging studies have found reduced prefrontal recruitment in high-anxious individuals relative to low-anxious individuals when faced with threat or threat-relevant stimuli. For example, using a perceptual discrimination task in the presence of fearful versus neutral facial expressions, Bishop, Duncan, Brett, and Lawrence (2004) found lower rACC and ventrolateral

PFC activation in high-anxious than in low-anxious individuals, and the opposite pattern in the amygdala. This result is consistent with a model in which rACC detects the need for control over incoming information that conflicts with task demands, and ventrolateral PFC is subsequently involved in regulating attention toward task-relevant information and away from threatening, task-irrelevant information. The lower rACC and ventrolateral PFC activation and higher amygdala activation seen in high-anxious individuals presumably reflects reduced prefrontal control. This deficit in top-down attentional control of task-irrelevant distractors might not be specific to threatening or even emotional distractors: In a later study, Bishop (2008) reported that high trait anxious individuals showed greater interference from nonemotional distractors, along with reduced activation in dorsolateral PFC. Thus elevated trait anxiety is associated with generally reduced lateral PFC-mediated top-down control of attention, resulting in less efficient attentional regulation of distracting emotional information. Coupled with a hypersensitive bottom-up threat detection system, the consequence would be hypervigilance to and an inability to disengage from potentially threatening stimuli.

The picture of dysfunctional prefrontal regulation in anxiety disorders (as opposed to high trait anxiety) is not as clear-cut as one might presume from this model, however. The patterns of observed prefrontal hypoactivity and hyperactivity differ across anxiety disorders (Etkin & Wager, 2007; Shin & Liberzon, 2010). Of these, the most consistent neuroimaging findings have been reported for posttraumatic stress disorder (PTSD). A number of studies have shown impaired extinction of conditioned responses to aversive stimuli in PTSD, and hypoactivation in vmPFC/rACC has been reported in response to both trauma-related and non-trauma-related aversive stimuli, as well as during extinction, other emotional and cognitive tasks, and resting baseline (Shin & Liberzon, 2010). Activation in this region also negatively correlates with PTSD symptom severity. All of these results suggest that PTSD is characterized by a disrupted ability to regulate previously conditioned fear responses through the engagement of vmPFC extinction circuitry. In contrast with PTSD, neuroimaging studies of other anxiety disorders have shown inconsistent results, with both hyper- and hypoactivation in vmPFC, though there are fewer such studies than those examining PTSD, and they have used a variety of different experimental paradigms (Etkin & Wager, 2007).

Dysregulation of Emotion in Negative Mood and Depression

Brain imaging studies of depressed individuals have demonstrated abnormal patterns of activation in many of the same prefrontal brain regions thought to contribute to the regulation of mood and emotion, though the findings have not been consistent. A number of PET studies have indicated reduced metabolism in dorsolateral PFC and dACC in both the resting state and during exposure to negative stimuli (Fitzgerald, Laird, Maller, & Daskalakis, 2008). These results are consistent with the notion that dysregulation of negative mood in depression might be due to underrecruitment of dorsal prefrontal brain regions important for the regulation of negative emotions, yet the fact that these studies did not directly measure emotion regulation limits the inferences that can be made.

Some PET resting state studies have also reported elevated metabolism in depressed versus nondepressed individuals in ventral regions of the PFC, including ventrolateral and orbitofrontal cortex (Savitz & Drevets, 2009), which is more difficult to reconcile with a deficit in emotion regulation. It should be noted though that this region of the PFC serves a more general role in emotion processing than its posited role in top-down emotion regulation, including flexible updating of stimulus value, as well as monitoring of the autonomic state (Rolls, 2004). In addition to top-down projections to the amygdala, it also receives excitatory input from both the amygdala and sensory cortices. In the absence of top-down regulatory signals, then, hyperactivity in ventral PFC in depressed individuals might reflect more bottom-up-driven elaborative or ruminative processing of emotional information.

A number of fMRI studies have measured prefrontal cortical activation during instructed emotion regulation tasks. Beauregard, Paquette, and Levesque (2006)

reported increased dorsal ACC activation in depressed compared with healthy individuals when they were decreasing emotional responses to sad films. No group differences were found in the more anterior and lateral PFC regions found previously in studies of reappraisal in nondepressed samples. In a study of reappraisal of negatively valenced emotional pictures, Johnstone et al. (2007) found that healthy individuals showed left-lateralized activation of ventrolateral PFC, whereas depressed individuals showed bilateral activation. In the healthy group, vmPFC activation was negatively correlated with amygdala activation and found statistically to mediate a negative association between ventrolateral PFC and the amygdala, consistent with a top-down regulation pathway from ventrolateral PFC via vmPFC to the amygdala. However, for the depressed group, a positive correlation was found between vmPFC and amygdala activation, with no correlation between lateral PFC and amygdala. One possible explanation for this finding is that in depressed individuals, the lack of regulatory input from left dorsal or lateral prefrontal regions results in sustained vmPFC activation, under either greater bottom-up or right lateral PFC influence. Both explanations would be consistent with the elevated vmPFC metabolism in depression seen in some PET studies, perhaps reflecting greater self-referential processing.

Results consistent with a model of reduced lateral PFC top-down regulation of amygdala in depression have now been seen in a number of emotion regulation studies, although lateralization of this reduced PFC involvement has not been consistently found across studies. In a study with partially remitted depressed patients, Erk et al. (2010) demonstrated that although depressed patients are able to down-regulate their amygdala using distancing, a form of reappraisal, the degree of down-regulation was diminished with increasing scores of depression (i.e., the more depressed the patients were, the less they were able to down-regulate the amygdala). This could be explained by reduced activation of the right dorsolateral PFC, which also showed significantly reduced coupling with the amygdala in depressed patients.

Erk et al. (2010) also showed differences between healthy and depressed individuals in *sustained* effects of emotion regulation. In healthy controls, regulation effects on the amygdala could still be demonstrated after the emotion regulation task; negative pictures that had been paired with a down-regulation instruction invoked not only reduced amygdala activation compared to an attend condition during the initial emotion regulation task but also reduced amygdala activation in a simple picture-watching task 15 minutes later. Furthermore, the degree of reduction in the second task was correlated with dorsolateral PFC activation in the first task. In contrast, depressed patients had no such lasting effect of emotion regulation; activation in response to previously down-regulated pictures was no different than that in response to nonregulated pictures. Together, these findings suggest that depression is characterized not by the negative mood itself but by the tendency to get stuck in that mood (Holtzheimer & Mayberg, 2011).

Kanske, Heissler, Schönfelder, and Wessa (2012) recently described how remitted patients with a diagnosis of depression show a deficit in down-regulation of the amygdala when reappraising socially negative pictures but not when using a distraction strategy. Additionally, they found that down-regulation of the amygdala was better in those patients who habitually used reappraisal strategies, as measured with the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003). This finding extends the work of Abler, Erk, Herwig, and Walter (2007), whose examination of patients with concurrent depression demonstrated that those who had higher reappraisal scores, as measured with the ERQ, showed less anticipatory amygdala activation to negative stimuli. Kanske et al.'s study (2012) shows that neurobiological deficits during reappraisal extend into remission, pointing to the possibility that impaired emotion regulation of negative stimuli might be a trait marker of depression, as suggested previously in behavioral studies (Ehring, Fischer, Schnülle, Bösterling, & Tuschen-Caffier, 2008).

Dysregulation of Positive Emotion: Anhedonia

The dysregulation of emotion in depression is not limited to negative mood and emo-

tions. One of the core features of depression is *anhedonia*, a deficit in the experience of positive emotions and the ability to take pleasure from previously rewarding stimuli. Anhedonia is a particularly debilitating condition, because it removes the pleasurable experiences that motivate much of what we do in life. The neural basis of anhedonia is not well understood, but it is likely that it involves the frontostriatal network, which includes connected regions of the striatum, particularly the ventral striatum,[3] and the PFC. Based on extensive animal and human research, the frontostriatal network is regarded as a core system for the learning of reward signals, the experience of pleasure, and the motivation of reward-seeking behaviors. Despite its role in positive emotion, however, research linking the frontostriatal network to anhedonia has been mixed, with some studies reporting reduced activity in the striatum (Epstein et al., 2006; Keedwell, Andrew, Williams, Brammer, & Phillips, 2005) and others showing no such effect (Knutson, Bhanji, Cooney, Atlas, & Gotlib, 2008; Schaefer, Putnam, Benca, & Davidson, 2006).

One explanation for these inconsistencies might be the model of anhedonia being tested. Anhedonia has often been considered to reflect a tonically dampened positive emotion system, in which the general capacity to experience pleasure is reduced (Meehl, 1975). An alternative hypothesis is that an inability to prolong the duration of positive emotion, or increase the intensity of a rewarding experience—both aspects of emotion regulation—might underlie at least some anhedonic symptoms (Tomarken & Keener, 1998). The ventral striatum has bidirectional connections with a number of prefrontal cortical regions, and brain imaging studies have shown top-down modulation of ventral striatal activation in humans (Delgado, Gillis, & Phelps, 2008; Staudinger, Erk, & Walter, 2011).

Compared with the regulation of emotional responses to negative stimuli, there have been very few studies of top-down regulation of positive emotions. Kim and Hamann (2007) showed that regulation of positive emotion engaged prefrontal regions partially overlapping those involved in regulating negative emotions, and that subcortical (e.g., amygdala) effects of regulating positive emotions were more pronounced than those for negative emotions. In a recent study of up-regulation of positive emotion in response to pleasant pictures, Heller et al. (2009) found that, compared with controls, patients with depression were unable to sustain activation in the ventral striatum across an experimental session, and that the degree to which ventral striatal activity was sustained correlated with self-reported positive emotion, as well as functional connectivity between the ventral striatum and a region in lateral PFC. In a follow-up study on the same patients after 8 weeks of antidepressant treatment, Heller et al. (2013) found that those who demonstrated the greatest increase in their ability to sustain ventral striatal activation and frontostriatal connectivity while up-regulating positive emotion also showed the largest increases in positive emotion. It is worth noting that this study only involved antidepressant treatment, and it is unclear whether those showing greatest symptom improvement did so as a consequence of increased frontostriatal engagement. Future studies that examine the neural impact of cognitive-behavioral therapies on regulation of positive emotions will shed further light on the causes and possible treatments for anhedonia. In an example of neurally guided emotion regulation therapy, a recently published proof of concept study, Linden et al. (2012) used neurofeedback with fMRI to target this problem in depression. Eight patients with depression learned to increase activation in brain regions thought to be related to positive emotions, such as the insula and the vMPFC, during four neurofeedback sessions. Their depressive symptoms were reduced significantly, whereas a control group that underwent a training procedure with the same cognitive strategies, but without neurofeedback, did not show clinical improvement.

Genes, Development, and Emotion Regulation

Although much has been learned in recent years about the neural underpinnings of disorders of emotion regulation, we are a long way from understanding the enormous individual differences in our ability to regulate our moods and emotions. What are the

causes of these individual differences? To what extent do they reflect innate biological differences? Are there aspects of development that render some individuals more at risk to psychopathology related to emotion dysregulation?

It is well known that individual differences in emotional experience and regulation are modulated by differences in genetic makeup. One prominent example is genetic variation within the promoter region of the serotonin transporter gene (*5-HTTLPR*) in which the long variant has been associated with anxiety and neuroticism, as well as with depression, particularly in combination with stressful life events (Karg, Burmeister, Shedden, & Sen, 2011). Hariri, Mattay, Tessitore, Fera, and Weinberger (2003) showed that the short variant of the *5-HTTLPR* is also associated with increased activation of the amygdala in reaction to aversive stimuli, a finding that has been replicated quite consistently (Caspi, Hariri, Holmes, Uher, & Moffitt, 2010).

Indirect evidence suggesting that genetic differences in *5-HTTLPR* might be associated with regulation of emotion came from findings that short allele carriers (s-carriers) show decreased connectivity of amygdala and the perigenual cingulate (a ventral prefrontal region previously implicated in extinction of aversive conditioning), and that the lower this connectivity was in s-carriers, the higher their anxious temperament (Pezawas et al., 2005). Heinz et al. (2005) found greater vmPFC–amygdala coupling in s-carriers, however, a result that is difficult to reconcile with the Pezewas et al. (2005) results, although the locus of prefrontal activation was different. Another study using a sad mood induction and arterial spin-labeling brain imaging (which measures blood perfusion) showed increased amygdala activation in s-carriers not during sad mood induction but during recovery, in which emotion regulatory processes can be assumed (Gillihan et al., 2010).

More direct evidence of the possible links between genetic variation in the *5-HTTLPR* and emotion regulation comes from studies that used cognitive reappraisal tasks. Schardt et al. (2010) found that, consistent with prior studies, amygdala activation during a passive viewing condition was increased in s-carriers. However, during active emotion regulation there were no differences in amygdala activation. Moreover, it was shown that subjects with an s-allele showed higher coupling of right amygdala with the vmPFC when they regulated their emotions. Although this appears to be counterintuitive, it has to be noted that prior studies investigating amygdala reactivity in s-carriers used passive viewing conditions. It seems that although s-carriers have an increased reactivity to passive stimulation, they can compensate with active regulation, possibly mediated by increased connectivity of vmPFC and amygdala relative to long-allele carriers (l-carriers). This is consistent with a study in which Lemogne et al. (2011) found decreased amygdala activation and increased connectivity of right amygdala and subgenual ACC in s-carriers when they compared labeling of affective pictures (a simple form of emotion regulation) with self-referential processing of those pictures. A study by Friedel et al. (2009) has demonstrated increased prefrontal activation and PFC–amygdala connectivity in healthy s-carriers, but no such effects in s-carriers with depression, indicating that the ability to regulate negative emotion may be a protective factor against elevated risks associated with the short allele. The question then arises as to what enables some individuals, but not others, to compensate in this way. As a cautionary note, it also should be mentioned that within the long allele there exists a rare variant (with an A to G exchange) that behaves like a short allele with respect to transcription of *5-HTTLPR*. This was only taken into account by some researchers (e.g., Friedel et al., 2009), so that results of other studies have to be taken as preliminary.

Whereas it is clear that some aspects of emotional reactivity might be tied to innate individual differences in temperament, our ability to regulate emotions flexibly and appropriately is something that develops over time. Emotion regulation draws on a range of cognitive and attentional skills and strategies that have differing developmental profiles. In addition, the prefrontal brain regions that have consistently been associated with emotion regulation are among the latest parts of the brain to mature fully. Accumulating evidence from brain imaging studies points to differential engagement of prefrontal brain regions in children and ado-

lescents compared to adults when required to modulate attention to emotional stimuli (Monk et al., 2003; Nelson et al., 2003) or when using explicit strategies to regulate emotions (Levesque et al., 2004). Some researchers point to the mismatch between development of subcortical brain regions involved in emotion generation and prefrontal regions exerting top-down control as key to understanding emotion dysregulation in children and adolescents (Casey, Tottenham, Liston, & Durston, 2005). Because the PFC and its connections with subcortical limbic structures continue to mature from childhood through adolescence into early adulthood, there is a particular risk that maladaptive prefrontal–limbic connectivity and associated dysregulation of emotion will develop, rendering individuals vulnerable to psychopathology later in life.

Very few researchers have addressed the neurobiology of childhood or adolescent emotion regulation. Perlman et al. (2012) examined emotion regulation in 14 depressed adolescents (ages 13–17 years). Compared to an age-matched healthy control group, the depressed group showed increased amygdala activation and less connectivity between amygdala and medial PFC/insula during the control condition. Frontal–amygdala connectivity was correlated with psychosocial function, hinting that those depressed adolescents with less intact PFC–amygdala regulatory circuitry were more severely affected. No differences between the depressed and nondepressed groups were seen during the active regulation condition, however, which might indicate that active emotion regulation via cognitive reappraisal is able to overcome differences in baseline emotion reactivity, at least in the short term, when explicitly prompted.

In a study of adult patients with depression, Frodl, Reinhold, Koutsouleris, Reiser, and Meisenzahl (2010) have shown that previous childhood stress is associated with reductions in prefrontal gray matter, and that prefrontal gray matter volume and childhood neglect interact to predict adult depression duration. A recent study (Burghy et al., 2012) showed that elevated cortisol levels associated with early life stress was predictive of reduced vmPFC-amygdala resting state functional connectivity in females. Furthermore, vmPFC-amygdala resting state connectivity was inversely correlated with anxiety symptoms but positively correlated with depressive symptoms in female adolescents, pointing to one possible factor in the development of PFC-subcortical emotion regulation circuitry. The developmental neurobiological profile of individual differences in emotion regulation has barely been studied, yet it is likely to have far-reaching consequences for our understanding of emotion dysregulation throughout life.

Cortical Brain Stimulation as a Treatment for Depression

Up to now we have considered psychological self-regulation of emotion. However, it is also possible to regulate emotions using technical devices that alter activity in brain regions that are involved in emotion regulation. These are particularly important when dysregulation of emotion is present as a pathological condition. Three techniques of brain stimulation—repetitive transcranial magnetic stimulation (rTMS), a closely related technique transcranial direct current stimulation (tDCS), and deep brain stimulation (DBS)—have recently been proposed as methods offering a viable treatment for otherwise untreatable depression.

Repetitive Transcranial Magnetic Stimulation

rTMS involves generating trains of brief bursts of a strong magnetic field close to the scalp. When the magnetic field is oriented perpendicular to the cortical surface, electric currents are induced in the underlying neural tissue to a depth of several millimeters (a consequence of Faraday's law of electromagnetic induction). While the exact mechanism of action of rTMS on networks of neurons is not yet fully understood, different frequencies of rTMS can have either short- or long-term facilitatory or disruptive effects on cortical processing (Fitzgerald, Fountain, & Daskalakis, 2006). In general, low-frequency (< 1 Hz) rTMS is thought to inhibit cortical processing, whereas high-frequency (> 5 Hz) is thought to enhance cortical processing. Supporting evidence for this model of rTMS effects on cortical function comes from PET studies following

rTMS to lateral PFC, which show greater blood flow and glucose metabolism following high-frequency rTMS and reduced metabolism following low-frequency rTMS (Speer et al., 2000). It is also possible that rTMS can affect the neurotransmitter systems that have been linked to depression. In rodent models, altered dopaminergic neurotransmission in the striatum and altered serotoninergic neurotransmission the amygdala and cortex have also been observed following rTMS, though there are concerns about the applicability of these results to humans for methodological and anatomical reasons (Padberg & George, 2009).

For treatment of depression, rTMS has typically been applied to dorsolateral PFC—sometimes as low-frequency rTMS to right dorsolateral PFC to inhibit cortical processing, but most often as high-frequency rTMS to left dorsolateral PFC to enhance processing. Although results have been mixed, three recent meta-analytic reviews have concluded that high-frequency rTMS applied to left dorsolateral PFC is an effective treatment for depression. Schutter (2009) analyzed 30 double-blind sham-controlled high-frequency rTMS studies, and concluded that rTMS was significantly better than sham and produced effects comparable in reliability and magnitude of response to antidepressant drugs. In a meta-analysis of 34 studies, Slotema, Blom, Hoek, and Sommer (2010) also found rTMS to be superior to sham TMS, although the authors concluded that rTMS was not as effective as electroconvulsive treatment (ECT). A meta-analysis by Allan, Herrmann, and Ebmeier (2011) of 31 randomized trials of TMS versus a sham control yielded the same conclusion. Some caution is needed, however, in that most if not all the studies have not been truly blind (i.e., the experimenter applying the stimulation or sham knows which condition is being applied) and sham TMS is perceptually quite different from actual TMS.

Transcranial Direct Current Stimulation

Although first applied prior to TMS, tDCS has only relatively recently received an upsurge in interest as a treatment for depression. It is easy to apply and, compared to TMS, very inexpensive. tDCS consists of passing a small direct electrical current of 1–2 milliamps (mA) between two electrodes placed on the scalp. Most typically in treatment for depression, the *anode* (the electrode from which electrons flow) is placed on the left dorsolateral PFC, and the *cathode* (the electrode to which electrons flow) is placed on the right PFC. Stimulation is then applied for 10–20 minutes, with repeated treatment sessions daily or on alternate days. Compared with TMS, relatively more is known about how tDCS affects neural activity. When current flows parallel to the axon–dendrite axis of pyramidal cells in the cortex, neurons under the anode become more depolarized, which makes them more excitable and increases neural firing. The opposite occurs under the cathode, with neurons becoming hyperpolarized, which leads to a reduction in firing. Thus, tDCS with an anode placed over the left dorsolateral PFC will tend to increase activity in that region of the brain. The effects of tDCS on cortical excitability can last for a few hours after application, and there is some evidence that tDCS can also produce longer term effects through synaptic changes to glutamate receptors and possible effects on long-term potentiation, though evidence is scarce at present.

Early efforts to use tDCS to treat depression were unconvincing, possibly due to the much smaller currents that were used, as well as the mode and site of electrode placements typically used. More recent studies with greater consistency of target region (left dorsolateral PFC), stimulation strength, and timing have led to more promising results, with a meta-analysis reporting significant improvements in depression symptoms following tDCS versus sham (Allan et al., 2011), though many studies are small and there are fewer overall studies than those using rTMS. Most recently, in a double-blind study, 64 depressed patients showed improvement in mood when tDCS versus sham was used, though the overall percentage of responders did not differ between the two conditions (Loo et al., 2012), highlighting the tentative nature of results to date.

Both rTMS and tDCS have achieved their most promising results for treatment of depression when applied over left dorsolateral PFC in an excitatory mode (e.g., high frequency for rTMS and anode placement

for tDCS). Although the exact mechanism of action for both brain stimulation treatments remains unknown, it is noteworthy that the dorsolateral PFC is consistently activated in brain imaging studies of reappraisal, and plays an important role in working memory and executive control. The implication is that lack of engagement of dorsolateral PFC, specifically left dorsolateral PFC, in depressed patients compromises their ability to regulate their emotions, and that stimulating these areas leads to an improvement in emotion regulation through reappraisal or executive control. Until researchers specifically test the effects of dorsolateral PFC stimulation (either tDCS or rTMS) on regulation of emotion, this hypothesis remains speculative. One possibility to enhance the effect of rTMS or tDCS is to use neuroimaging to determine which hemisphere of the PFC is dysfunctional to direct the stimulation protocol according to these findings (Schönfeldt-Lecuona et al., 2010), possibly using an emotion regulation task. Given that prefrontal tDCS has been shown to improve learning in working memory tasks (Brasil-Neto, 2012) it is conceivable that tDCS could be applied during the training of psychological emotion regulation in order to boost the learning of emotion regulation strategies.

Deep Brain Stimulation

DBS involves the delivery of a small electric current directly to parts of the brain through implanted wire electrodes, with stimulation delivered via an implantable pulse generator. As an invasive surgical procedure, it has so far been investigated as a treatment for the most intractable and debilitating cases of major depressive disorder in a number of small, open-label (i.e., nonblind) studies. The most common site for stimulation is the white matter in the subcallosal cingulate region, chosen on the basis of its connectivity with brain regions thought to be important for antidepressant response (Holtzheimer & Mayberg, 2011; Lozano et al., 2008; Mayberg et al., 2005). These studies have shown remission rates of 40–60% over an extended period (up to several years) in patients with severe depression who have failed to respond to any other treatments. Given the relatively small sample sizes, it is difficult to pinpoint exactly how subcallosal DBS is achieving these promising results, although Holtzheimer and Mayberg (2011) suggest that modulation of a mood regulation network may be the underlying mechanism.

Conclusions

Research on how the human brain creates and regulates our emotions is still in its infancy, but it has already given us remarkable insights into the complexity of the brain systems responsible for shaping our moods and emotions. In many ways human brain imaging studies have validated the notion that the neural systems supporting flexible emotion regulation are a key component of mental health. Knowledge of the role of specific corticolimbic networks in emotion dysregulation and psychopathology is now leading to new experimental treatments involving brain stimulation and neurofeedback. This progress notwithstanding, there are many outstanding questions to be resolved. One central question is the exact role of different subregions of PFC in different types of emotion (dys)regulation. For example, although there is consistent evidence for vmPFC involvement in extinction recall, the exact role of vmPFC in more explicit, cognitive types of emotion regulation such as reappraisal remains unclear. It is possible that this question will be answered not by relatively crude fMRI measures of the *amount* of vmPFC activation but with more nuanced techniques that can capture changes to the way vmPFC encodes the hedonic value of stimuli, perhaps under "direction" from lateral PFC. Multivariate pattern analysis (cf. Davis & Poldrack, 2013), for example, can probe not only how much a brain region is engaged during a task but also the extent to which it represents different types of information. Combining such methods with measures of whole-brain effective and functional connectivity should lead to testable models of emotion regulation networks.

Another unresolved question is how different emotion regulation disturbances map onto different symptoms. In this chapter, we have rather simplistically discussed anxiety disorders and depression separately, even though there is a large amount of comorbidity between these and other disorders. Are

there common or distinct emotion regulation processes involved? To what extent is emotion dysregulation distinct, or does it overlap with more general disturbances in top-down control? To address these fundamental questions will require larger scale studies (including meta-analyses) that combine data from different samples and experimental tasks (e.g., nonemotional tasks involving attentional control, working memory, and response inhibition).

Further research on the impact of genetic variation and early life environment on the development of the brain systems that allow us to regulate our emotions will also be important if we are to develop useful predictors of vulnerability to disorders of emotion regulation. Along similar lines, we know almost nothing about how age-related neural decline affects emotion regulation. Some of the brain regions showing the greatest age-related atrophy, such as lateral PFC, are a key component of emotion regulation circuitry. Do older individuals show the same sorts of age-related decline in emotion regulation as they do in cognitive function? Early evidence suggests that the picture might not be so straightforward (van Reekum et al., 2011; Winecoff, LaBar, Madden, Cabeza, & Huettel, 2011). Far more cross-sectional and longitudinal research relating age, measures of cortical integrity, cognitive function, and emotion regulation capability is required to answer these important questions.

Notes

1. In this chapter we have adopted a liberal consideration of *emotion* and *mood*, using the terms interchangeably. Although these terms might be considered more or less distinct by emotion theorists, the evidence for a clear distinction in terms of neurobiology is lacking. In addition, from a clinical standpoint, mood dysregulation is at least as important as emotion dysregulation.
2. Most studies have not explicitly tested the hemispheric lateralization of prefrontal activation, so caution must be exercised in inferring preferential involvement of one hemisphere in reappraisal of emotional stimuli.
3. Although the term *ventral striatum* is sometimes used interchangeably with *nucleus accumbens*, the latter refers to a specific nucleus, and the former refers to a subregion of the striatum, which might contain a more varied collection of neurons.

References

Abler, B., Erk, S., Herwig, U., & Walter, H. (2007). Anticipation of aversive stimuli activates extended amygdala in unipolar depression. *Journal of Psychiatric Research, 41*(6), 511–522.

Allan, C. L., Herrmann, L. L., & Ebmeier, K. P. (2011). Transcranial magnetic stimulation in the management of mood disorders. *Neuropsychobiology, 64*(3), 163–169.

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*(1), 1–24.

Beauregard, M., Paquette, V., & Levesque, J. (2006). Dysfunction in the neural circuitry of emotional self-regulation in major depressive disorder. *NeuroReport, 17*(8), 843–846.

Bilkei-Gorzo, A., Erk, S., Schurmann, B., Mauer, D., Michel, K., Boecker, H., et al. (2012). Dynorphins regulate fear memory: From mice to men. *Journal of Neuroscience, 32*(27), 9335–9343.

Bishop, S. J. (2008). Trait anxiety and impoverished prefrontal control of attention. *Nature Neuroscience, 12*(1), 92–98.

Bishop, S. J., Duncan, J., Brett, M., & Lawrence, A. D. (2004). Prefrontal cortical function and anxiety: Controlling attention to threat-related stimuli. *Nature Neuroscience, 7*(2), 184–188.

Blair, K. S., Smith, B. W., Mitchell, D. G. V., Morton, J., Vythilingam, M., Pessoa, L., et al. (2007). Modulation of emotion by cognition and cognition by emotion. *NeuroImage, 35*(1), 430–440.

Bouton, M. E. (2004). Context and behavioral processes in extinction. *Learning and Memory, 11*(5), 485–494.

Brasil-Neto, J. P. (2012). Learning, memory, and transcranial direct current stimulation. *Frontiers in Neuropsychiatric Imaging and Stimulation, 3*, 80.

Burghy, C. A., Stodola, D. E., Ruttle, P. L., Molloy, E. K., Armstrong, J. M., Oler, J. A., et al. (2012). Developmental pathways to amygdala–prefrontal function and internaliz-

ing symptoms in adolescence. *Nature Neuroscience, 15*(12), 1736–1741.

Campbell-Sills, L., Ellard, K., & Barlow, D. (this volume, 2014). Emotion regulation in anxiety disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 393–412). New York: Guilford Press.

Casey, B. J., Tottenham, N., Liston, C., & Durston, S. (2005). Imaging the developing brain: What have we learned about cognitive development? *Trends in Cognitive Sciences, 9*(3), 104–110.

Caspi, A., Hariri, A. R., Holmes, A., Uher, R., & Moffitt, T. E. (2010). Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex diseases and traits. *American Journal of Psychiatry, 167*(5), 509–527.

Cisler, J. M., & Koster, E. H. W. (2010). Mechanisms of attentional biases towards threat in anxiety disorders: An integrative review. *Clinical Psychology Review, 30*(2), 203–216.

Clarke, R. J., & Johnstone, T. (2013). Prefrontal inhibition of threat processing protects working memory from interference. *Frontiers in Human Neuroscience, 7*, 228.

Corson, H. (1890). *An introduction to the study of Shakespeare*. Boston: Heath. Retrieved from *http://archive.org/details/introductiontost01cors*.

Davidson, R. J., Pizzagalli, D., Nitschke, J. B., & Putnam, K. (2002). Depression: Perspectives from affective neuroscience. *Annual Review of Psychology, 53*(1), 545–574.

Davis, M. (2006). Neural systems involved in fear and anxiety measured with fear-potentiated startle. *American Psychologist, 61*(8), 741–756.

Davis, T., & Poldrack, R. A. (2013). Measuring neural representations with fMRI: Practices and pitfalls. *Annals of the New York Academy of Sciences*.

Delgado, M. R., Gillis, M. M., & Phelps, E. A. (2008). Regulating the expectation of reward via cognitive strategies. *Nature Neuroscience, 11*(8), 880–881.

Delgado, M. R., Nearing, K. I., LeDoux, J. E., & Phelps, E. A. (2008). Neural circuitry underlying the regulation of conditioned fear and its relation to extinction. *Neuron, 59*(5), 829–838.

Ehring, T., Fischer, S., Schnülle, J., Bösterling, A., & Tuschen-Caffier, B. (2008). Characteristics of emotion regulation in recovered depressed versus never depressed individuals. *Personality and Individual Differences, 44*(7), 1574–1584.

Epstein, J., Pan, H., Kocsis, J. H., Yang, Y., Butler, T., Chusid, J., et al. (2006). Lack of ventral striatal response to positive stimuli in depressed versus normal subjects. *American Journal of Psychiatry, 163*(10), 1784–1790.

Erk, S., Kleczar, A., & Walter, H. (2007). Valence-specific regulation effects in a working memory task with emotional context. *NeuroImage, 37*(2), 623–632.

Erk, S., Mikschl, A., Stier, S., Ciaramidaro, A., Gapp, V., Weber, B., et al. (2010). Acute and sustained effects of cognitive emotion regulation in major depression. *Journal of Neuroscience, 30*(47), 15726–15734.

Erthal, F., De Oliveira, L., Mocaiber, I., Pereira, M., Machado-Pinheiro, W., Volchan, E., et al. (2005). Load-dependent modulation of affective picture processing. *Cognitive, Affective, and Behavioral Neuroscience, 5*(4), 388–395.

Etkin, A., Egner, T., Peraza, D. M., Kandel, E. R., & Hirsch, J. (2006). Resolving emotional conflict: A role for the rostral anterior cingulate cortex in modulating activity in the amygdala. *Neuron, 51*(6), 871–882.

Etkin, A., Klemenhagen, K. C., Dudman, J. T., Rogan, M. T., Hen, R., Kandel, E. R., et al. (2004). Individual differences in trait anxiety predict the response of the basolateral amygdala to unconsciously processed fearful faces. *Neuron, 44*(6), 1043–1055.

Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry, 164*(10), 1476–1488.

Fitzgerald, P. B., Fountain, S., & Daskalakis, Z. J. (2006). A comprehensive review of the effects of rTMS on motor cortical excitability and inhibition. *Clinical Neurophysiology, 117*(12), 2584–2596.

Fitzgerald, P. B., Laird, A. R., Maller, J., & Daskalakis, Z. J. (2008). A meta-analytic study of changes in brain activation in depression. *Human Brain Mapping, 29*(6), 683–695.

Friedel, E., Schlagenhauf, F., Sterzer, P., Park, S., Bermpohl, F., Ströhle, A., et al. (2009). 5-HTT genotype effect on prefrontal–amygdala coupling differs between major depression and controls. *Psychopharmacology, 205*(2), 261–271.

Frijda, N. H. (1986). *The emotions*. Cambridge, UK: Cambridge University Press.

Frodl, T., Reinhold, E., Koutsouleris, N., Reiser,

M., & Meisenzahl, E. M. (2010). Interaction of childhood stress with hippocampus and prefrontal cortex volume reduction in major depression. *Journal of Psychiatric Research, 44*(13), 799–807.

Ghashghaei, H. T., Hilgetag, C. C., & Barbas, H. (2007). Sequence of information processing for emotions based on the anatomic dialogue between prefrontal cortex and amygdala. *NeuroImage, 34*(3), 905–923.

Gilbert, K. E. (2012). The neglected role of positive emotion in adolescent psychopathology. *Clinical Psychology Review, 32*(6), 467–481.

Gillihan, S. J., Rao, H., Wang, J., Detre, J. A., Breland, J., Sankoorikal, G. M. V., et al. (2010). Serotonin transporter genotype modulates amygdala activity during mood regulation. *Social Cognitive and Affective Neuroscience, 5*(1), 1–10.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross, (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*(2), 348–362.

Gyurak, A., & Etkin, A. (this volume, 2014). A neurobiological model of implicit and explicit emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 76–90). New York: Guilford Press.

Hariri, A. R., Mattay, V. S., Tessitore, A., Fera, F., & Weinberger, D. R. (2003). Neocortical modulation of the amygdala response to fearful stimuli. *Biological Psychiatry, 53*(6), 494–501.

Heinz, A., Braus, D. F., Smolka, M. N., Wrase, J., Puls, I., Hermann, D., et al. (2005). Amygdala–prefrontal coupling depends on a genetic variation of the serotonin transporter. *Nature Neuroscience, 8*(1), 20–21.

Heller, A. S., Johnstone, T., Light, S. N., Peterson, M. J., Kolden, G. G., Kalin, N. H., et al. (2013). Relationships between changes in sustained fronto-striatal connectivity and positive affect in major depression resulting from antidepressant treatment. *American Journal of Psychiatry, 170*(2), 197–206.

Heller, A. S., Johnstone, T., Shackman, A. J., Light, S. N., Peterson, M. J., Kolden, G. G., et al. (2009). Reduced capacity to sustain positive emotion in major depression reflects diminished maintenance of fronto-striatal brain activation. *Proceedings of the National Academy of Sciences, 106*(52), 22445–22450.

Holtzheimer, P. E., & Mayberg, H. S. (2011). Stuck in a rut: Rethinking depression and its treatment. *Trends in Neurosciences, 34*(1), 1–9.

Jackson, D. C., Malmstadt, J. R., Larson, C. L., & Davidson, R. J. (2000). Suppression and enhancement of emotional responses to unpleasant pictures. *Psychophysiology, 37*(4), 515–522.

Johnstone, T., van Reekum, C. M., Urry, H. L., Kalin, N. H., & Davidson, R. J. (2007). Failure to regulate: Counterproductive recruitment of top-down prefrontal–subcortical circuitry in major depression. *Journal of Neuroscience, 27*(33), 8877–8884.

Kalisch, R., Korenfeld, E., Stephan, K. E., Weiskopf, N., Seymour, B., & Dolan, R. J. (2006). Context-dependent human extinction memory is mediated by a ventromedial prefrontal and hippocampal network. *Journal of Neuroscience, 26*(37), 9503–9511.

Kanske, P., Heissler, J., Schönfelder, S., & Wessa, M. (2012). Neural correlates of emotion regulation deficits in remitted depression: The influence of regulation strategy, habitual regulation use, and emotional valence. *NeuroImage, 61*(3), 686–693.

Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry, 68*(5), 444–454.

Keedwell, P. A., Andrew, C., Williams, S. C. R., Brammer, M. J., & Phillips, M. L. (2005). The neural correlates of anhedonia in major depressive disorder. *Biological Psychiatry, 58*(11), 843–853.

Kim, S. H., & Hamann, S. (2007). Neural correlates of positive and negative emotion regulation. *Journal of Cognitive Neuroscience, 19*(5), 776–798.

Knutson, B., Bhanji, J. P., Cooney, R. E., Atlas, L. Y., & Gotlib, I. H. (2008). Neural responses to monetary incentives in major depression. *Biological Psychiatry, 63*(7), 686–692.

Lemogne, C., Gorwood, P., Boni, C., Pessiglione, M., Lehéricy, S., & Fossati, P. (2011). Cognitive appraisal and life stress moderate the effects of the 5-HTTLPR polymorphism on amygdala reactivity. *Human Brain Mapping, 32*(11), 1856–1867.

Levesque, J., Joanette, Y., Mensour, B., Beaudoin, G., Leroux, J. M., Bourgouin, P., et al. (2004). Neural basis of emotional self-regulation in childhood. *Neuroscience, 129*(2), 361–369.

Linden, D. E. J., Habes, I., Johnston, S. J., Linden, S., Tatineni, R., Subramanian, L., et al. (2012). Real-time self-regulation of emotion networks in patients with depression. *PLoS ONE, 7*(6), e38115.

Loo, C. K., Alonzo, A., Martin, D., Mitchell, P. B., Galvez, V., & Sachdev, P. (2012). Transcranial direct current stimulation for depression: 3-week, randomised, sham-controlled trial. *British Journal of Psychiatry, 200*(1), 52–59.

Lozano, A. M., Mayberg, H. S., Giacobbe, P., Hamani, C., Craddock, R. C., & Kennedy, S. H. (2008). Subcallosal cingulate gyrus deep brain stimulation for treatment-resistant depression. *Biological Psychiatry, 64*(6), 461–467.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95*(1), 15–20.

Mathers, C. D., Lopez, A. D., & Murray, C. J. L. (2006). The burden of disease and mortality by condition: Data, methods, and results for 2001. In A. D. Lopez, C. D. Mathers, M. Ezzati, D. T. Jamison, & C. J. L. Murray (Eds.), *Global burden of disease and risk factors* (pp. 45–241). New York: World Bank/Oxford University Press.

Mayberg, H. S., Lozano, A. M., Voon, V., McNeely, H. E., Seminowicz, D., Hamani, C., et al. (2005). Deep brain stimulation for treatment-resistant depression. *Neuron, 45*(5), 651–660.

Meehl, P. E. (1975). Hedonic capacity: Some conjectures. *Bulletin of the Menninger Clinic, 39*(4), 295–307.

Milad, M. R., & Quirk, G. J. (2002). Neurons in medial prefrontal cortex signal memory for fear extinction. *Nature, 420*(6911), 70–74.

Milad, M. R., & Quirk, G. J. (2012). Fear extinction as a model for translational neuroscience: Ten years of progress. *Annual Review of Psychology, 63*(1), 129–151.

Milad, M. R., Wright, C. I., Orr, S. P., Pitman, R. K., Quirk, G. J., & Rauch, S. L. (2007). Recall of fear extinction in humans activates the ventromedial prefrontal cortex and hippocampus in concert. *Biological Psychiatry, 62*(5), 446–454.

Mitchell, D. G. V. (2011). The nexus between decision making and emotion regulation: A review of convergent neurocognitive substrates. *Behavioural Brain Research, 217*(1), 215–231.

Monk, C. S., McClure, E. B., Nelson, E. E., Zarahn, E., Bilder, R. M., Leibenluft, E., et al. (2003). Adolescent immaturity in attention-related brain engagement to emotional facial expressions. *NeuroImage, 20*(1), 420–428.

Nelson, E. E., McClure, E. B., Monk, C. S., Zarahn, E., Leibenluft, E., Pine, D. S., et al. (2003). Developmental differences in neuronal engagement during implicit encoding of emotional faces: An event-related fMRI study. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 44*(7), 1015–1024.

Ochsner, K. N., & Gross, J. J. (this volume, 2014). The neural bases of emotion and emotion regulation: A valuation perspective. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 23–42). New York: Guilford Press.

Padberg, F., & George, M. S. (2009). Repetitive transcranial magnetic stimulation of the prefrontal cortex in depression. *Experimental Neurology, 219*(1), 2–13.

Perlman, G., Simmons, A. N., Wu, J., Hahn, K. S., Tapert, S. F., Max, J. E., et al. (2012). Amygdala response and functional connectivity during emotion regulation: A study of 14 depressed adolescents. *Journal of Affective Disorders, 139*(1), 75–84.

Pezawas, L., Meyer-Lindenberg, A., Drabant, E. M., Verchinski, B. A., Munoz, K. E., Kolachana, B. S., et al. (2005). 5-HTTLPR polymorphism impacts human cingulate–amygdala interactions: A genetic susceptibility mechanism for depression. *Nature Neuroscience, 8*(6), 828–834.

Phelps, E. A., Delgado, M. R., Nearing, K. I., & LeDoux, J. E. (2004). Extinction learning in humans: Role of the amygdala and vmPFC. *Neuron, 43*(6), 897–905.

Posner, M. I., Snyder, C. R., & Davidson, B. J. (1980). Attention and the detection of signals. *Journal of Experimental Psychology, 109*(2), 160–174.

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004). The role of the medial frontal cortex in cognitive control. *Science, 306*(5695), 443–447.

Rolls, E. T. (2004). The functions of the orbitofrontal cortex. *Brain and cognition, 55*(1), 11–29.

Savitz, J., & Drevets, W. C. (2009). Bipolar and major depressive disorder: Neuroimaging the developmental-degenerative divide. *Neuro-*

science and Biobehavioral Reviews, 33(5), 699–771.

Schaefer, H. S., Putnam, K. M., Benca, R. M., & Davidson, R. J. (2006). Event-related functional magnetic resonance imaging measures of neural activity to positive social stimuli in pre- and post-treatment depression. *Biological Psychiatry, 60*(9), 974–986.

Schardt, D. M., Erk, S., Nüsser, C., Nöthen, M. M., Cichon, S., Rietschel, M., et al. (2010). Volition diminishes genetically mediated amygdala hyperreactivity. *NeuroImage, 53*(3), 943–951.

Scherer, K. R., Schorr, A., & Johnstone, T. (2001). *Appraisal processes in emotion: Theory, methods, research.* New York: Oxford University Press.

Schönfeldt-Lecuona, C., Lefaucheur, J.-P., Cardenas-Morales, L., Wolf, R. C., Kammer, T., & Herwig, U. (2010). The value of neuronavigated rTMS for the treatment of depression. *Neurophysiologie Clinique, 40*(1), 37–43.

Schutter, D. J. L. G. (2009). Antidepressant efficacy of high-frequency transcranial magnetic stimulation over the left dorsolateral prefrontal cortex in double-blind sham-controlled designs: A meta-analysis. *Psychological Medicine, 39*(1), 65–75.

Schwartz, C. E., & Rauch, S. L. (2004). Temperament and its implications for neuroimaging of anxiety disorders. *CNS Spectrums, 9*(4), 284–291.

Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants "grown up": Adult amygdalar response to novelty. *Science, 300*(5627), 1952–1953.

Shackman, A. J., Sarinopoulos, I., Maxwell, J. S., Pizzagalli, D. A., Lavric, A., & Davidson, R. J. (2006). Anxiety selectively disrupts visuospatial working memory. *Emotion, 6*(1), 40–61.

Shin, L. M., & Liberzon, I. (2010). The neurocircuitry of fear, stress, and anxiety disorders. *Neuropsychopharmacology, 35*(1), 169–191.

Slotema, C. W., Blom, J. D., Hoek, H. W., & Sommer, I. E. C. (2010). Should we expand the toolbox of psychiatric treatment methods to include repetitive transcranial magnetic stimulation (rTMS)? *Journal of Clinical Psychiatry, 71*(7), 873–884.

Somerville, L. H., Wagner, D. D., Wig, G. S., Moran, J. M., Whalen, P. J., & Kelley, W. M. (2012). Interactions between transient and sustained neural signals support the generation and regulation of anxious emotion. *Cerebral Cortex, 23*(1), 49–60.

Speer, A. M., Kimbrell, T. A., Wassermann, E. M., Repella, J., Willis, M. W., Herscovitch, P., et al. (2000). Opposite effects of high and low frequency rTMS on regional brain activity in depressed patients. *Biological Psychiatry, 48*(12), 1133–1141.

Staudinger, M. R., Erk, S., & Walter, H. (2011). Dorsolateral prefrontal cortex modulates striatal reward encoding during reappraisal of reward anticipation. *Cerebral Cortex, 21*(11), 2578–2588.

Tomarken, A. J., & Keener, A. D. (1998). Frontal brain asymmetry and depression: A self-regulatory perspective. *Cognition and Emotion, 12*(3), 387–420.

Urry, H. L., van Reekum, C. M., Johnstone, T., Kalin, N. H., Thurow, M. E., Schaefer, H. S., et al. (2006). Amygdala and ventromedial prefrontal cortex are inversely coupled during regulation of negative affect and predict the diurnal pattern of cortisol secretion among older adults. *Journal of Neuroscience, 26*(16), 4415–4425.

Van Dillen, L. F., Heslenfeld, D. J., & Koole, S. L. (2009). Tuning down the emotional brain: An fMRI study of the effects of cognitive load on the processing of affective images. *NeuroImage, 45*(4), 1212–1219.

van Reekum, C. M., Schaefer, S. M., Lapate, R. C., Norris, C. J., Greischar, L. L., & Davidson, R. J. (2011). Aging is associated with positive responding to neutral information but reduced recovery from negative information. *Social Cognitive and Affective Neuroscience, 6*(2), 177–185.

Wager, T. D., Davidson, M. L., Hughes, B. L., Lindquist, M. A., & Ochsner, K. N. (2008). Prefrontal-subcortical pathways mediating successful emotion regulation. *Neuron, 59*(6), 1037–1050.

Walker, D. L., Toufexis, D. J., & Davis, M. (2003). Role of the bed nucleus of the stria terminalis versus the amygdala in fear, stress, and anxiety. *European Journal of Pharmacology, 463*(1–3), 199–216.

Winecoff, A., LaBar, K. S., Madden, D. J., Cabeza, R., & Huettel, S. A. (2011). Cognitive and neural contributors to emotion regulation in aging. *Social Cognitive and Affective Neuroscience, 6*(2), 165–176.

CHAPTER 5

A Neurobiological Model of Implicit and Explicit Emotion Regulation

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Emotions take center stage in our lives through the ways they influence how we think and behave. Yet emotions are not deterministic; just as much as they influence us, we have the ability to regulate and change the way our emotional responses unfold. Most research on emotion regulation has focused on how people explicitly (using deliberate, effortful means) accomplish the regulation of emotion (e.g., Ochsner & Gross, 2005). We have previously argued that it is useful to anchor emotion regulatory processes along an implicit (nonconscious, automatic) to explicit (deliberate, effortful) dimension within a dual-process framework (Gyurak, Gross, & Etkin, 2011). In this chapter, we demonstrate the utility of the implicit–explicit framework to organize a wealth of neurobiological findings from basic affective and clinical neuroscience. We utilize this new expanded framework to illustrate its benefits for understanding healthy and disordered emotion regulatory processes.

We present paradigmatic cases of explicit and implicit regulation to highlight the parallels and differences between implicit and explicit forms of regulation. We then explore cases in which implicit and explicit regulatory processes blend together, to illustrate the fluid boundaries between implicit and explicit regulation in many emotion regulatory contexts. By anchoring this conceptual framework in neurobiological findings, this model of emotion regulation brings together basic, clinical, neuroscientific, and translational research. Our goal in this chapter is to demonstrate how an understanding anchored at the implicit–explicit spectrum informs an understanding of psychopathology that cuts across traditional diagnostic boundaries.

Component Processes and Definition of Emotion Regulation

The Modal Model of Emotions

Using previous definitions (Gross & Thompson, 2007; Gross, this volume) we define "emotions" as interpersonal and transactional (Keltner & Haidt, 2001) processes that play out in the rich fabric of real or imagined social relationships, for example, feelings of embarrassment over a social *faux pas*, or anger when one is slighted. Emotions arise when individuals attend to a situation and see it as relevant to their goals, either in the short or long term (Levenson, 1999). As emotions unfold, they engage loosely coupled changes in the domains of subjective experience, behavior, and central and peripheral physiology (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). Many

of the central and peripheral physiological reactions have phylogenetic continuity across species (LeDoux, 2012). Moreover, these reactions simultaneously include processes generally seen as "emotional" (e.g., a withdrawal response or a feeling), as well as "cognitive" (e.g., an attentional focus on a relevant stimulus; Pessoa, 2010). Finally, emotions can be viewed as "push" forces that predispose us to act in certain ways (Frijda, 1987; Keltner & Gross, 1999; Levenson, 2003).

Together, these features of emotion constitute the *modal model* of emotion: a goal-driven person–situation transaction that structures attention, has particular meaning to an individual, and gives rise to an evolutionarily rooted coordinated yet flexible multisystem response (Gross & Thompson, 2007; Gross, this volume). However, just as emotions have many compulsory features that are well-conserved evolutionarily, emotions have also been understood to be malleable since the work of William James (1884). It is this latter aspect of emotion that is most crucial for an analysis of emotion regulation, because it is this feature that makes any form of regulation possible.

Emotion Regulation

Following past work, we define "emotion regulation" as functional processes that influence the *intensity, duration*, and *type of emotion* experienced (Gross & Thompson, 2007). Emotion regulation permits flexibility in emotional responding in accord with one's momentary as well as longer-term goals. Emotion regulatory processes may have their effects at one or more points in the emotion generative process, and may dampen, intensify, or simply maintain emotions, depending on an individual's current implicit or explicit goals. Emotion regulatory processes may also change the degree to which emotion response components cohere as the emotion unfolds.

We conceptualize emotion regulation as occurring along a spectrum from conscious, effortful, and controlled regulation (which we call *explicit*) to unconscious, and possibly effortless, or automatic regulation (*implicit*). The history of explicit–implicit process description in psychology has been marked by arguments over terms and definitions (Bargh, 1994; Moors & De Houwer, 2006). In general, attempts to create a clear dichotomy capable of completely dividing implicit and explicit processes have been unsuccessful. Drawing on this body of literature, for the purposes of this review, we define a spectrum, rather than discrete categories, and characterize explicit emotion regulation as those processes that require *conscious effort* for initiation, demand *some level of monitoring* during implementation, and are associated with *some level of insight* and *awareness*. Implicit regulation, is *evoked automatically*, run to completion *without monitoring*, and can happen *without insight* and *awareness*. As outlined below, specific examples of types of emotion regulation may vary with respect to the degree of explicitness or implicitness and, as such, this definition most clearly describes processes at the extreme ends of this spectrum. Our view is that there is scientific utility in using the explicit–implicit spectrum: It is more parsimonious to understand different emotion regulatory paradigms currently in the literature within this framework rather than characterize each paradigm at the level of constituent processes (level of monitoring, awareness, etc.).

We view both implicit and explicit forms of regulation as functional—as having arisen to help navigate the rich social world in which humans exist. The ability to deploy either form of regulation flexibly in a context-sensitive manner might be a marker of mental health. Additionally, the conscious, effortful operations of explicit emotion regulatory processes are useful in a variety of contexts for adjusting emotional reactions. Because of its cognitively demanding nature, however, explicit regulation is not something in which one can effectively engage all of the time. Rather, use of efficient implicit emotion regulation processes, which may run without awareness, is likely a critical component to well-being and everyday emotional functioning.

Most contexts in which emotions are regulated involve a variable mixture of implicit and explicit processes. The degree to which ongoing regulation is implicit or explicit may also change depending on the individual, context, or time course of the emotion or its regulation. For example, a particular regulatory process may be explicit in healthy

individuals (suppressing a testy response to a colleague) but implicit in patients (expressive suppression), in whom it may have been automatized through overrepetition, to the point that it has become habitual. Additionally, explicit and implicit regulation change in quick succession within one person over time. For example, one might go from consciously labeling one's feelings of frustration at work to reflexively looking at the positive outcome that might result from putting in the extra time.

Taken together, our proposal is that it is most fruitful to view emotion regulation as reflecting a spectrum of processes, ranging from the mostly explicit to the mostly implicit. In applying this framework and systematically testing its components we can gain important insights into the neural circuits that are critical for the dynamic process of emotion regulation, and into the utility of implicit and explicit processes and their relevance for well-being and, by extension, psychopathology.

Core Neural Structures of Emotion Regulation

Our focus in this chapter is on neurobiologically investigated paradigms of emotion regulation. This section provides a brief overview of the core structures in the prefrontal cortex that are thought to be critical for the monitoring and regulation of emotion. Figure 5.1 introduces the lateral–medial gradient along which explicit and implicit processes can be usefully lined up along. We then expand on these as we review each paradigm below.

The Lateral Prefrontal Cortex

The dorsolateral (dlPFC) and ventrolateral (vlPFC) prefrontal cortices are commonly associated with emotion regulation (e.g., Kalisch, 2009). Along with their role in cognitive control and executive functioning, these regions are typically described in the context of deliberate, effortful, and conscious regulation of emotion (Gyurak et al., 2011). However, as described earlier, emotion regulation can occur more reflexively and outside of our awareness, and this type of regulation implicates more medial prefrontal regions and might be more directly relevant to fear- and anxiety-related regulation (Gyurak et al., 2011).

The Medial Prefrontal Cortex

Regions of the medial frontal lobes, including the ventral portions of the anterior cingulate cortex (vACC) and ventromedial prefrontal cortex (vmPFC) have long been implicated in emotional processes, specifically in regulation of emotions. We review relevant evidence in turn below. In addition to emotion regulation, these regions are important in fear extinction and recalling inhibitory extinction memories a day or more after training (Milad et al., 2009; Phelps, Delgado, Nearing, & LeDoux, 2004). Similarly, the vACC/vmPFC is activated when exposure to a distant versus close threat occurs, suggesting that it may be involved in planning adaptive responses, a regulatory function (Mobbs et al., 2007). The vACC/vmPFC shows negative functional connectivity with the amygdala in a range of emotional tasks and has anatomical connectivity to the amygdala (Myers-Schulz & Koenigs, 2012). Finally, during explicit emotion regulation, the vACC may also act as an intermediary between dlPFC and the amygdala (Ochsner & Gross, 2005).

Selected Empirical Findings across the Spectrum of Emotion Regulation

Below we review a number of neurobiologically investigated examples of emotion regulatory processes along the explicit–implicit spectrum. Though these examples are ordered roughly in the degree to which they involve implicit or explicit processes, we remind the reader of the fluid and relative nature of implicit and explicit regulatory processes. We review the relevance of each example for increasing our understanding of psychopathology. We start on the implicit end.

Regulation of Emotional Conflict

Our first case of implicit emotion regulation is based on the emotional conflict task (Egner, Etkin, Gale, & Hirsch, 2008; Etkin,

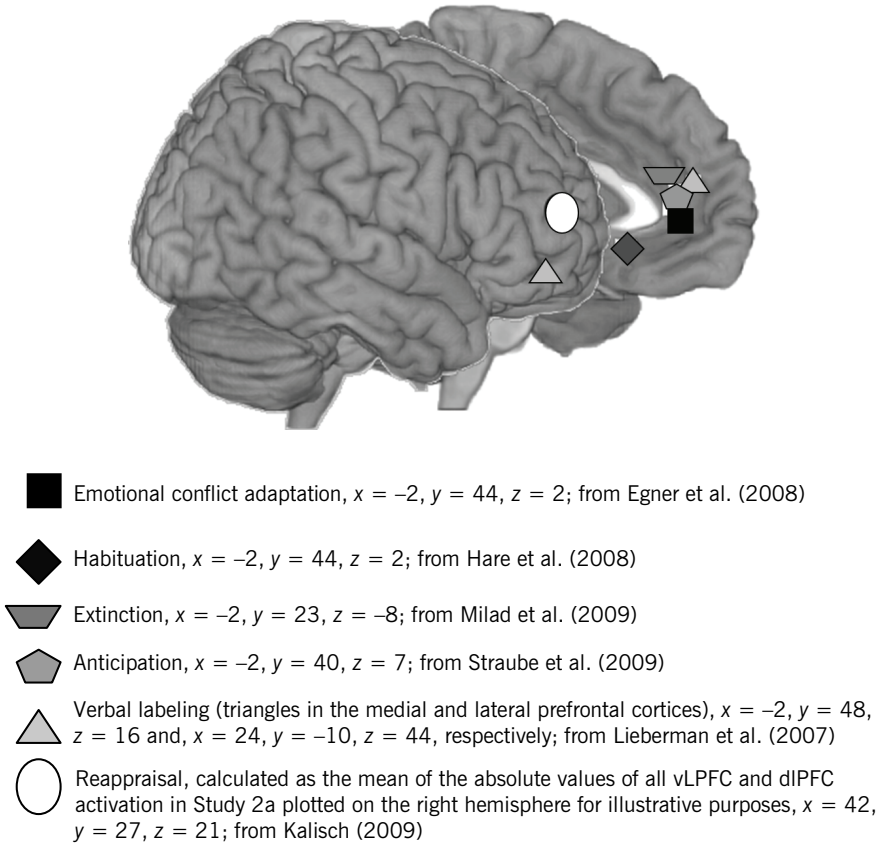

FIGURE 5.1. Putative foci for implicit and explicit forms of emotional regulation on a spectrum. Foci plotted are approximate; *x* coordinate = –2 for visualization purposes on the medial slice.

Egner, Peraza, Kandel, & Hirsch, 2006). The task is the emotional analogue of the classic Stroop paradigm (Stroop, 1935), and takes advantage of a variant of the congruency sequence effects originally reported by Gratton, Coles, and Donchin (1992) in non-emotional conflict tasks (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999). In the emotional conflict task, participants are presented with photographs of emotional faces (fearful or happy) with the words *fear* or *happy* written over them. The task is to indicate whether the facial expression is happy or fearful by pressing a button and to do this fast and accurately. The word written on the photo either matches the facial expression (no-conflict trials: happy face with the word *happy* written over it, or fearful face with the word *fear* written over it) or is incongruent with the facial expression (conflict trials: happy face with the word *fear* written over it, or fearful face with the word *happy* written over it). Because reading is automatized (Stroop, 1935), during conflict trials, participants need to enact control over reading the word in favor of labeling the emotional expression. Consistent with this, it takes longer to respond to conflict trials than to no-conflict trials; this slow-down in response time due to emotional conflict has been termed the *congruency effect*. Implicit emotion regulation in this task is indexed by trial-to-trial changes in the congruency effect as a function of the congruency effect on the previous trial (trial $n - 1$). Specifically, conflict on trial $n - 1$ triggers an increase in regulation, thereby reducing susceptibility to emotional conflict on trial n. Consistent with this, response times to incongruent trials are usually faster after incongruent trials (iI = current Incongruent preceded by an incongruent trial) than after congruent trials

(cI = current Incongruent preceded by a congruent trial). Implicit emotion regulation is therefore quantified by contrasting response times on iI with cI trials. This behavioral slowdown effect was shown to occur outside of awareness (Etkin, Prater, Hoeft, Menon, & Schatzberg, 2010). The task qualifies as an emotion regulation paradigm, because the stimulus itself evokes behavioral adjustment in the context of the goal of responding fast and accurately to emotionally conflicting stimuli. This task fits our definition of implicit regulation because the process is uninstructed, likely results from the stimulus properties itself, and is effortless and proceeds without awareness; despite careful probing, participants do not report any awareness of the key processes of the task (Etkin et al., 2010).

Parallel imaging (Egner et al., 2008) and lesion (Maier & di Pellegrino, 2012) studies of regulation of nonemotional conflict suggest that key elements of the neural circuitry involved in implicit regulation of emotional conflict is specific to the emotional content. Specifically, neuroimaging studies suggest a regulatory interplay between anterior cingulate cortex (ACC) and emotion reactivity regions in the limbic system in the emotional conflict task: *increased* activation in the vACC, and *dampened* reactivity in the amygdala (Etkin et al., 2006; Etkin et al., 2010; see Figure 5.1). In the nonemotional analogue of the task, in which subjects judged the gender of emotional faces, while trying to ignore congruent or incongruent gender labels, regulation was achieved through a dissociable neural pathway, involving *increased* activation in the dlPFC, and *positive coupling* to target-specific visual cortical areas in the fusiform face area, and as such, activation of the vACC and dampening of amygdalar reactivity was specific to implicit emotion regulation (Egner et al., 2008).

Applications of this task to clinical disorders revealed a marked deficit both behaviorally and in neural network engagement as a function of anxiety and depression (Etkin et al., 2010; Etkin & Schatzberg, 2011). Specifically, nonmedicated patients with either generalized anxiety disorder (GAD) or comorbid depression and GAD were unable to regulate the effect of emotional conflict as compared to healthy controls. Furthermore, analysis of functional magnetic resonance imaging (fMRI) data acquired during the emotional conflict task revealed that patients with GAD or depression failed to engage the circuitry normally implicated in this task. Specifically, patients failed to activate the vACC during regulation of emotional conflict and to dampen emotional conflict evaluation-related activity in the amygdala. These data indicate that regulation of emotional conflict, as a prototypical exemplar of implicit emotion regulation, captures the critical inability of patients to manage emotional processing and engage in task-related behavior.

Extinction and Habituation

Both extinction and habituation are basic forms of learning that are important for adaptive behavioral responding to emotional challenges. Both of these qualify as emotion regulation because they underlie the ability to change emotional responding to fit situational demands. Both of these processes have aspects that happen outside of conscious awareness and, as such, share implicit emotion regulatory elements but are likely to be a blend of implicit and explicit processes in humans, as detailed below (Delgado, Nearing, LeDoux, & Phelps, 2008; Dijksterhuis & Smith, 2002; Lovibond, 2004).

Extinction involves repeated presentation of an innocuous stimulus (e.g., a blue cross) that was previously associated with an aversive stimulus (e.g., a loud noise) and had acquired the ability to elicit a defensive response (e.g., startle eyeblink) in the absence of the aversive stimulus. Through repeated presentation without the aversive outcome, a new inhibitory memory is formed between the innocuous stimulus and absence of the aversive outcome, such that the conditioned defensive reaction is no longer elicited. Even though extinction can happen without the individual being aware of the change in contingency, consciously registering the contingency changes during extinction has been shown to facilitate extinction (for a review, see Lovibond, 2004). This evidence implicates the presence of both implicit and explicit processes in extinction.

Importantly, the neural circuitry supporting extinction shares common elements with the circuitry mapped out for the regulation

of emotional conflict (i.e., vACC and amygdala). Specifically, the vACC and adjacent vmPFC reduce fear responses through interconnections with the amygdala and hippocampus during extinction learning and recall of extinction memories (Ehrlich et al., 2009; Milad & Quirk, 2002; Phelps, Delgado, Nearing, & LeDoux, 2004). More successful extinction is associated with decreased conditioned stimulus (CS)-driven activation in the dorsal anterior cingulate cortex (dACC) in human (Milad et al., 2009) and animal models (Livneh & Paz, 2012). This is consistent with a recent review of the role of the dACC in evaluation and expression of negative emotion (Etkin, Egner, & Kalisch, 2011) and suggest the presence of a second pathway between the dACC and the amygdala where activation might be actually detrimental to extinction of fear memories.

Clinical models document impaired extinction behaviorally in posttraumatic stress disorder (PTSD; Norrholm et al., 2011; Wessa & Flor, 2007; for a review, see Lissek et al., 2005). Behavioral symptoms in GAD and specific anxiety disorders also indicate an inability to inhibit dysfunctional fear responses. In addition, the presence of safety and avoidance behaviors in these disorders might function to prevent the uncoupling of fear responses (Graham & Milad, 2011; Milad et al., 2009). Consistent with this, heightened amygdala activation was documented in PTSD during fear extinction following fear conditioning (Milad et al., 2009). However, there were no differences in behavioral or neural patterns of extinction as a function of social anxiety (Pejic, Hermann, Vaitl, & Stark, 2011), which might suggest preservations of extinction in social anxiety.

Habituation is the adaptive reduction of the initial response to emotional stimuli that carries no immediate reward or punishment value to the individual. A typical paradigm used in studying the neurobiological substrates of habituation in humans is to repeatedly present emotionally salient stimuli (e.g., emotional faces) over several runs of the scan and look at the time course of the neuronal signal change from early to late trials (Hare et al., 2008) within regions typically associated with processing the stimuli. Behavioral manipulations of attention to different aspects of the stimulus have been shown to alter the rate of habituation to nonemotional stimuli (e.g., tones; Gruzelier & Eves, 1987), suggesting that there are both implicit and explicit processes at play in habituation.

Studies have shown that the signal from the amygdala is reliably reduced as participants repeatedly encounter a salient but emotionally nonrewarding or punishing stimulus (Fisher et al., 2009; Hare et al., 2008). The extent of habituation, however, appears to be a function of the engagement of medial PFC circuitry that overlaps with the circuit associated with regulation of emotional conflict (i.e., vACC). For example, during habituation, engagement of the medial PFC is related to lower amygdala responses (Hare et al., 2008). A recent fMRI–PET (positron emission tomography) study also showed that higher density of serotonergic neurons in the vACC—considered an indicator of the capacity to modulate emotional arousal—is related to better habituation to negative faces and lower amygdala responses (Fisher et al., 2009). Failure of habituation of the amygdala has also been documented in severely inhibited individuals (Blackford, Allen, Cowan, & Avery, 2013) and in high trait anxiety (Hare et al., 2008) as well as PTSD (Shin et al., 2005). Furthermore, impaired habituation of auditory startle responses (a process mediated through the amygdala) was found to be a marker of later development of PTSD in a longitudinal study (Pole et al., 2009).

Anticipatory Responding to Emotional Stimuli

Anticipation of negative emotional events prepares the individual to respond adaptively and flexibly to actual emotional challenges as they arise. This process has typically been studied in experimental paradigms by presenting cues that with some probability predict the occurrence of a threatening or negative event (e.g., electric shock, thermal pain, negative image) (Drabant et al., 2011; Nitschke et al., 2009; Straube, Schmidt, Weiss, Mentzel, & Miltner, 2009). Anticipatory responding fits our definition of emotion regulation, because it allows the individual to optimize and prepare future responding in the face of an emotional challenge. The conscious anticipatory component gives a more explicit flavor to these paradigms, but

in the absence of explicit instruction to regulate the response, the implicit component is retained.

More importantly, these paradigms have proved to be useful in mapping the neural circuits associated with anticipatory responses and associated anxiety. These results show the involvement of the vACC and the broader ACC with typically higher activation during anticipation (Butler et al., 2005; Nitschke et al., 2009; Ploghaus, Becerra, Borras, & Borsook, 2003; Ploghaus et al., 1999; Straube et al., 2009), and often tracking concomitant anxiety. Sarinopoulos et al. (2010) reported that anticipatory activation in the vACC following uncertain cues was related to systematic overestimation of the frequency with which the cue was predictive of aversive images. In terms of the relationship to anticipatory responding, informative moderation effects have been found. Specifically, during moderate levels of threat (mildly painful electric shock), higher vACC activation was related to higher anticipatory preparedness, whereas during high levels of threat of electric shocks, higher vACC activation was related to lower anticipatory preparedness (Straube et al., 2009), suggesting that higher vACC activation indices anticipatory preparedness, whereas deactivation might track disengagement from the task, a perhaps more explicit form of regulation. vACC activation was also documented in a virtual chasing game while participants expected painful levels of electric shocks, and this activation increased as a function of anticipated pain (Mobbs et al., 2007).

Clinically, abnormalities in activation to anticipatory cues preceding negative anxiety-provoking images have been documented in GAD (Nitschke et al., 2009). Similarly, patients with unipolar major depressive disorder (Abler, Erk, Herwig, & Walter, 2007) also showed higher amygdala activation compared to healthy controls while anticipating negative pictures.

Incidental Regulation through Language

Goal-directed engagement of higher order cognitive processes, such as language, in emotional contexts can have "accidental" emotion regulatory consequences. In experimental paradigms, participants are instructed to label the emotion displayed on a face (e.g., indicate whether the face is angry or afraid), match the affect shown on the face to another face showing the same emotion, or match the gender of the face (Hariri, Bookheimer, & Mazziotta, 2000; Lieberman et al., 2007; Lieberman, Hariri, Jarcho, Eisenberger, & Bookheimer, 2005). There are important similarities between verbal labeling as done in this task and creation of a verbal narrative that is supposed to lessen emotional arousal seen in explicit regulatory tasks. Because of these parallels, the task fits our definition of emotion regulation, as it results in the regulation of emotional arousal in a goal-directed fashion. Semantically labeling emotional stimuli, or "putting feelings into words," as these paradigms are typically described, versus matching the emotion on another face or labeling the gender has a marked explicit component, yet the regulation still happens in the absence of an explicit goal to do so.

At the neural level, matching emotional expressions on faces with words that label affect results in lower amygdala activation, as compared to matching expressions to other expressions, matching the gender to gender labels, or simply viewing the face. Moreover, labeling the emotion of facial expressions or focusing on physical rather than emotional features of an emotionally evocative scene results in increased activation in the lateral and medial prefrontal cortices, despite the fact that emotion regulation in this paradigm is unintentional or implicit (Hariri et al., 2000; Lieberman et al., 2005). Importantly, Lieberman and colleagues (2007) documented that a vmPFC activation mediated the relationship between lateral PFC and decreased amygdala activity, suggesting that similar to implicit emotion regulation, it may be the medial PFC–amygdala pathway that plays a critical role in dampening emotional reactivity in these tasks (see Figure 5.1).

Variants of the affect-labeling task have been used in pediatric anxiety and mood disorder populations with or at-risk for psychopathology (Beesdo et al., 2009; Monk et al., 2007; Taylor, Eisenberger, Saxbe, Lehman, & Lieberman, 2006). These studies show that among at-risk adolescents, focusing attention on the physical features of an emotional face (nose width) or labeling one's subjective emotions while viewing

an emotional face, normalized concomitant neural activation in the PFC and amygdala. Similarly, McClure and colleagues (2007) found that the performance of children with GAD did not differ from that of their healthy counterparts in rating physical features of negative emotional faces, and that they recruited the same amygdala, dACC, and vmPFC networks as their healthy adolescent counterparts. However, in another study, Taylor et al. (2006) found that children from high-stress family environments showed higher amygdala and dlPFC activation during an affect labeling of faces task than children from low-risk families, who showed lower amygdala activation during affect labeling. Low-risk children did not differ in dlPFC activation. Taken together, these result shows that disordered and at-risk children are capable of labeling as a form of regulation behaviorally, and they might do so largely through the normative circuitry.

Instructed Regulation of Emotional Responses

A paradigmatic case of explicit regulation may be found in work by James Gross and colleagues (e.g., Gross & Levenson, 1993; McRae et al., 2010; Ochsner, Bunge, Gross, & Gabrieli, 2002). As reviewed by Ochsner and Gross (this volume), in a typical test of explicit emotion regulation, participants are presented with a task that involves processing stimuli under two different conditions—one in which participants are instructed to react naturally (reactivity trial), and another in which they are instructed to regulate their emotional responses (regulation trial) using a strategy specified by the researcher that participants have had ample opportunity to practice beforehand. Explicit emotion regulation performance is indexed by contrasting emotional responses in the reactivity and regulation trials. These tasks fit our definition of explicit emotion regulation, because the process is effortful and carried out with considerable awareness and monitoring. Specifically, individuals are (1) aware of the cues that elicit emotional responses (i.e., images, films), (2) aware of the emotion itself (i.e., they report reduction in accompanying feelings), and (3) aware of the effect of the regulation on their behavior (i.e., if prompted, they can report having engaged in emotion regulation). Often-studied forms of explicit regulation are reappraisal, in which participants are instructed to attempt to change the way they think about the emotional event so that they feel less negative emotions, and suppression, in which participants are instructed not to show how they feel.

Neuroimaging studies show that reappraisal results in a dynamic interchange between frontal lobe areas implicated in cognitive control and executive function, and emotion reactivity areas (for reviews, see Ochsner & Gross, 2005; Ochsner & Gross, this volume; Kalisch, 2009). Specifically, imaging studies indicate that attempts to reappraise negative stimuli result in *increased* activation in ventrolateral (vlPFC), dlPFC, dACC—areas traditionally implicated in non-emotional forms of cognitive control (Kalisch, 2009). This frontal lobe activation in turn is related to *reduced* activation in emotional reactivity-related areas (amygdala and insula), suggesting that engagement of control-related circuitry dampens reactivity in these critical emotion reactivity regions (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Goldin, McRae, Ramel, & Gross, 2008; Ochsner et al., 2002; Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008). Furthermore, there appears to be a temporal specificity in the engagement of frontal lobe regions as a function of implementation stage (Paret et al., 2011). Specifically, Kalisch and colleagues (Kalisch, 2009; Paret et al., 2011) propose that instructed reappraisal can be partitioned into two temporarily distinct phases: an early phase of implementation that comprises strategy selection and retrieval of reappraisal material into working memory, and a later phase of maintenance of performance-monitoring processes. Corresponding shifts in the recruitment of the lateral PFC are observed as a function of these phases from posterior to anterior parts of the lateral frontal cortex (Paret et al., 2011).

Assessment of instructed emotion regulatory abilities in clinical populations, however, has produced mixed results. There appears to be agreement that behaviorally, patients with mood and anxiety disorders are not deficient in using regulation when instructed to do so in the laboratory (Ehring, Tuschen-Caffier, Schnülle, Fischer, & Gross, 2010; Goldin, Manber, Hakimi,

Canli, & Gross, 2009; Goldin, Manber-Ball, Werner, Heimberg, & Gross, 2009), but they might spontaneously use certain beneficial forms of regulation such as reappraisal less frequently, or report feeling less successful in regulating their emotions as a result (Gruber, Harvey, & Gross, 2012). By contrast, a variety of neural deficits have been found in different patient groups. For example, currently depressed patients activated bilateral PFC during reappraisal of negative scenes, while healthy subjects did so only unilaterally (Johnstone et al., 2007). in another study, there was a lack of dlPFC activation in patients with major depressive disorder altogether, but activated dACC, amygdala, and insular cortices (Beauregard, Paquette, & Lévesque, 2006). Patients with social anxiety disorder showed delayed but overall enhanced engagement of dlPFC and dACC when instructed to reappraise negative self-relevant thoughts (Goldin, Manber-Ball, et al., 2009). By contrast, during reappraisal of angry/contemptuous faces, patients with social anxiety disorder failed to activate dACC and anterior medial frontal cortex (neither dlPFC nor vlPFC were involved in this task; Goldin, Manber-Ball, et al., 2009). Patients with GAD and social phobia both failed to activate the dACC when reappraising either positive or negative pictures (no dlPFC or vlPFC was involved in this task; Blair et al., 2012). Finally, trauma exposed patients with borderline personality disorder hypoactivated lateral and medial PFC more during reappraisal of negative scenes, relative to trauma-exposed healthy controls. Taken together, these results show a rather mixed set of abnormalities in instructed emotion regulation, with differences likely due to different tasks, stimuli, or instructions. Regardless, explicit regulation at the behavioral level does not appear to be perturbed in psychiatric disorder, even if patients recruit different neural circuits. Impairments may exist, however, in the spontaneous and potentially more implicit engagement of these effortful forms of regulation.

Implications and Future Research Directions

Our goal in this review was to provide a unifying conceptual framework for emotion regulation, highlighting and expanding our prior model of an implicit–explicit emotion regulatory spectrum. We have argued several key points.

First, types of emotion regulation vary in the degree to which they are implicit versus explicit. We demonstrated that the relative degree of implicit or explicit features depends on multiple factors, including the stimulus, task, context, individual differences, psychopathology, and time. Compounding this complexity, we showed that many existing experimental paradigms involve various admixtures of implicit and explicit types of emotion regulation. Greater attention should therefore be paid to the implications of these design features in light of a conceptualization of implicit regulation. Studies need to address the relative degree to which regulation is at any time implicit or explicit in both existing and new paradigms. Probing awareness about regulation is one way to assess implicitness versus explicitness. Other factors that influence whether a particular regulatory task is implicit or explicit are personality features, such as low trait rumination, which might predispose individuals to have more automatic tendencies to use reappraisal, and they might do so in a more implicit, automatic fashion (Ray et al., 2005). Another example is pathological worry in GAD (Borkovec, Alcaine, & Behar, 2004), which might parallel explicit forms of regulation by virtue of engaging effortful resources (e.g., prefrontal areas). This in turn might influence the ability of people with GAD engage explicit regulation in a goal-directed fashion.

Additionally, several researchers have pointed out the inherent difficulties involved in differentiating emotional regulation from emotional reactivity, with some questioning the utility of emotion regulation as a scientific construct altogether (Campos, Frankel, & Camras, 2004; Cole, Martin, & Dennis, 2004). Neuroimaging studies get around this problem and routinely include a contrasting control condition when the effects of emotion regulation are compared to those of canonical emotional reactivity trials (i.e., adaptation to emotional conflict is a contrast between iI and cI conditions; reappraisal is a contrast between uninstructed "watch" trials and instructed regulation). However, studies that purportedly measure the effects of spontaneous use of effortful regulation

often do not include a contrasting reactivity condition and do not carefully control for the effects of reactivity. Future research has to address emotional reactivity by systematically including a baseline unregulated condition, in addition to assessing implicit and explicit features. Future studies examining the explicit–implicit dimension systematically should also test the separability and interactions between these circuits.

Second, we showed that there appears broadly to be a medial–lateral differentiation between implicit and explicit regulatory processes (see Figure 5.1). That is, paradigmatic forms of implicit emotion regulation (e.g., emotional conflict regulation) involve ventromedial prefrontal regions in regulation, while paradigmatic forms of explicit regulation (e.g., reappraisal) involve mainly dlPFC and vlPFC for regulation. Other forms of regulation that contain a mixture of implicit and explicit processes (e.g., language-based incidental regulation) involve roles for both lateral PFC and vmPFC. While we do not consider this medial–lateral gradient to be an absolute rule, neither would we be confident in interpreting medial or lateral activation differences in patients as necessarily reflecting deficits in implicit or explicit regulation, respectively; this model provides a useful framework for describing the range of emotion regulation processes, anchored in relevant neurocircuitry. Moreover, we would readily concede that our framework is useful for anchoring our theoretical understanding of emotion regulation, but it is unlikely that implicit and explicit modules per se exist at the level of the brain. Future research might parse the operations of implicit and explicit regulation into circuit-level components (e.g., working memory, interference resolution) and our understanding of the implicit–explicit anchors might transform as our understanding of the interactions between participating brain areas develops. Our goal was to illustrate the fluid boundaries between implicit and explicit forms of regulation, and move away from characterizing implicit and explicit regulation in a dichotomous fashion. New paradigms in which the level of explicitness and implicitness is parametrically manipulated (e.g., in the form of increased explicit regulatory effort in the context of an otherwise implicit regulatory process or by repetitively training explicit regulation until it becomes automatized) will allow us to ground implicit and explicit regulation in neurobiology and further chart the interactive and dynamic processes by which the brain accomplishes regulation.

Our third point is that in striving for a neurobiological understanding of emotion regulation and developing these new paradigms, it will be important to refine the circuitry underlying implicit and explicit regulation. The human brain is likely to accomplish emotion regulation through an economical process that relies on a distributed network of brain areas. Three brain areas highlighted here as underlying implicit and explicit forms of regulation (medial PFC and dlPFC/vlPFC, respectively) have been documented to be involved in a wide array of processes besides emotion regulation. In this vein, further research will likely broaden our understanding of the relevant circuitry and how these functions relate to the variety of implicit and explicit regulatory mechanisms. Moreover, most studies focused on corticoamygdalar circuitry, but less attention has been paid to the role of the insula and midbrain structures essential for emotional processing (e.g., periaqueductal gray). The role of these nodes in the emotion regulatory circuitry with respect to implicit and explicit features remains to be explored.

Another important element that has thus far been unmapped in terms of implicit and explicit regulation is the modulation of approach tendencies and positive emotions (e.g., reward). There is evidence that approach might also be regulated in an implicit and explicit fashion through somewhat dissociable circuitry (Kable & Glimcher, 2007; McClure, Laibson, Loewenstein, & Cohen, 2004). For example, value-based decision making about food while focusing on the health aspect of the stimuli modulates activation in medial prefrontal regions that overlap with the vACC region mapped to types of implicit regulation, such as emotional conflict adaptation, and this modulation seems to be orchestrated by the dlPFC, which is involved in more explicit forms of regulation (Hare, Malmaud, & Rangel, 2011). Additionally, explicit regulation of monetary rewards in a group of self-assessed, successful regulators was related to lower striatal activation and fewer risky behavioral choices (Martin & Delgado, 2011), but without medial prefrontal or dlPFC involvement. These findings in the

realm of positive approach related will need to be integrated into our explicit–implicit framework through systematic studies that manipulate explicitness and implicitness in the context of positive emotions.

Our fourth point is that better definition of implicit and explicit tasks at the process level, and a refined understanding of the neural circuits involved, will help concretize how we can best improve these functions using targeted, neurobiologically derived, customized, and adaptive training methods (e.g., Vinogradov, Fisher, & de Villers-Sidani, 2011). One question is whether targeted training aimed at the dlPFC and vlPFC will impart emotion regulatory benefits, and whether these benefits will be evident in implicit or explicit regulatory tasks.

As our fifth point, we presented evidence that difficulties in emotion regulation in psychiatric disorders cut across implicit and explicit types. Specifically, we found behavioral and neural deficits in implicit forms of regulation: emotional adaptation; habituation; and documented, more subtle, largely neural deficits in more explicit forms of regulation (affect labeling and instructed reappraisal). These results support clinical observations of pervasive emotion regulatory symptoms in psychopathology and provide neurobiological anchors for the observed deficits that might track with treatment.

Conclusions

Strategies that individuals use to alter their emotions affect not only their current emotional experience but also broader emotional, cognitive, and interpersonal functioning. Thus, emotion regulation is of clear importance for both basic and applied translational investigations. Implicit processes are hidden for measurement reasons but are now more tractable, thanks to neuroscience methods. We have reached an exciting point at which neurobiologically anchored understanding of both implicit and explicit processes is rapidly advancing. Implicit and explicit processes arose to help us navigate our social and emotional worlds, and it is important that we conduct systematic studies on this domain. Our hope is that a unifying framework will facilitate this process and result in research advances for both basic and translational affective neuroscience fields.

References

Abler, B., Erk, S., Herwig, U., & Walter, H. (2007). Anticipation of aversive stimuli activates extended amygdala in unipolar depression. *Journal of Psychiatric Research, 41*(6), 511–522.

Banks, S. J., Eddy, K. T., Angstadt, M., Nathan, P. J., & Phan, K. L. (2007). Amygdala–frontal connectivity during emotion regulation. *Social Cognitive and Affective Neuroscience, 2*(4), 303–312.

Bargh, J. A. (1994). The four horsemen of automaticity: Awareness, intention, efficiency, and control in social cognition. In R. S. Wyer & T. K. Srull (Eds.), *Handbook of social cognition* (2nd ed., Vol. 1, pp. 1–40). Hillsdale, NJ: Erlbaum.

Beauregard, M., Paquette, V., & Lévesque, J. (2006). Dysfunction in the neural circuitry of emotional self-regulation in major depressive disorder. *NeuroReport, 17*(8), 843–846.

Beesdo, K., Lau, J. Y. F., Guyer, A. E., McClure-Tone, E. B., Monk, C. S., et al. (2009). Common and distinct amygdala-function perturbations in depressed vs. anxious adolescents. *Archives of General Psychiatry, 66*(3), 275–285.

Blackford, J. U., Allen, A. H., Cowan, R. L., & Avery, S. N. (2013). Amygdale and hippocampus fail to habituate to faces in individuals with an inhibited temperament. *Social Cognitive and Affective Neuroscience, 8*(2), 143–150.

Blair, K. S., Geraci, M., Smith, B. W., Hollon, N., DeVido, J., Otero, M., et al. (2012). Reduced dorsal anterior cingulate cortical activity during emotional regulation and top-down attentional control in generalized social phobia, generalized anxiety disorder, and comorbid generalized social phobia/generalized anxiety disorder. *Biological Psychiatry, 72*(6), 476–482.

Borkovec, T. D., Alcaine, O. M., & Behar, E. (2004). Avoidance theory of worry and generalized anxiety disorder. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 77–108). New York: Guilford Press.

Botvinick, M., Nystrom, L. E., Fissell, K., Carter, C. S., & Cohen, J. D. (1999). Conflict moni-

toring versus selection-for-action in anterior cingulate cortex. *Nature, 402*(6758), 179–181.

Butler, T., Pan, H., Epstein, J., Protopopescu, X., Tuescher, O., Goldstein, M., et al. (2005). Fear-related activity in subgenual anterior cingulate differs between men and women. *NeuroReport, 16*(11), 1233–1236.

Campos, J. J., Frankel, C. B., & Camras, L. (2004). On the nature of emotion regulation. *Child Development, 75*(2), 377–394.

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development, 75*(2), 317–333.

Delgado, M. R., Nearing, K. I., LeDoux, J. E., & Phelps, E. A. (2008). Neural circuitry underlying the regulation of conditioned fear and its relation to extinction. *Neuron, 59*(5), 829–838.

Dijksterhuis, A., & Smith, P. K. (2002). Affective habituation: Subliminal exposure to extreme stimuli decreases their extremity. *Emotion, 2*(3), 203–214.

Drabant, E. M., Kuo, J. R., Ramel, W., Blechert, J., Edge, M. D., Cooper, J. R., et al. (2011). Experiential, autonomic, and neural responses during threat anticipation vary as a function of threat intensity and neuroticism. *NeuroImage, 55*(1), 401–410.

Egner, T., Etkin, A., Gale, S., & Hirsch, J. (2008). Dissociable neural systems resolve conflict from emotional versus nonemotional distracters. *Cerebral Cortex, 18*(6), 1475–1484.

Ehring, T., Tuschen-Caffier, B., Schnülle, J., Fischer, S., & Gross, J. J. (2010). Emotion regulation and vulnerability to depression: Spontaneous versus instructed use of emotion suppression and reappraisal. *Emotion, 10*(4), 563–572.

Ehrlich, I., Humeau, Y., Grenier, F., Ciocchi, S., Herry, C., & Lüthi, A. (2009). Amygdala inhibitory circuits and the control of fear memory. *Neuron, 62*(6), 757–771.

Etkin, A., Egner, T., & Kalisch, R. (2011). Emotional processing in anterior cingulate and medial prefrontal cortex. *Trends in Cognitive Sciences, 15*(2), 85–93.

Etkin, A., Egner, T., Peraza, D. M., Kandel, E. R., & Hirsch, J. (2006). Resolving emotional conflict: A role for the rostral anterior cingulate cortex in modulating activity in the amygdala. *Neuron, 51*(6), 871–882.

Etkin, A., Prater, K. E., Hoeft, F., Menon, V., & Schatzberg, A. F. (2010). Failure of anterior cingulate activation and connectivity with the amygdala during implicit regulation of emotional processing in generalized anxiety disorder. *American Journal of Psychiatry, 167*(5), 545–554.

Etkin, A., & Schatzberg, A. F. (2011). Common abnormalities and disorder-specific compensation during implicit regulation of emotional processing in generalized anxiety and major depressive disorders. *American Journal of Psychiatry, 168*(9), 968–978.

Fisher, P. M., Meltzer, C. C., Price, J. C., Coleman, R. L., Ziolko, S. K., Becker, C., et al. (2009). Medial prefrontal cortex 5-HT2A density is correlated with amygdala reactivity, response habituation, and functional coupling. *Cerebral Cortex, 19*(11), 2499–2507.

Frijda, N. H. (1987). *The emotions*. Cambridge, UK: Cambridge University Press.

Goldin, P. R., Manber, T., Hakimi, S., Canli, T., & Gross, J. J. (2009). Neural bases of social anxiety disorder: Emotional reactivity and cognitive regulation during social and physical threat. *Archives of General Psychiatry, 66*(2), 170–180.

Goldin, P. R., Manber-Ball, T., Werner, K., Heimberg, R., & Gross, J. J. (2009). Neural mechanisms of cognitive reappraisal of negative self-beliefs in social anxiety disorder. *Biological Psychiatry, 66*(12), 1091–1099.

Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The neural bases of emotion regulation: Reappraisal and suppression of negative emotion. *Biological Psychiatry, 63*(6), 577–586.

Graham, B. M., & Milad, M. R. (2011). The study of fear extinction: Implications for anxiety disorders. *American Journal of Psychiatry, 168*(12), 1255–1265.

Gratton, G., Coles, M. G., & Donchin, E. (1992). Optimizing the use of information: Strategic control of activation of responses. *Journal of Experimental Psychology. General, 121*(4), 480–506.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gross, J. J, & Levenson, R. W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64*(6), 970–986.

Gross, J. J., & Thompson, R. A. (2007). *Emotion regulation: Conceptual foundations*. In J.

J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York: Guilford Press.

Gruber, J., Harvey, A. G., & Gross, J. J. (2012). When trying is not enough: Emotion regulation and the effort-success gap in bipolar disorder. *Emotion, 12*(5), 997–1003.

Gruzelier, J., & Eves, F. (1987). Rate of habituation of electrodermal orienting responses: A comparison of instructions to stop responding, count stimuli, or relax and remain indifferent. *International Journal of Psychophysiology, 4*(4), 289–291.

Gyurak, A., Gross, J. J., & Etkin, A. (2011). Explicit and implicit emotion regulation: A dual-process framework. *Cognition and Emotion, 25*(3), 400–412.

Hare, T. A., Malmaud, J., & Rangel, A. (2011). Focusing attention on the health aspects of foods changes value signals in vmPFC and improves dietary choice. *Journal of Neuroscience, 31*(30), 11077–11087.

Hare, T. A., Tottenham, N., Galvan, A., Voss, H. U., Glover, G. H., & Casey, B. J. (2008). Biological substrates of emotional reactivity and regulation in adolescence during an emotional go–no-go task. *Biological Psychiatry, 63*(10), 927–934.

Hariri, A. R., Bookheimer, S. Y., & Mazziotta, J. C. (2000). Modulating emotional responses: Effects of a neocortical network on the limbic system. *NeuroRreport, 11*(1), 43–48.

James, W. (1884). What is an emotion? *Mind, 9*, 188–205.

Johnstone, T., Van Reekum, C. M., Urry, H. L., Kalin, N. H., & Davidson, R. J. (2007). Failure to regulate: Counterproductive recruitment of top-down prefrontal–subcortical circuitry in major depression. *Journal of Neuroscience, 27*(33), 8877–8884.

Kable, J. W., & Glimcher, P. W. (2007). The neural correlates of subjective value during intertemporal choice. *Nature Neuroscience, 10*(12), 1625–1633.

Kalisch, R. (2009). The functional neuroanatomy of reappraisal: Time matters. *Neuroscience and Biobehavioral Reviews, 33*(8), 1215–1226.

Keltner, D., & Gross, J. J. (1999). Functional accounts of emotions. *Cognition and Emotion, 13*(5), 467–480.

Keltner, D., & Haidt, J. (2001). Social functions of emotions at four levels of analysis. In W. G. Parrott (Ed.), *Emotions in social psychology: Essential readings* (pp. 175–184). New York: Psychology Press.

LeDoux, J. (2012). Rethinking the emotional brain. *Neuron, 73*(4), 653–676.

Levenson, R. W. (1999). The intrapersonal functions of emotion. *Cognition and Emotion, 13*(5), 481–504.

Levenson, R. W. (2003). Blood, sweat, and fears: The autonomic architecture of emotion. *Annals of the New York Academy of Sciences, 1000*, 348–366.

Lieberman, M. D., Eisenberger, N. I., Crockett, M. J., Tom, S. M., Pfeifer, J. H., et al. (2007). Putting feelings into words: Affect labeling disrupts amygdala activity in response to affective stimuli. *Psychological Science, 18*(5), 421–428.

Lieberman, M. D., Hariri, A. R., Jarcho, J. M., Eisenberger, N. I., & Bookheimer, S. Y. (2005). An fMRI investigation of race-related amygdala activity in African-American and Caucasian-American individuals. *Nature Neuroscience, 8*(6), 720–722.

Lissek, S., Powers, A. S., McClure, E. B., Phelps, E. A., Woldehawariat, G., Grillon, C., et al. (2005). Classical fear conditioning in the anxiety disorders: A meta-analysis. *Behaviour Research and Therapy, 43*(11), 1391–1424.

Livneh, U., & Paz, R. (2012). Amygdala–prefrontal synchronization underlies resistance to extinction of aversive memories. *Neuron, 75*(1), 133–142.

Lovibond, P. F. (2004). Cognitive processes in extinction. *Learning and Memory, 11*(5), 495–500.

Maier, M. E., & di Pellegrino, G. (2012). Impaired conflict adaptation in an emotional task context following rostral anterior cingulate cortex lesions in humans. *Journal of Cognitive Neuroscience, 24*(10), 2070–2079.

Martin, L. N., & Delgado, M. R. (2011). The influence of emotion regulation on decision-making under risk. *Journal of Cognitive Neuroscience, 23*(9), 2569–2581.

Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. H., & Gross, J. J. (2005). The tie that binds? Coherence among emotion experience, behavior, and physiology. *Emotion, 5*, 175–190.

McClure, S. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. (2004). Separate neural systems value immediate and delayed monetary rewards. *Science, 306*(5695), 503–507.

McClure, E. B., Monk, C. S., Nelson, E. E., Parrish, J. M., Adler, A., Blair, R. J. R., et al. (2007). Abnormal attention modulation of fear circuit function in pediatric generalized

anxiety disorder. *Archives of General Psychiatry, 64*(1), 97–106.

McRae, K., Hughes, B., Chopra, S., Gabrieli, J. D. E., Gross, J. J., & Ochsner, K. N. (2010). The neural bases of distraction and reappraisal. *Journal of Cognitive Neuroscience, 22*(2), 248–262.

Milad, M. R., Pitman, R. K., Ellis, C. B., Gold, A. L., Shin, L. M., Lasko, N. B., et al. (2009). Neurobiological basis of failure to recall extinction memory in posttraumatic stress disorder. *Biological Psychiatry, 66*(12), 1075–1082.

Milad, M. R., & Quirk, G. J. (2002). Neurons in medial prefrontal cortex signal memory for fear extinction. *Nature, 420*(6911), 70–74.

Mobbs, D., Petrovic, P., Marchant, J. L., Hassabis, D., Weiskopf, N., Seymour, B., et al. (2007). When fear is near: Threat imminence elicits prefrontal–periaqueductal gray shifts in humans. *Science, 317*(5841), 1079–1083.

Monk, C. S., Klein, R. G., Telzer, E. H., Schroth, E. A., Mannuzza, S., Moulton, J. L., et al. (2007). Amygdala and nucleus accumbens activation to emotional facial expressions in children and adolescents at risk for major depression. *American Journal of Psychiatry, 165*(1), 90–98.

Moors, A., & De Houwer, J. (2006). Automaticity: A theoretical and conceptual analysis. *Psychological Bulletin, 132*(2), 297–326.

Myers-Schulz, B., & Koenigs, M. (2012). Functional anatomy of ventromedial prefrontal cortex: Implications for mood and anxiety disorders. *Molecular Psychiatry, 17*(2), 132–141.

Nitschke, J. B., Sarinopoulos, I., Oathes, D. J., Johnstone, T., Whalen, P. J., Davidson, R. J., et al. (2009). Anticipatory activation in the amygdala and anterior cingulate in generalized anxiety disorder and prediction of treatment response. *American Journal of Psychiatry, 166*(3), 302–310.

Norrholm, S. D., Jovanovic, T., Olin, I. W., Sands, L. A., Karapanou, I., Bradley, B., et al. (2011). Fear extinction in traumatized civilians with posttraumatic stress disorder: Relation to symptom severity. *Biological Psychiatry, 69*(6), 556–563.

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. E. (2002). Rethinking feelings: An FMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14*(8), 1215–1229.

Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9*(5), 242–249.

Ochsner, K. N., & Gross, J. J. (this volume, 2014). The neural bases of emotion and emotion regulation: A valuation perspective. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 23–42). New York: Guilford Press.

Paret, C., Brenninkmeyer, J., Meyer, B., Yuen, K. S. L., Gartmann, N., Mechias, M.-L., et al. (2011). A test for the implementation–maintenance model of reappraisal. *Frontiers in Psychology, 2*, 216. [E-publication ahead of print]

Pejic, T., Hermann, A., Vaitl, D., & Stark, R. (2011). Social anxiety modulates amygdala activation during social conditioning. *Social Cognitive and Affective Neuroscience, 8*(3), 267–276.

Pessoa, L. (2010). Emergent processes in cognitive–emotional interactions. *Dialogues in Clinical Neuroscience, 12*(4), 433–448.

Phelps, E. A., Delgado, M. R., Nearing, K. I., & LeDoux, J. E. (2004). Extinction learning in humans: Role of the amygdala and vmPFC. *Neuron, 43*(6), 897–905.

Ploghaus, A., Becerra, L., Borras, C., & Borsook, D. (2003). Neural circuitry underlying pain modulation: Expectation, hypnosis, placebo. *Trends in Cognitive Sciences, 7*(5), 197–200.

Ploghaus, A., Tracey, I., Gati, J. S., Clare, S., Menon, R. S., Matthews, P. M., et al. (1999). Dissociating pain from its anticipation in the human brain. *Science, 284*(5422), 1979–1981.

Pole, N., Neylan, T. C., Otte, C., Henn-Hasse, C., Metzler, T. J., & Marmar, C. R. (2009). Prospective prediction of posttraumatic stress disorder symptoms using fear potentiated auditory startle responses. *Biological Psychiatry, 65*(3), 235–240.

Ray, R. D., Ochsner, K. N., Cooper, J. C., Robertson, E. R., Gabrieli, J. D., & Gross, J. J. (2005). Individual differences in trait rumination and the neural systems supporting cognitive reappraisal. *Cognitive, Affective, and Behavioral Neuroscience, 5*(2), 156–168.

Sarinopoulos, I., Grupe, D. W., Mackiewicz, K. L., Herrington, J. D., Lor, M., Steege, E. E., et al. (2010). Uncertainty during anticipation modulates neural responses to aversion in human insula and amygdala. *Cerebral Cortex, 20*(4), 929–940.

Shin, L. M., Wright, C. I., Cannistraro, P. A., Wedig, M. M., McMullin, K., Martis, B., et al. (2005). A functional magnetic resonance imaging study of amygdala and medial pre-

frontal cortex responses to overtly presented fearful faces in posttraumatic stress disorder. *Archives of General Psychiatry, 62*(3), 273–281.

Straube, T., Schmidt, S., Weiss, T., Mentzel, H.-J., & Miltner, W. H. R. (2009). Dynamic activation of the anterior cingulate cortex during anticipatory anxiety. *NeuroImage, 44*(3), 975–981.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*, 643–662.

Taylor, S. E., Eisenberger, N. I., Saxbe, D., Lehman, B. J., & Lieberman, M. D. (2006). Neural responses to emotional stimuli are associated with childhood family stress. *Biological Psychiatry, 60*(3), 296–301.

Vinogradov, S., Fisher, M., & de Villers-Sidani, E. (2011). Cognitive training for impaired neural systems in neuropsychiatric illness. *Neuropsychopharmacology, 37*(1), 43–76.

Wager, T. D., Davidson, M. L., Hughes, B. L., Lindquist, M. A., & Ochsner, K. N. (2008). Prefrontal–subcortical pathways mediating successful emotion regulation. *Neuron, 59*(6), 1037–1050.

Wessa, M., & Flor, H. (2007). Failure of extinction of fear responses in posttraumatic stress disorder: Evidence from second-order conditioning. *American Journal of Psychiatry, 164*(11), 1684–1692.

Williams, L. M., Liddell, B. J., Kemp, A. H., Bryant, R. A., Meares, R. A., Peduto, A. S., et al. (2006). Amygdala–prefrontal dissociation of subliminal and supraliminal fear. *Human Brain Mapping, 27*(8), 652–661.

PART III

COGNITIVE APPROACHES

CHAPTER 6

Delay Discounting: A Two-Systems Perspective

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Throughout our daily lives, we confront decisions and situations that require self-control. Generally, these are of relatively minor consequence, such as when one resists the urge to order dessert or spend money on an expensive electronic gadget. However, failure to control one's impulses can also have serious ramifications, as with eating disorders, substance abuse, and compulsive gambling (Dawe & Loxton, 2004). In this chapter, we discuss the cognition–emotion interactions that occur with temptations and self-control as they are understood and have been studied in psychology and neuroscience.

Most often this work focuses on a set of phenomena that together comprise delay discounting and the class of choices that require selecting between rewards available at different points in time, known as *intertemporal choices*. *Delay discounting* refers to the simple fact that people tend to prefer to receive rewards sooner rather than later. In an intertemporal choice, the subjective value of each outcome is discounted in proportion to the delay until its receipt, through the process of delay discounting. One of the advantages of focusing on delay discounting is that it is possible to analyze how discounting takes place with the use of *discount functions*, which describe mathematically how subjective value declines as a function of delay.

There have been tremendous advances in research on delay discounting over the past 10 years, spurred in part by experiments using brain imaging as people engage in intertemporal choices. To a first approximation, the processes involved in evaluating outcomes have been distinguished by the extent to which they rely on "emotion" versus "reason" (e.g., McClure, Laibson, Loewenstein, & Cohen, 2004; Hare, Camerer, & Rangel, 2009; Figner et al., 2010). This dichotomy roughly corresponds to the tradeoff between satisfying immediate desires and adhering to abstract, long-term goals. The terms used to describe these processes have been diverse: hot and cold; automatic and controlled; impulsive and deliberative; passions and reason; visceral and abstract; and doer and planner, among others (Posner & Snyder, 1975; Shiffrin & Schneider, 1977; Thaler & Shefrin, 1981; Chaiken & Trope, 1999; Lieberman, Gaunt, Gilbert, & Trope, 2002; Mischel, Ayduk, & Mendoza-Denton, 2003; Loewenstein, 1996).

The interaction between cognition and emotion is common to emotion regulation generally; delay discounting can therefore be

thought of as one class of behavior in which emotion regulation strategies are employed. Indeed, the self-regulation strategies used in the context of intertemporal choice can be interpreted through the regulatory process model proposed by Gross (1998). Specifically, regulation can occur at several points in the decision-making process. First, we can intervene before the decision takes place, avoiding situations and circumstances that are likely to require self-control. In intertemporal choice, one such strategy referred to as *precommitment* involves committing to one course of behavior ahead of time so as to avoid unwanted temptations (Strotz, 1956). Second, we can regulate our valuation processes at the time of decision making, shifting our attention or reappraising our perception of an alluring stimulus (e.g., Hutcherson, Plassman, Gross, & Rangel, 2012). Third, following the process of valuation, we can attempt to suppress impulsive choice tendencies by exerting top-down cognitive regulation, a mechanism commonly related to self-control (Hare et al., 2009). Choice regulation by self-control implies that we select an option that is consistent with long-term goals even while we experience the temptation to choose a different option (cf. Figner et al., 2010). In each of these regulatory strategies, the interplay between valuation and cognitive regulation aligns closely with the functions of the "hot" and "cold" systems described earlier. As such, studying intertemporal choices as a combination of these two factors provides a useful framework for understanding the mechanisms by which we regulate our decisions.

In the remainder of this chapter we consider the various cognitive and neural processes that together compose delay discounting, and emphasize the regulatory mechanisms involved in controlling our decision making. We begin with the brain, and describe the large-scale distinction that is commonly made between brain networks linked to the behaviorally defined constructs of valuation and self-control. We then summarize available evidence suggesting that these processes account for many aspects of delay discounting behavior. Recent research has been expanding on this framework, with the goal of identifying the diversity of brain processes involved in intertemporal choice. Progress along these lines has come in part from investigations of how different decision processes interact in guiding behavior, highlighting important functions that seem to be played by regions of the medial prefrontal cortex. Finally, we return to emotion regulation, and discuss some of the intentional strategies that we can (and do) employ to overcome temptations, described from the perspective of the neural systems involved in delay discounting.

Neural Systems Involved in Delay Discounting

Our argument is that multiple systems contribute to delay discounting. Although this model provides a reasonable account of how decision making might be thought to occur, an important question should be addressed at the outset: Is it necessary to posit the existence of multiple separate systems? Many economic theories indicate that decision making can be wholly explained by the maximization of a unitary function of expected utility. Given that a single utility function can reasonably account for decision making in most contexts, why not settle for a more parsimonious, single-system account? Our answer is two pronged. First, as will be discussed, the variability in delay discounting behavior observed within and between individuals is difficult to capture with a single-system account, but it follows naturally from a multiple systems model (van den Bos & McClure, 2013). Second, to examine decision making with techniques such as functional magnetic resonance imaging (fMRI) provides strong evidence for qualitatively different neural systems involved in delay discounting (although some controversy still remains; McClure et al., 2004; McClure, Ericson, Laibson, Loewenstein, & Cohen, 2007; Kable & Glimcher, 2007; Peters & Büchel, 2011). In many aspects of their function and relationship to behavior, these neural systems appear to be specialized for the qualitatively different purposes of valuation and top-down cognitive control.

Valuation

We face a tremendous number of choices every day. Occasionally, these choices are

very significant, such as when we select which college to attend or decide how to invest our money. Such decisions warrant careful deliberation and integration of the various factors that contribute to the value of each course of action. However, for the vast majority of the choices that we face, careful deliberation would be prohibitive. If we stopped to think about all of the factors that determine what we should eat for breakfast, which clothes to wear, and which direction to drive to work, it would be difficult to accomplish anything during the day.

We refer to *valuation processes* as the set of relatively automatic processes that guide our behavior outside of our deliberative control, facilitating the myriad choices we face every day. In many instances, such automatic judgments are accompanied by an affective response (Kahneman, 2003). This appears to be particularly true for delay discounting. An immediate reward can be extremely tempting, producing affectively charged, high-arousal approach tendencies. Influential theories of emotions posit that one of the functions of these processes is to signal assessments, such as danger, so as to influence behavior without necessarily making us fully aware of the source of the threat (LeDoux, 2000). Generally, we make these assessments reflexively, without intention or deliberate control, and in a manner that is obscure to our explicit understanding. The subjective experiences generated by immediate outcomes share these properties.

At the neural level, a complex network of brain structures, both cortical and subcortical, underlies these automatic valuation processes (Figure 6.1). A wealth of data now indicate that positive motivational and affective signals are encoded in and communicated by regions of the brain that are targeted by midbrain dopamine neurons, including the nucleus accumbens (NAcc) and portions of the ventromedial prefrontal cortex (vmPFC; e.g., Knutson, Fong, Bennett, Adams, & Hommer, 2003). Other regions, including the amygdala and insular cortex, are also involved in affective processing, particularly with regard to signaling salient or aversive events (LaBar, Gatenby, Gore, LeDoux, & Phelps, 1998; Wicker et al., 2003). The NAcc and amygdala, core structures in these circuits, are evolutionarily quite old and exist with relatively conserved function in many animal species. Due partly to this fact, the motivational processes supported by this network of brain regions are thought to form a system that is separable from deliberative thought and reasoning that is likely to be specific to humans.

Dopaminergic projections (i.e., neuronal connections that rely on the neurotransmitter dopamine) into the NAcc are known to

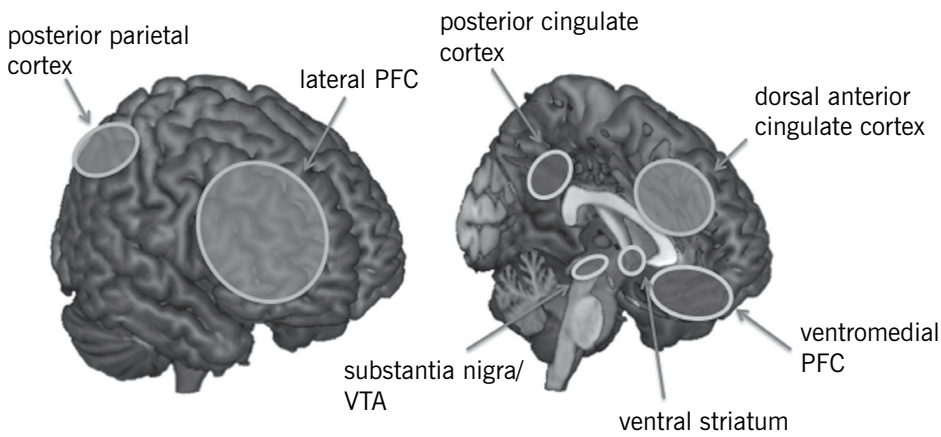

FIGURE 6.1. Two systems in delay discounting. Reward prediction, affect, and automatic behavioral responses are associated with the ventral striatum (nucleus accumbens), ventromedial PFC, and posterior cingulate cortex. These functions are components of automatic valuation. By contrast, regions in the dorsolateral and dorsomedial PFC, as well as the posterior parietal cortex, are associated with cognitive control processes and self-control.

be an especially crucial motivational signal. Some of the most compelling evidence for this comes from studies in which rats were given the opportunity to perform an action to self-stimulate different regions of their brains (Olds, 1977; Gallistel, Shizgal, & Yeomans, 1981; Ikemoto & Panksepp, 1999). Rats in these studies are especially motivated to self-stimulate regions of the midbrain that evoke dopamine release into the NAcc (Ikemoto & Panksepp, 1999). In fact, self-stimulation to these brain regions is so reinforcing that rats will self-stimulate continuously (i.e., 2,000 times an hour, or roughly once every 2 seconds), to the exclusion of all other activities, until they are too physically exhausted to continue (Olds, 1977). Rats also exert significant physical effort and endure pain (electric shock) to receive this stimulation (Olds, 1977). They even prefer brain stimulation to rewards such as food, when the stimulation is strong enough (Shizgal & Conover, 1996).

Dopamine release in the NAcc seems to be the crucial mediator of this self-stimulating behavior. Pharmacologically inhibiting the effects of dopamine release in the NAcc greatly diminishes the rewarding properties of this stimulation, and direct release of dopamine into the NAcc can substitute for electrical stimulation (Wise, 1982). These reward effects of dopamine also seem to apply in humans. This is clearly demonstrated by many drugs of abuse that directly increase dopamine availability in the brain, such as cocaine and methamphetamine (Koob & Bloom, 1988). Together, these data underscore the importance of dopamine and the NAcc in motivating behavior (Ikemoto & Panksepp, 1999; Berridge, Robinson, & Aldridge, 2009).

Functional neuroimaging experiments in humans have also demonstrated the involvement of dopamine-associated brain regions such as the NAcc and vmPFC in tracking value and signaling the expectation of rewarding outcomes (Knutson et al., 2003; McClure, Berns, & Montague, 2003; Delgado, 2007). Neural activity in these regions can be measured using fMRI, and such measurements have been shown to scale proportionally with the magnitude of expected reward (Knutson et al., 2003). In most situations, these dopamine-based value signals are quite helpful, as they allow us to predict value and guide our behavior on the basis of these predictions (Schultz, Dayan, & Montague, 1997). However, when these processes go awry, as is the case in addiction, exaggerated value signals for things such as drug-related cues can lead us to overvalue and seek out maladaptive outcomes (Redish, 2004).

The brain contains a separate network for encoding aversive signals and detecting outcomes in our environment that ought to be avoided. The amygdala has been the brain structure most clearly implicated with learning and detecting negative stimuli and environmental states (although recent evidence suggests that, at the spatial resolution afforded by fMRI, the amygdala may respond more generally to salient events, irrespective of valence; Hamann, Ely, Hoffman, & Kilts, 2002; Phelps, 2006). For example, presenting human or animal subjects with a stimulus that predicts an aversive event, such as a shock, is associated with increased amygdala activation, even if the shock does not actually occur (Phillips & LeDoux, 1992; Phelps et al., 2001). Similarly, amygdala lesions in animals have been shown to produce a number of odd behavioral effects, including increased approach behaviors in monkeys toward objects that they would normally fear (Blanchard & Blanchard, 1972). As might be expected, although aversive signals from the amygdala are generally necessary for adaptive behavior, they can also go awry, as is the case in social phobias (Phan, Fitzgerald, Nathan, & Tancer, 2006).

A clear feature of both NAcc and amygdala function is their automaticity. Amygdala responses to fearful or salient stimuli occur even when participants are unaware of seeing them, such as when images are shown so fast that they are only perceived subliminally (Whalen et al., 1998). Subliminal responses are also found with respect to rewards in the NAcc (Pessiglione et al., 2008). Additionally, the NAcc is a component of the basal ganglia, a subcortical motor circuit that underlies the development of habits (e.g., Jog, Kubota, Connolly, Hillegaart, & Graybiel, 1999). As such, it is well positioned to facilitate the development of new automatic behaviors.

Thus, there is strong evidence that both the amygdala and NAcc underlie the auto-

matic and stereotyped behaviors that are often accompanied by affective responses. It is easy to see why such a system might have evolved: The automatic generation of value and behaviors allows quick and effortless formulation of responses to rewards or threats in the environment. These systems are therefore believed to allow us to get on with our day, and, perhaps just as importantly, once helped us in ancestral environments to respond quickly enough to avoid predators and obtain fleeting rewards.

Although rapid, effortless, adaptive responses are a key benefit of this system, it is at the same time limited by its reliance on stereotyped behaviors. In the absence of regulatory influences, our valuation system seems to overgeneralize responses, leading to behaviors that are inappropriate in some circumstances (e.g., Hershberger, 1986). Moreover, in the domain of delay discounting, automatic valuation processes are inadequate for long-term planning. The system is hypothesized to learn to produce behaviors that are appropriate given what is occurring in the immediate environment. For goals that require planning over extended periods of time (weeks or years) valuation processes are simply not equipped to determine appropriate behaviors. Instead, in these cases, decision making requires careful consideration of goals and simulation of future states. These goal-related functions are commonly referred to as cognitive control.

Cognitive Control

The top-down cognitive control system is defined by deliberative, rule-based consideration of different courses of action (Kahneman, 2003). By definition, the computations involved in cognitive control occur within our conscious awareness; this is the kind of reasoning that we think about when we consider our subjective mental life. These capacities are particularly important when we face unfamiliar circumstances for which we do not have enough experience to know the appropriate (automatic) response. We also need to rely on cognitive control when there is a high chance for error. Consider as an example the famous Stroop task in which you have to name the color in which a word is written, while you suppress reading the word (think of the word *red* written in green ink; Cohen, Dunbar, & McClelland, 1990). Without explicitly controlling behavior to arrive at the appropriate response, people are likely to commit errors by automatically reading the word.

The brain structures associated with cognitive control are distinct from those that we previously attributed to valuation. These structures include dorsolateral and anterior (frontopolar) regions of the prefrontal cortex, regions within the dorsomedial prefrontal cortex, as well as the posterior parietal cortex (PPC). These brain regions are consistently observed to be involved in a variety of cognitive processes, such as working memory (e.g., Cohen et al., 1997), abstract reasoning (e.g., Kroger et al., 2002), and general problem solving (e.g., Duncan et al., 2000). There is broad consensus that these systems are central to the brain's ability to respond flexibly to rapidly changing task demands, as well as the pursuit of longer-term, goal-directed behaviors, especially when faced with competition from more salient stimuli or automatic responses (Miller & Cohen, 2001). Although our brain's cognitive control system involves an extensive network of interconnected structures, it is possible to illustrate some of its mechanistic features by restricting our focus to two regions: the dorsolateral prefrontal cortex (dlPFC) and dorsal anterior cingulate cortex (dACC).

The dlPFC seems to be crucial for successful planning and other executive functions. Lesions of the dlPFC are associated with dysfunction in organization, planning, working memory, and attention (Shallice & Burgess, 1991; Stuss & Levine, 2002). Similarly, brain imaging experiments in healthy adults have shown that the completion of tasks that require planning and problem solving is associated with greater activity in this region (e.g., Baker et al., 1996). The dlPFC is also associated with the maintenance and manipulation of information stored in working memory, consistent with its hypothesized role in integrating and manipulating stored mental representations in rational problem solving (Owen, McMillan, Laird, & Bullmore, 2005). Finally, the dlPFC is associated with the control of actions during tasks that require people to override automatic responses and instead respond based on a higher-level rule (MacDonald, Cohen, Stenger, & Carter, 2000).

The dACC also plays an important role in high-level cognition. In particular, the dACC has been shown to be especially important for the online monitoring of performance, as well as conflict and error detection (Carter et al., 1995). According to a dominant theory of dACC function, conflict detection in the dACC acts as a gating mechanism that signals when additional control is necessary (Botvinick, Braver, Barch, Carter, & Cohen, 2001). According to this theory, the dACC detects when our automatic processes are leading us awry and modulates activity in the dlPFC, leading to changes in behavior (i.e., so that we respond appropriately in circumstances in which errors are likely). The dACC's purported roles in conflict monitoring and dlPFC modulation are evident in tasks that span a variety of domains, ranging from simple motor responses to moral and social decision making (MacDonald et al., 2000; Greene, Nystrom, Engell, Darley, & Cohen, 2004; Sanfey, Rilling, Aronson, Nystrom, & Cohen, 2003).

The dlPFC and dACC are two regions in a network of structures that encode contextual information and facilitate deliberative processing and planning. These are precisely the functions ascribed to cognitive control. A critical limitation of this system is that information processing is slow and computationally expensive relative to automatic valuation. Moreover, we can only explicitly consider one thing at a time, so processing in this system is serial (Kahneman, 2003). These constraints limit the set of the decisions that are best addressed by this system.

Intertemporal Choice

To this point, we have considered valuation and cognitive control in relative isolation. In many situations, we are presented with choices for which we must arbitrate between following our impulses to achieve immediate gratification and choosing the actions that we know will be more beneficial in the long run. Intertemporal choice experiments target precisely this dilemma, asking people to decide between smaller rewards (most commonly money) that are available sooner in time and larger rewards that are available after a longer delay. In such cases, it must be determined whether the additional amount to be gained is worth the wait. That is, as in any value-based decision, the desirability of the available rewards must be determined, and a choice must be made on the basis of these relative values (Rangel, Camerer, & Montague, 2008). In intertemporal choice, the desirability of each reward depends on its magnitude, as well as the delay until its receipt.

Although everyone would prefer to receive reward sooner and in greater magnitude when all else is kept equal, people differ substantially in how they discount the value of future compensation. For example, people with chronic deficits in impulse control, such as pathological gamblers and cocaine addicts, tend to discount the value of delayed reward far more than control subjects (Bickel, Jarmolowicz, Mueller, Koffarnus, & Gatchalian, 2012). People suffering from attention-deficit/hyperactivity disorder (ADHD), a condition characterized by deficits in self-control, also discount rewards more steeply than healthy controls (Barkley, Edwards, Lanieri, Fletcher, & Metevia, 2001). Discounting rates also tend to decrease as people age from adolescence into adulthood, consistent with the general improvements in self-control that occur over the course of development (Green, Fry, & Myerson, 1994).

As already stated, from a mechanistic point of view, making intertemporal choices requires discounting the value of outcomes based on their delay, and choosing the option with greatest discounted values. Rationally, preferences generated in this way should be consistent across time. If you prefer outcome *A* over an outcome *B* that is to occur some fixed amount of time after *A* (say a week), then you should *always* prefer *A* to *B*, whether *A* occurs today (and *B* in a week) or *A* occurs in a year (and *B* in a year and a week). If this were not the case, then there would be some critical point in time at which preferences must switch, so that you initially prefer *B* but then reverse to prefer *A* some time thereafter. Mathematically, the only discount function that ensures consistency of preference is one that declines exponentially with delay (Frederick, Leowenstein, & O'Donoghue, 2003). This sounds reasonable enough, but neither humans nor animals demonstrate such consistency. In an often-cited example, when deciding between

one apple available in a year and two apples available in a year and a day, people generally prefer the latter. However, when the choice is between one apple available today and two apples available tomorrow, people generally prefer the former (Thaler, 1981). Such preference reversals indicate that humans (and other animals) discount more steeply over the near term than over the longer term; that is, people have a tendency to respond impulsively to tempting rewards that are immediately available.

Time-dependent preference reversals are most often explained by positing that delay discounting follows a hyperbolic discount function (e.g., Ainslie, 1975; Kirby, 1997; see Figure 6.2 for more detail). Mathematically, the hyperbolic function captures the observation that discount rates decline with time. However, this framework does little to explain the broad range of discount rates that people exhibit for different goods under different circumstances. In a well-known example, it was pointed out that it is hard to imagine people being impulsive for writing paper or gasoline (Hoch & Loewenstein, 1991). Instead, hyperbolic discounting seems to apply most when we are dealing with goods that are somehow viscerally arousing, as is commonly the case with money and food (Loewenstein, 1996). Similarly, we tend to be impulsive when we are aroused, either because of some exogenous emotionally charged event (e.g., Li, 2008) or because of internal reasons such as hunger (Giordano et al., 2002; Wang & Dvorak, 2010). While people appear to exhibit hyperbolic discounting under all of these conditions, the nature of the discount function varies substantially by circumstance (van den Bos & McClure, 2013).

An alternative approach to describing intertemporal choice views discounting behavior as resulting from the engagement of separate evaluative systems, each of which uses a different discount function (e.g., Loewenstein, 1996; van den Bos & McClure, 2013). The combined effect of multiple distinct discount functions can produce hyperbolic-like behavior. The simplest version of this account suggests that there are two types of discounting systems: one that values only goods that are immediately available, and another system that discounts more modestly over time (Laibson, 1997). This corresponds well to the two processing systems discussed earlier, with the automatic valuation system exhibiting steep discount-

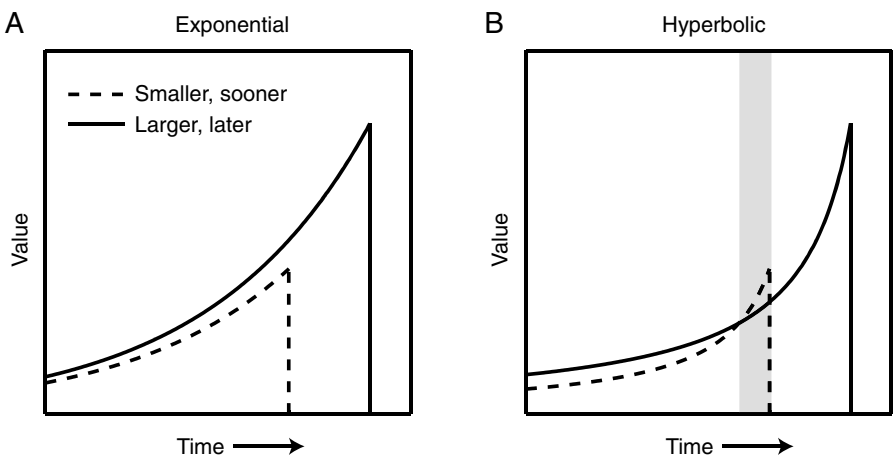

FIGURE 6.2. Value and preference reversal for smaller, sooner (SS) and larger, later (LL) reward. As time progresses, and the delay until receipt of SS and LL decreases, delay discounting also decreases and subjective value grows. (A) With exponential discounting, if LL is preferred to SS initially (solid line greater than dashed line at Time 0), then this ordinal preference is necessarily maintained. (B) However, with hyperbolic discounting, preference can reverse as the time to the SS reward approaches. The gray bar indicates the period of time, just prior to the availability of SS, when preferences favor the SS outcome.

ing and the cognitive control system placing more equal weight on immediate and future rewards (McClure et al., 2004, 2007).

A tremendous advantage of the two-system model of discounting is that it provides a simple explanation for why people are impulsive for certain goods and not others (e.g., writing paper) for which there is no associated affective response. Moreover, it suggests alternative routes to meliorating impulsive tendencies. One can work to dampen the emotional impact of a stimulus, exert deliberative control for the sake of satisfying longer-term goals, or undertake behaviors to avoid situations in which temptations hold sway. Many of these approaches have been investigated in recent research, and they coincide with different strategies common to instances of emotion regulation.

Neuroscientific studies of intertemporal choice provide support for the dissociation between automatic and controlled systems involved in the decision making. In one of our early fMRI studies, people were presented with two types of monetary choices: a choice between an immediately available option or a larger, delayed option, and a choice in which both options were available only after delays (McClure et al., 2004). According to the dual-process model of discounting, one would expect that choices involving an immediately available reward would be more affectively arousing in nature and therefore partially reflect the function of the impulsive valuation system. By contrast, choices involving only delayed rewards would not have this property, and would instead rely more heavily on deliberative processes to form judgments. The brain activation that was observed while subjects performed these two types of trials was consistent with this proposed formulation. A number of valuation-associated brain structures, including the NAcc and vmPFC, were activated during decisions that involved choices with an immediately available option. In contrast, structures involved in deliberative processing, including the dACC and dlPFC, were more engaged during decisions that involved two delayed outcomes. Additionally, for choices that pit an immediate reward against a delayed one, relative brain activation in the regions that comprise these two systems was predictive of the choice that people ultimately made. In other words, when structures in the automatic valuation system were more active, people tended to choose more impulsively, but when structures in the cognitive control system were more active, people were more likely to choose the larger, delayed option.

Subsequent fMRI studies have further elaborated and confirmed the dissociation of these neural systems. For example, the results from the experiment just described were replicated in an experiment in which thirsty subjects had to make choices between quantities of juice delivered at different periods of time, demonstrating that this type of dual processing is not specific to monetary rewards (McClure et al., 2007). Moreover, when deciding between foods, dlPFC activity tends to support the selection of healthier options via suppressing responses in vmPFC (Hare et al., 2009; discussed in greater detail below). There are important individual differences in discounting that are well explained by this two-system framework as well. Hariri and colleagues (2006) showed that individual differences in NAcc responses to winning money predict discount rates. In other words, the more that the automatic valuation system activates for the receipt of rewards, the more impulsively one tends to seek them. By contrast, individual differences in cognitive control predict reduced discounting. People who perform better on a working memory task and show greater responses in anterior parts of the lateral prefrontal cortex also tend to be more patient in intertemporal choices (Shamosh et al., 2008).

One important limitation of the results that we have discussed up to this point is that functional brain imaging provides data that are correlational in nature. In other words, fMRI provides information regarding which regions of the brain are associated with a particular cognitive process but reveals little about whether these brain regions actually cause the observed differences in behavior. To address this limitation, two recent studies assessed the causal importance of these structures through more direct manipulations. The first manipulated intertemporal decision making using repetitive transcranial magnetic stimulation (rTMS; Figner et al., 2010). In rTMS, electromagnetic pulses are produced to disrupt activity in particular regions of an otherwise typically functioning

brain. In this experiment, rTMS was administered the left and right dlPFC. After rTMS administration, subjects completed a set of intertemporal choice questions to assess the extent to which the procedure would disrupt the decision-making process. rTMS to the left lateral prefrontal cortex caused people to make more impatient choices, demonstrating that activation in this region causally promotes goal-directed decision making. A second study produced a similar effect by enhancing dopamine function pharmacologically by giving participants drugs that increase dopaminergic activity (Pine, Shiner, Seymour, & Dolan, 2010). Consistent with the two-system framework, this study found that discount rates were elevated as well.

Toward Multiple Interacting Systems

The two-system model we discussed earlier was primarily derived from behavioral studies. We now know that many of the properties of this two-system model correspond well with what is known about brain reward systems (i.e., dopamine and the NAcc) and regions linked to executive functions (i.e., dlPFC and dACC). However, additional details of brain function urge a more complex model, with more precise functions ascribed to individual brain areas, inclusion of additional systems in the delay discounting processes, as well as a more complete theory for how brain systems interact during choice.

At least three lines of research support a more refined, multiple-systems model of delay discounting. First, work by Bechara, Damasio, Damasio, and Lee (1999) examining the behavioral deficits associated with lesions to the vmPFC indicates a role for this structure in integrating cognitive and emotional influences on behavior, suggesting that this structure's function is more nuanced than allowed for in a two-system model. Second, although the available evidence suggests that the dACC plays a critical role in regulating the relative influence of automatic and controlled processes in decision making, no studies have clarified exactly how this regulatory function is implemented in complex scenarios such as intertemporal choice. Finally, recent studies suggest an important role for a brain region that has received very little attention in the literature on delay discounting—the hippocampus—in making intertemporal choices (Peters & Büchel, 2011). As a whole, these findings indicate the need to understand how automatic and controlled processes are integrated to produce coherent behavior, and how this integration might be expanded to include additional brain systems.

Value Integration in the vmPFC

A recent fMRI study dissociated the contributions of the brain systems we have linked to valuation and self-control in the context of decision making relative to unhealthy foods (Hare et al., 2009). In this study, subjects were scanned as they chose between food options that varied in health and taste qualities. For example, potato chips are low on health value but high on taste, and the opposite is true for broccoli. Subjects were classified as "self-controllers" or "non-self-controllers" based on their food preferences as expressed during the task. Self-controllers were people who made their decisions based on both health and taste, whereas non-self-controllers made their decisions based solely on taste. Activation in the vmPFC was shown to be associated with the total subjective value assigned to individual food items, such that activity was correlated with subjects' rated preference for the item at the time of choice. Additionally, for self-controllers, vmPFC activation at the time of choice was correlated with both the health and the taste of the items, whereas for non-self-controllers, vmPFC activation was correlated only with taste. Thus, this study indicates that the vmPFC encodes the total value assigned to individual food items.

Importantly, the separate factors that contribute to total value could also be identified in this study. The dlPFC showed elevated activation when participants successfully engaged self-control, so that dlPFC activity was greatest when subjects chose to reject liked but unhealthy foods. Additionally, comparison of functional activity in the dlPFC between self-controllers and non-self-controllers on these trials indicated that self-controllers displayed greater activation in this region at the time of choice, providing confirmatory evidence that activity in this region facilitates controlled decision making.

Finally, the investigators found a relationship between activation in the vmPFC and dlPFC for decisions on liked but unhealthy foods (i.e., those that should require the most self-control). In particular, greater activity in the dlPFC was associated with reduced activity in the vmPFC on these trials. These results indicate a means by which valuation and self-control processes can interact during decision making to facilitate successful self-control. Critically, the vmPFC seems to function as the site of interaction.

These findings fit into a larger body of data indicating that the vmPFC is important for integrating cognitive and emotional influences on behavior. Some of the most compelling evidence for this function comes from studies of patients with lesions to the vmPFC. These patients have normal executive functioning, with normal IQ and overall performance on tests of cognitive abilities. They also express normal emotional responses in tasks such as fear conditioning, in which an initially neutral stimulus (e.g., a visual stimulus) is paired with something aversive (e.g., an unpleasant sound) until participants learn to fear the stimulus (Bechara et al., 1999). Where patients with vmPFC lesions show striking deficits is in situations in which cognition and emotion must be combined to behave appropriately. Damasio (1994) summarizes this by saying that reason and emotion "intersect" at the vmPFC.

This intersection is clearly illustrated by behavioral deficits expressed by Elliot, a famous patient with vmPFC impairment (Eslinger & Damasio, 1985). Elliot had a tumor surgically removed from his vmPFC, compromising the function of neighboring cortical areas. The consequences were disastrous. By all accounts, Elliot seemed normal when in conversations. His IQ was significantly above normal, he was appropriately emotionally expressive, and he passed all manner of neuropsychological tests. Despite this, he made terrible financial decisions, squandering all his money in poor business decisions. He was unable to hold a job, and he interacted poorly with others, eventually leading to his wife divorcing him. Elliot's behavior seemed generally normal, but he had trouble with specific types of choices. For example, choosing a restaurant for dinner could be an onerous chore. He would study the menu, considering prices and the different types of food available. He would even drive to the restaurants to assess their ambiance. Despite this effort, he would still be unable to decide. On open-ended neuropsychiatric tests, Elliot could provide many reasonable solutions to complex problems, only to exclaim at the end "and after all this, I still wouldn't know what to do!" (Damasio, 1994, p. 49). Elliot's problem lay in linking automatic valuation and regulatory control when necessary to make a decision. Specifically, when decisions become too complex to be handled by the limited capacity of our cognitive control system, we seem to rely on the combination of affective and cognitive judgments—something like a gut feeling—to make the decision. This seems to be the function of the vmPFC.

Valuation and Control Interactions Involving the dACC–dlPFC Network

The dlPFC and dACC, key components of the cognitive control system discussed earlier, have been extensively studied in cognitive neuroscience and are primary regions involved in deliberated choices. They are critical nodes in a general-purpose cognitive control network that guides behavior in a wide variety of tasks, including intertemporal decision making (Ridderinkhof et al., 2004). We understand the most about the interactions between the dlPFC and dACC from their function in perceptual decision-making tasks. Although these perceptual tasks are mechanistically different from intertemporal choice, they provide useful insight regarding the role these regions may play in more complex decision scenarios.

Two experimental paradigms, the Eriksen flanker task and the Stroop task, have revealed a wealth of mechanistic detail about dACC and dlPFC function. In both tasks, successful performance requires participants to override a prepotent motor response in order to respond accurately to the stimulus presented. We discussed the Stroop task earlier, and why deliberation and control are necessary for appropriate behavior under the conditions of this experiment. The flanker task presents similar requirements. In this task, participants are asked to identify the central item among a set of distracter stimuli (e.g., respond "right" to > > > > > and

"left" to > > < > >). In incongruent trials, in which the flanking stimuli support a different response than the target stimulus (i.e., > > < > >), cognitive control becomes essential. People have a tendency to emit the incorrect response, as evidenced by an elevated error rate (Gratton, Coles, & Donchin, 1992). Furthermore, even when people are able to suppress the erroneous response, they are slower to respond than when the flanking stimuli support the correct answer. Thus, as in the Stroop task, successful performance on incongruent trials requires inhibition of the automatic tendency to respond to the predominant stimulus attribute.

Imaging and neurophysiological studies indicate that the type of control required by these tasks is primarily facilitated by contributions from the dlPFC and the dACC (Yeung, Botvinick, & Cohen, 2004; Kerns et al., 2004; Botvinick et al., 2001). As discussed earlier, these regions play complementary roles. The dACC is believed to monitor for the coactivation of incompatible responses, essentially serving as a detector for situations in which errors are likely and control is necessary. The dlPFC is thought to maintain the appropriate task goals and to direct action selection accordingly. When conflict is detected, the dACC shows increased activity, which then activates the dlPFC (Kerns et al., 2004). Consistent with this account, increased activity in this dACC–dlPFC network is commonly associated with improved performance during perceptual decision making.

An analogous mechanism seems likely to be at play in delay discounting. Imagine, for example, that you are asked to choose between two options that seem equally appealing (say, $10 today or $20 in a month). Response conflict is inevitable in this circumstance, and dACC activity may be anticipated to increase. Indeed, this has been found in several studies of intertemporal choice (e.g., Marco-Pallarés, Mohammadi, Samii, & Münte, 2010). In our own work we find evidence that the dACC signals conflict as well. For example, Figure 6.3 shows an analysis (McClure et al., 2004) that identifies the dACC as a region whose activity scales with reaction time (a surrogate for choice difficulty, and hence conflict).

The consequences of increased dlPFC activity following response conflict in delay discounting remain somewhat unresolved. On the one hand, a bulk of evidence suggests that increased dlPFC activity promotes the selection of larger, later rewards. In choices between immediate and delayed rewards,

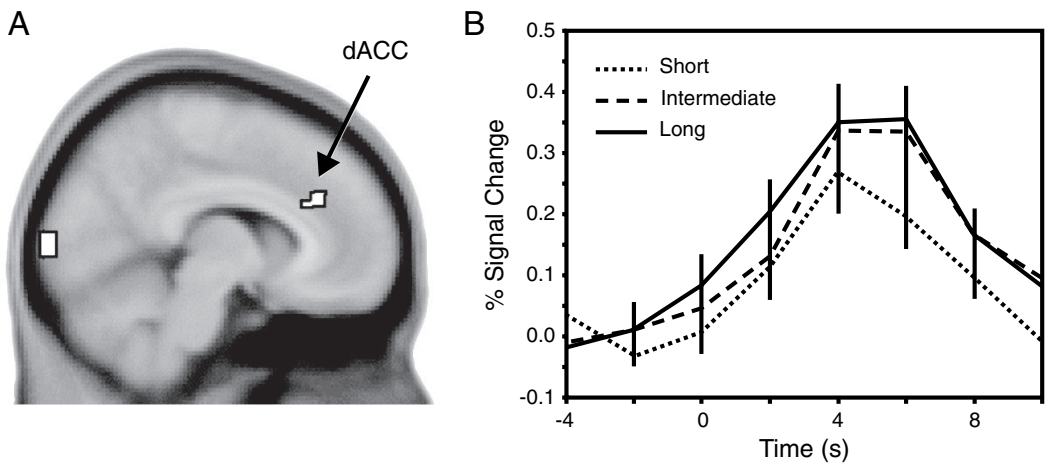

FIGURE 6.3. dACC activity in intertemporal choice. (A) Reanalysis of the data from McClure et al. (2004) reveals that dACC responses scale with choice difficulty, as approximated by choice response time. (B) Trials were split into three groups based on reaction time. Mean hemodynamic responses in the dACC scale linearly with response time. Time of zero seconds is when participants submitted their responses. The lag to the peak of the hemodynamic responses (~5–6s) is typical for this measurement modality.

increased dlPFC activity, particularly relative to NAcc responses, predicts selection of the latter alternative (McClure et al., 2004, 2007). Furthermore, disruption of the dlPFC by rTMS increases the proportion of choices for the immediate rewards, particularly in conditions where conflict is anticipated to be high (Figner et al., 2010). These results support the hypothesis that when conflict arises, the dACC may modulate the dlPFC, which in turn implements self-control by directly biasing choices toward future-oriented goals or by modulating the integrated valuation processes occurring in the vmPFC.

On the other hand, recent evidence from perceptual decision-making experiments (e.g., Philiastides, Auksztulewicz, Heekeren, & Blankenburg, 2011), as well as theoretical proposals (Kable & Glimcher, 2009), suggests that the dlPFC might instead facilitate the selection of the option assigned the highest discounted value by the valuation system. This evidence supports an alternative hypothesis in which the function of cognitive control is not necessarily linked to future-oriented goals independent of automatic valuation processes. Instead, this hypothesis suggests that automatic valuation and controlled processes may interact more generally to facilitate decision making. However, both of these proposals are consistent with the notion that the dACC–dlPFC network is the basis of deliberative decision making, guiding choices toward options that maximize value, either specifically for the long-term or generally. In either case, the dACC–dlPFC network appears to influence behavior through interactions with valuation regions, particularly the vmPFC (Hare et al., 2009).

Controlled Decisions and Memory in the Hippocampus

The hippocampus is well known in cognitive neuroscience primarily because of its role in remembering specific life events, known as episodic memories (Burgess, Maguire, & O'Keefe, 2002). This is best exemplified by patients such as HM, who are unable to form new memories after losing hippocampal function (Scoville & Milner, 1957). Episodic memory is also very important for conceiving of the future (Schacter, Addis, & Buckner, 2008); patients with hippocampal damage are unable to reason about hypothetical future events, perhaps because people rely on past experiences in order to conceive of probable future occurrences.

For delay discounting, this function of the hippocampus turns out to be very important. Intuitively, bringing past experiences to mind and envisioning future occurrences is an integral part of controlled decision making. Directed memory recall is guided in a goal-directed manner by dlPFC inputs to the hippocampus (Levy & Anderson, 2002). Consequently, rewards promised at future times linked to specific events (e.g., a $25 payment on December 23, during the holidays) elicit increased responses in the dlPFC and hippocampus (Peters & Büchel, 2010). Such tangible future episodes have profound effects on delay discounting; the greater the recruitment of the hippocampus for future dates, the less the discounting of future rewards. Similarly, when people are given identical intertemporal choices, they are more patient when future times are linked to concrete events (e.g., a holiday) than when they are expressed only with respect to the delay until receipt (e.g., in 3 months; Peters & Büchel, 2010). This suggests that controlled processes interact not only with valuation but also with cognitive functions such as memory, underscoring the need to expand existing two-system models of intertemporal choice.

Emotion Regulation in Intertemporal Choice

We face intertemporal dilemmas every day and employ a host of strategies to overcome temptations. In this section we discuss a few of those strategies and relate them to the brain systems and cognitive processes on which they impinge.

The most famous strategy for avoiding temptations is termed "precommitment," in which one commits to a course of action prior to the actual time of choice to avoid responding impulsively. One famous example of precommitment involves a product that banks used to offer, known as "Christmas Clubs" (Strotz, 1956). Christmas Clubs were savings accounts that offered no interest and forbade withdrawals until just before Christmas. They were popular; people will-

fully signed up to have access to their money restricted for months at a time, with no compensation, so that they could afford to buy presents at the holidays. Analogous programs for precommitment continue to exist today. For example, people pay money to join weight loss programs that restrict their calorie intake, and they join the military to improve self-discipline. Furthermore, there now exist websites where one can sign up to be punished financially for not accomplishing some goal (e.g., *stickk.com* and *joesgoals.com*).

Precommitment devices are extremely effective. For example, people ordinarily perform abysmally at saving for retirement. However, savings rates increase dramatically when retirement plans include a precommitment to increase savings progressively by committing a portion of future raises (Thaler & Benartzi, 2004). Additionally, when we understand that we have "weaknesses," we often take actions in anticipation of this fact. Since precommitment requires anticipation and planning about future situations, we suspect that precommitment depends on the involvement of the dlPFC and hippocampus, because concretely contemplating the future depends on these structures.

In the absence of precommitment, when we confront tempting stimuli, a number of strategies can be applied to curb our emotions and help us respond appropriately. For example, in a recent study, people were scanned while they attempted cognitively to regulate their cravings and to make decisions about whether to eat unhealthy foods (Hutcherson et al., 2012). As expected, participants made more healthy decisions when actively regulating their cravings, and they showed less controlled decision making when they focused on the pleasurable aspects of the food options. Furthermore, active regulation of cravings was associated with decreased activity in regions of the brain associated with automatic valuation, whereas value-related activation increased when these cravings were indulged. Thus, active regulation of our emotional responding can facilitate controlled decision making by suppressing the brain's automatic value responses.

Similarly, in one particularly revealing study by Mischel, Shoda, and Rodriguez (1989), children chose between receiving one reward now and receiving two rewards at some indeterminate time in the future. This delay is ordinarily very difficult for children to endure; however, Mischel et al. found that instructing children to think of the abstract qualities of the rewards (thereby reducing their desirability) dramatically improved their ability to wait for the better outcome. Furthermore, children who were worse at delaying gratification expressed greater activation in reward-related regions when attempting to suppress responses to an alluring cue (when studied again as adults; Casey et al., 2011). This provides further evidence that suppression of reward-related activation is an important factor in controlled decision making during intertemporal choice.

A number of other strategies have been shown to help regulate decision making during intertemporal choice. For example, based on the work by Peters and Büchel (2010) discussed earlier, thinking of the future in a more concrete manner can increase the appeal of obtaining a delayed reward. Such a change in the way future reward is construed may compensate for the fact that future benefits tend to be thought of in very general terms that do not have the same appeal as the much more precisely considered immediate outcomes (Trope & Liberman, 2003). Indeed, priming people to think more concretely also makes them more patient (Fujita & Han, 2009).

Another strategy that has been shown to regulate decision making in intertemporal choice is focusing one's attention on the future consequences of an action. Consider the manipulation discovered by Magen, Dweck, and Gross (2008). Usually, intertemporal choices are presented as decisions between smaller, more immediate rewards (i.e., $10 available right now) and larger, delayed rewards (i.e., $20 available in a month). Of course, it is equivalent to reexpress this choice as either $10 now and nothing in a month or nothing now but $20 in a month. In this "explicit zero" framing, people are significantly more patient. Recently, we examined the mechanisms underlying this effect. Based on our studies, we believe that expressing the future consequences of immediate reward forces people to think about the future, thereby reducing a natural present bias that leads to underconsideration of future desires. In fact, priming people to

think about the future is sufficient to reduce impulsivity in intertemporal choice (Radu, Yi, Bickel, Gross, & McClure, 2011).

There are undoubtedly many ways that one can reframe alluring immediate rewards to reduce impulsivity. Furthermore, there are numerous known influences on delay discount rates that remain unexplained, such as the "date-delay effect," in which people are more patient when future times are expressed as exact dates (i.e., July 25 vs. 30 days from now), or the fact that people are more patient when deciding between larger rather than smaller rewards (e.g., Thaler, 1981; Read, Frederick, Orsel, & Rahman, 2005). We suspect that these effects work through a common pathway that ultimately suppresses reward-related activation in the NAcc, as meliorating this myopic and impulsive signal could facilitate far-sighted behavior. Dampening of NAcc responses can occur either exogenously (i.e., by presenting rewards in different ways) or endogenously (i.e., by deliberatively reconstruing a tempting outcome). Identifying the precise mechanisms by which these manipulations work to reduce impulsivity is an exciting future direction in this research domain.

Conclusions

Delay discounting encompasses a spectrum of important behaviors in which emotion regulation strategies have important consequences, particularly for meliorating impulsivity. Our aim has been to illustrate cognitive approaches for understanding how delay discounting occurs, with a particular emphasis on two-systems models. These models posit the existence of an automatic, affect-laden set of processes that interact with a more flexible, deliberate system to govern our behavior.

Studying delay discounting from the purview of neuroscience is exciting for a number of reasons. First, neuroscience offers a complementary empirical approach for establishing the function of constructs such as valuation and self-control in decision making. Following on this, neuroscience contributes additional details to a mechanistic understanding of delay discounting. For example, we originally identified vmPFC and dACC as components of automatic valuation and controlled decision making, respectively, based on analyses that sought to identify qualities of these hypothesized cognitive processes. Subsequent work has refined our understanding of these structures. We now have more detailed understanding of how self-control works (via dACC–dlPFC interactions) and how valuation and self-control interact (in part, via the vmPFC). Our suspicion is that over the next several years the nature of these kinds of processes will be even better understood, and our conceptualization of the overall architecture supporting intertemporal decision making will be expanded to include additional brain systems and cognitive processes.

Although we have a basic understanding of how emotion regulation impacts neural activation during intertemporal choice, further research is necessary to refine our understanding of how different regulatory strategies function with respect to the brain. This will give us a better understanding of how known regulatory strategies work, allow us to understand better individual differences in impulsivity, and provide a framework for developing ever more effective strategies to overcome problems with self-control. Addiction, obesity, and other self-control problems are rife in our society. They can all be conceptualized through the framework of intertemporal choice, and a more detailed mechanistic understanding of delay discounting can only facilitate efforts to combat these problems.

References

Ainslie, G. (1975). Specious reward: A behavioral theory of impulsiveness and impulse control. *Psychological Bulletin, 82*, 463–496.

Baker, S. C., Rogers, R. D., Owen, A. M., Frith, C. D., Dolan, R. J., Frackowiak, R. S. J., et al. (1996). Neural systems engaged by planning: A PET study of the Tower of London task. *Neuropsychologia, 34*, 515–526.

Barkley, R. A., Edwards, G., Laneri, M., Fletcher, K., & Metevia, L. (2001). Executive functioning, temporal discounting, and sense of time in adolescents with attention deficit hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD). *Journal of Abnormal Child Psychology, 29*, 541–556.

Bechara, A., Damasio, H., Damasio, A. R., &

Lee, G. P. (1999). Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *Journal of Neuroscience, 19*, 5473–5481.

Bergh, B. V. D., Dewitte, S., & Warlop, L. (2008). Bikinis instigate generalized impatience in intertemporal choice. *Journal of Consumer Research, 35*, 85–97.

Berridge, K. C., Robinson, T. E., & Aldridge, J. W. (2009). Dissecting components of reward: "Liking," "wanting," and learning. *Current Opinion in Pharmacology, 9*, 65–73.

Bickel, W. K., Jarmolowicz, D. P., Mueller, E. T., Koffarnus, M. N., & Gatchalian, K. M. (2012). Excessive discounting of delayed reinforcers as a trans-disease process contributing to addiction and other disease-related vulnerabilities: Emerging evidence. *Pharmacology and Therapeutics, 134*, 287–297.

Blanchard, D. C., & Blanchard, R. J. (1972). Innate and conditioned reactions to threat in rats with amygdaloid lesions. *Journal of Comparative and Physiological Psychology, 81*, 281–290.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*, 624–652.

Burgess, N., Maguire, E. A., & O'Keefe, J. (2002). The human hippocampus and spatial and episodic memory. *Neuron, 35*, 625–641.

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1995). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science, 280*, 747–749.

Casey, B. J., Somerville, L. H., Gotlib, I. H., Ayduk, O., Franklin, N. T., Askren, M. K., et al. (2011). Behavioral and neural correlates of delay of gratification 40 years later. *Proceedings of the National Academy of Sciences, 108*(36), 14998–15003.

Chaiken, S., & Trope, Y. (Eds.). (1999). *Dual-process theories in social psychology.* New York: Guilford Press.

Cohen, J. D., Dunbar, K., & McClelland, J. L. (1990). On the control of automatic processes: A parallel distributed processing account of the Stroop effect. *Psychological Review, 97*, 332–361.

Cohen, J. D., Perlstein, W. M., Braver, T. S., Nystrom, L. E., Noll, D. C., Jonides, J., et al. (1997). Temporal dynamics of brain activation during a working memory task. *Nature, 386*, 604–608.

Damasio, A. (1994). *Descartes' error.* New York: Penguin Books.

Dawe, S., & Loxton, N. J. (2004). The role of impulsivity in the development of substance use and eating disorders. *Neuroscience and Biobehavioral Reviews, 28*(3), 343–351.

Delgado, M. R. (2007). Reward-related responses in the human striatum. *Annals of the New York Academy of Sciences, 1104*, 70–88.

Duncan, J., Seitz, R. J., Kolodny, J., Bor, D., Herzog, H., Ahmed, A., et al. (2000). A neural basis for general intelligence. *Science, 289*, 457–460.

Eslinger, P. J., & Damasio, A. R. (1985). Severe disturbance of higher cognition after bilateral frontal lobe ablation: Patient EVR. *Neurology, 35*, 1731–1741.

Figner, B., Knoch, D., Johnson, E. J., Krosch, A. R., Lisanby, S. H., Fehr, E., & Weber, E. U. (2010). Lateral prefrontal cortex and self-control in intertemporal choice. *Nature Neuroscience, 13*, 538–539.

Frederick, S., Loewenstein, G., & O'Donoghue, T. (2003). Time discounting and time preference: A critical review. In G. Loewenstein, D. Read, & R. Baumeister (Eds.), *Decision and time* (pp. 13–86). New York: Russell Sage Foundation.

Fujita, K., & Han, H. (2009). Moving beyond deliberative control of impulses: The effect of construal levels on evaluative associations in self-control conflicts. *Psychological Science, 20*, 799–804.

Gallistel, C. R., Shizgal, P., & Yeomans, J. S. (1981). A portrait of the substrate for self-stimulation. *Psychological Review, 88*, 228–273.

Giordano, L. A., Bickel, W. K., Loewenstein, G., Jacobs, E. A., Marsch, L., & Badger, G. J. (2002). Opioid deprivation affects how opioid-dependent outpatients discount the value of delayed heroin and money. *Psychopharmacology, 163*, 174–182.

Gratton, G., Coles, M. G., & Donchin, E. (1992). Optimizing the use of information: Strategic control of activation of responses. *Journal of Experimental Psychology: General, 121*, 480–506.

Green, L., Fry, A., & Myerson, J. (1994). Discounting of delayed rewards: A life-span comparison. *Psychological Science, 5*, 33–36.

Greene, J. D., Nystrom, L. E., Engell, A. D., Darley, J. M., & Cohen, J. D. (2004). The neural bases of cognitive conflict and control in moral judgment. *Neuron, 44*, 389–400.

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224–237.

Hamann, S. B., Ely, T. D., Hoffman, J. M., & Kilts, C. D. (2002). Ecstasy and agony: Activation of the human amygdala in positive and negative emotion. *Psychological Science*, *13*(2), 135–141.

Hare, T. A., Camerer, C. F., & Rangel, A. (2009). Self-control in decision-making involves modulation of the VMPFC valuation system. *Science, 324*, 646–648.

Hariri, A. R., Brown, S. M., Williamson, D. E., Flory, J. D., de Wit, H., & Manuck, S. B. (2006). Preference for immediate over delayed rewards is associated with magnitude of ventral striatal activity. *Journal of Neuroscience, 26*, 13213–13217.

Hershberger, W. A. (1986). An approach through the looking glass. *Animal Learning and Behavior, 14*, 443–451.

Hoch, S. J., & Loewenstein, G. F. (1991). Time-inconsistent preferences and consumer self-control. *Journal of Consumer Research, 17*, 492–507.

Hutcherson, C. A., Plassmann, H., Gross, J. J., & Rangel, A. (2012). Cognitive regulation during decision-making shifts behavioral control between ventromedial and dorsolateral prefrontal value systems. *Journal of Neuroscience, 32*(39), 13543–13554.

Ikemoto, S., & Panksepp, J. (1999). The role of nucleus accumbens dopamine in motivated behavior: A unifying interpretation with special reference to reward-seeking. *Brain Research Reviews, 31*, 6–41.

Jog, M. S., Kubota, Y., Connolly, C. I., Hillegaart, V., & Graybiel, A. M. (1999). Building neural representations of habits. *Science, 286*, 1745–1749.

Kable, J. W., & Glimcher, P. W. (2007). The neural correlates of subjective value during intertemporal choice. *Nature Neuroscience, 10*, 1625–1633.

Kable, J. W., & Glimcher, P. W. (2009). The neurobiology of decision: Consensus and controversy. *Neuron, 63*(6), 733–745.

Kahneman, D. (2003). Maps of bounded rationality: Psychology for behavioral economics. *American Economic Review, 93*, 1449–1475.

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science, 303*, 1023–1026.

Kirby, K. N. (1997). Bidding on the future: Evidence against normative discounting of delayed rewards. *Journal of Experimental Psychology: General, 126*, 54–70.

Knutson, B., Adams, C. M., Fong, G. W., & Hommer, D. (2001). Anticipation of increasing monetary reward selectively recruits nucleus accumbens. *Journal of Neuroscience, 21*, RC159.

Knutson, B., Fong, G. W., Bennett, S. M., Adams, C. M., & Hommer, D. (2003). A region of mesial prefrontal cortex tracks monetarily rewarding outcomes: Characterization with rapid event-related fMRI. *NeuroImage, 18*, 263–272.

Koob, G. F., & Bloom, F. E. (1988). Cellular and molecular mechanisms of drug dependence. *Science, 242*, 715–723.

Kroger, J. K., Sabb, F. W., Fales, C. L., Bookheimer, S. Y., Cohen, M. S., & Holyoak, K. J. (2002). Recruitment of anterior dorsolateral prefrontal cortex in human reasoning: A parametric study of relational complexity. *Cerebral Cortex, 12*, 477–485.

LaBar, K. S., Gatenby, J. C., Gore, J. C., LeDoux, J. E., & Phelps, E. A. (1998). Human amygdala activation during conditioned fear acquisition and extinction: A mixed-trial fMRI study. *Neuron, 20*, 937–945.

Laibson, D. I. (1997). Golden eggs and hyperbolic discounting. *Quarterly Journal of Economics, 42*, 861–871.

LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience, 23*(1), 155–184.

Levy, B. J., & Anderson, M. C. (2002). Inhibitory processes and the control of memory retrieval. *Trends in Cognitive Sciences, 6*, 299–305.

Li, X. (2008). The effects of appetitive stimuli on out-of-domain consumption impatience. *Journal of Consumer Research, 34*, 649–656.

Lieberman, M. D., Gaunt, R., Gilbert, D. T., Trope, Y. (2002). Reflection and reflexion: A social cognitive neuroscience approach to attributional inference. *Advances in Experimental Social Psychology, 34*, 199–249.

Loewenstein, G. (1996). Out of control: Visceral influences on behavior. *Organizational Behavior and Human Decision Processes, 65*, 272–293.

Loewenstein, G., & Prelec, D. (1992). Anoma-

lies in intertemporal choice: Evidence and an interpretation. *Quarterly Journal of Economics, 107*, 573–597.

MacDonald, A. W., Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000). Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science, 288*, 1835–1838.

MacLean, P. D. (1990). *The triune brain in evolution: Role in paleocerebral functions*. New York: Plenum Press.

Magen, E., Dweck, C. S., & Gross, J. J. (2008). The hidden-zero effect: Representing a single choice as an extended sequence reduces impulsive choice. *Psychological Science, 19*, 648–649.

Marco-Pallarés, J., Mohammadi, B., Samii, A., & Münte, T. F. (2010). Brain activations reflect individual discount rates in intertemporal choice. *Brain Research, 1320*, 123–129.

McClure, S. M., Berns, G. S., & Montague, P. R. (2003). Temporal prediction errors in a passive learning task activate human striatum. *Neuron, 38*, 339–346.

McClure, S. M., Ericson, K. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. (2007). Time discounting for primary rewards. *Journal of Neuroscience, 27*, 5796–5804.

McClure, S. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. (2004). Separate neural systems value immediate and delayed monetary rewards. *Science, 306*, 503–507.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*, 167–202.

Mischel, W., Ayduk, O., & Mendoza-Denton, R. (2003). Sustaining delay of gratification over time: A hot-cool systems perspective. In G. Loewenstein, D. Read, & R. Baumeister (Eds.), *Decision and time* (pp. 175–200). New York: Russell Sage Foundation.

Mischel, W., Shoda, Y., & Rodriguez, M. L. (1989). Delay of gratification in children. *Science, 244*, 933–938.

Olds, J. (1977). *Drives and reinforcements: Behavioral studies of hypothalamic function*. New York: Raven Press.

Owen, A. M., McMillan, K. M., Laird, A. R., & Bullmore, E. (2005). N-back working memory paradigm: A meta-analysis of normative functional neuroimaging studies. *Human Brain Mapping, 25*, 46–59.

Pessiglione, M., Petrovic, P., Daunizeau, J., Palminteri, S., Dolan, R. J., & Frith, C. D. (2008). Subliminal instrumental conditioning demonstrated in the human brain. *Neuron, 59*, 561–567.

Peters, J., & Büchel, C. (2010). Episodic future thinking reduces reward delay discounting through an enhancement of prefrontal–mediotemporal interactions. *Neuron, 66*, 138–148.

Peters, J. & Büchel, C. (2011). The neural mechanisms of inter-temporal decision-making: Understanding variability. *Trends in Cognitive Sciences, 15*, 227–239.

Phan, K. L., Fitzgerald, D. A., Nathan, P. J., & Tancer, M. E. (2006). Association between amygdala hyperactivity to harsh faces and severity of social anxiety in generalized social phobia. *Biological Psychiatry, 59*(5), 424–429.

Phelps, E. A. (2006). Emotion and cognition: Insights from studies of the human amygdala. *Annual Review of Psychology, 57*, 27–53.

Phelps, E. A., O'Connor, K. J., Gatenby, J. C., Gore, J. C., Grillon, C., & Davis, M. (2001). Activation of the left amygdala to a cognitive representation of fear. *Nature Neuroscience, 4*, 437–441.

Philiastides, M. G., Auksztulewicz, R., Heekeren, H. R., & Blankenburg, F. (2011). Causal role of dorsolateral prefrontal cortex in human perceptual decision making. *Current Biology, 21*(11), 980–983.

Phillips, R. G., & LeDoux, J. E. (1992). Differential contribution of amygdala and hippocampus to cued and contextual fear conditioning. *Behavioral Neuroscience, 106*, 274–285.

Pine, A., Shiner, T., Seymour, B., & Dolan, R. J. (2010). Dopamine, time, and impulsivity in humans. *Journal of Neuroscience, 30*, 8888–8896.

Posner, M. I., & Snyder, C. R. R. (1975). Attention and cognitive control. In R. L. Solso (Ed.), *Information processing and cognition* (pp. 55–85). Hillsdale, NJ: Erlbaum.

Radu, P. T., Yi, R., Bickel, W. K., Gross, J. J., & McClure, S. M. (2011). A mechanism for reducing delay discounting by altering temporal attention. *Journal of the Experimental Analysis of Behavior, 96*, 363–385.

Rangel, A., Camerer, C. F., & Montague, P. R. (2008). A framework for studying the neurobiology of value-based decision-making. *Nature Reviews Neuroscience, 9*, 545–556.

Read, D., Frederick, S., Orsel, B., & Rahman, J. (2005). Four Score and Seven Years from Now: The date/delay effect in temporal discounting. *Management Science, 51*(9), 1326–1335.

Redish, A. D. (2004). Addiction as a computational process gone awry. *Science, 306*, 1944–1947.

Ridderinkhof, K. R. (2004). The role of the medial frontal cortex in cognitive control. *Science, 306*(5695), 443–447.

Sanfey, A. G., Rilling, J. K., Aronson, J. A., Nystrom, L. E., & Cohen, J. D. (2003). The neural basis of economic decision-making in the ultimatum game. *Science, 300*, 1755–1758.

Schacter, D. L., Addis, D. R., & Buckner, R. L. (2008). Remembering the past to imagine the future: The prospective brain. *Nature Reviews Neuroscience, 8*, 657–661.

Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science, 275*, 1593–1599.

Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery, and Psychiatry, 20*, 11–21.

Shallice, T., & Burgess, P. W. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain, 114*, 727–741.

Shamosh, N. A., DeYoung, C. G., Green, A. E., Reis, D. L., Johnson, M. R., Conway, A. R. A., et al. (2008). Individual differences in delay discounting: Relation to intelligence, working memory, and anterior prefrontal cortex. *Psychological Science, 19*, 904–911.

Shiffrin, R. M., & Snyder, W. (1977). Controlled and automatic processing: II. Perceptual learning, automatic attending, and a general theory. *Psychological Review, 84*, 127–190.

Shizgal, P., & Conover, K. (1996). On the neural computation of utility. *Current Directions in Psychological Science, 5*, 37–43.

Simon, H. A. (1959). Theories of decision-making in economics and behavioral science. *American Economic Review, 49*, 253–283.

Strotz, R. (1956). Myopia and inconsistency in dynamic utility maximization. *Review of Economic Studies, 23*, 165–180.

Stuss, D. T., & Levine, B. (2002). Adult clinical neuropsychology: Lessons from studies of the frontal lobes. *Annual Review in Psychology, 53*, 401–433.

Thaler, R. H. (1981). Some empirical evidence on time inconsistency. *Review of Economic Studies, 23*, 165–180.

Thaler, R. H., & Benartzi, S. (2004). Save More Tomorrow™: Using behavioral economics to increase employee saving. *Journal of Political Economy, 112*, S164–S187.

Thaler, R. H., & Shefrin, H. (1981). An economic theory of self-control. *Journal of Political Economy, 89*, 392–406.

Trope, Y., & Liberman, N. (2003). Temporal construal. *Psychological Review, 110*, 403–421.

van den Bos, W., & McClure, S. M. (2013). Towards a general model of delay discounting. *Journal of the Experimental Analysis of Behavior, 99*, 58–73.

Wang, X. T., & Dvorak, R. D. (2010). Sweet future: Fluctuating blood glucose levels affect future discounting. *Psychological Science, 21*, 183–188.

Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike, M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *Journal of Neuroscience, 18*, 411–418.

Wicker, B., Keysers, C., Plailly, J., Royet, J. P., Gallese, V., & Rizzolatti, G. (2003). Both of us disgusted in my insula: The common neural basis of seeing and feeling disgust. *Neuron, 40*, 655–664.

Wise, R. A. (1982). Neuroleptics and operant behavior: The anhedonia hypothesis. *Behavioral and Brain Sciences, 5*, 39–87.

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review, 111*(4), 931–959.

CHAPTER 7

The Role of Emotion and Emotion Regulation in the Ability to Delay Gratification

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You would be hard pressed to find a psychologist, professional or budding, who is not familiar with research on delay of gratification (DG)—the ability to put off a small, but immediate, reward in favor of receiving a larger reward later (e.g., Mischel & Ayduk, 2011). In fact, more and more, this work is being written about in the popular media (e.g., Brooks, 2006; Lehrer, 2009), and thus likely discussed in nonacademic circles, such as parents, educators, and clinicians. What makes DG, as a topic, so interesting? This fascination is likely due to the longitudinal studies linking performance on the classic DG paradigm, during which a child tries to wait for two treats instead of having one right away, to a variety of critical outcomes in later life, including drug use, social competence, and even Scholastic Aptitude Test (SAT) scores (e.g., Mischel, Shoda, & Rodriguez, 1989).

Although this early longitudinal research popularized DG for the general public, researchers have certainly expanded beyond it. For example, research has linked DG to other forms of cognitive control (e.g., Casey et al., 2011; Eigsti et al., 2006), identified mechanisms that underlie DG ability (e.g., Rodriguez, Mischel, & Shoda, 1989), and examined how these processes play out in adults, with whom treats are no longer enticing rewards (e.g., Green, Fry, & Myerson, 1994). The bulk of this work, however, has most often described and discussed DG as a form of *behavioral* self-control and focused on its cognitive and attentional underpinnings.

More recently, there has been a growing body of research linking DG to affective processes—emotional experience and emotion regulation. In this chapter, we examine these findings. To do so, we first provide a brief overview of DG, including its measurement across different age groups and a description of the most studied mechanism thought to underlie performance. Subsequently, we review four bodies of research on DG and emotion that in many ways overlap. First, we discuss research on the developmental precursors of DG ability, focusing on how caregivers' emotion expression and responsivity to child distress impact the development of this competency in young children (e.g., Rodriguez et al., 2005). Second, we describe how an individual's own affective state impacts DG—more particularly, the damaging effect of negative emotion on one's ability to put off long-term rewards (e.g., Tice, Baumeister, & Zhang, 2004). Third, we draw on literature suggesting that DG ability may serve an important role in the regulation of emotion—as

a resource for individuals to utilize in emotionally taxing situations (e.g., Ayduk et al., 2000). Finally, we describe neuroimaging research that has provided clues about the links between affective processes and DG (e.g., McClure, Laibson, Loewenstein, & Cohen, 2004).

Review of DG

Early research on DG was conducted primarily with preschool-age children using what has become the classic DG paradigm, referred to fondly as the "marshmallow task" (see Mischel & Ayduk, 2011, for review). First, the experimenter asks the child whether they would prefer a smaller or larger reward (e.g., one marshmallow or two marshmallows). Most children prefer the larger option, and once they say so, the experimenter goes on to explain the task contingencies. If, indeed, the child would like to have the larger reward, he or she must wait until the experimenter returns to the room, ostensibly after setting up another task. The child can have the smaller reward anytime, however, by ringing a bell and bringing the experimenter back. The experimenter leaves the child alone with their options sitting on a plate in front of them (e.g., one marshmallow on one side of the plate, two marshmallows on the other side) and the amount of time the child can wait is recorded—the primary measure of DG ability.

Longer wait-time scores on this task have been linked to varied long-term outcomes such as higher SAT scores, lower drug use, and better social-cognitive competence (e.g., Mischel et al., 1989; Shoda, Mischel, & Peake, 1990). As such, researchers have sought to understand what mechanisms underlie the ability of children to wait (e.g., Rodriguez et al., 1989). Experimental and individual-differences research has found that attention deployment during the waiting period is an important predictor of wait time. The more time a child spends looking at the rewards and the bell (i.e., hot-focused attention), the harder it is for him or her to wait. Alternatively, the more time a child spends looking away from the rewards and bell and instead focuses on other things in the room (i.e., cool-focused attention), the easier it is to wait. In this way, contemporary researchers using the classic paradigm often measure both wait time and attention deployment in their evaluation of DG performance.

Although the classic paradigm is the most studied DG measure, it was designed for preschool children and expanded for children up to 11 years of age (Rodriguez et al., 1989). Prior to preschool (before approximately age 4), children typically have neither the cognitive capacity to understand the contingencies of the task nor the ability to wait extended periods of time alone. Alternatively, for adolescents and adults, cookies and marshmallows may not be tempting in the way they are for children. Moreover, the experimental situation with a capped wait time may not tax adults' self-regulatory competencies sufficiently. For these reasons the classic task is unlikely to be diagnostic of individual differences in DG ability after childhood. Given that researchers have wanted to study DG in other age groups, they developed a variety of tasks better suited to these populations.

Researchers evaluating DG ability in very young children often employ variations of either the gift delay or snack delay tasks, both developed by Kochanska and Knaack (2003). These tasks require the child to exert impulse control before obtaining a desired reward. In a standard gift delay task, for example, the experimenter presents a wrapped gift to the child, then has to leave the room and asks the child to wait to open it until he or she returns. The child's behavior is scored with codes ranging from not touching or peeking inside the bag to opening the gift completely. In a typical snack delay task, children have to wait for the experimenter to ring a bell before taking M&M's "hidden" under a clear cup. There are a series of trials that vary in the time delay before the experimenter rings the bell (e.g., 10–30 seconds). Halfway through the delay, the experimenter lifts the cup but does not ring the bell. The child's behavior is observed and coded as waiting, touching, or eating the M&M's before the bell rings, with the assumption that these behaviors reflect decreasing levels of DG ability.

Choice delay tasks are used with adolescents and adults, though many studies also employ these tasks with children. In a standard choice delay task, the participant is asked to make a series of choices between

a smaller, immediate reward and a larger, delayed reward (e.g., $5 today vs. $10 in 1 week) (e.g., Seeman & Schwarz, 1974; Wertheim & Schwarz, 1983). The sizes of the small and large options, as well as the delay intervals, are varied between trials. In some studies, researchers calculate participants' temporal or delay discounting rate—how quickly larger rewards become unappealing when a delay is required to obtain them. For more on choice delay tasks, see Miller, Rodriguez, Kim, and McClure (this volume).

While we do not claim that these tasks perfectly parallel the classic paradigm, in this review we include research using these measures, with the assumption that there is at least a moderate degree of overlap in the psychological conditions between them. In summary, researchers have developed a variety of tasks to evaluate DG ability. In childhood, the classic paradigm or "marshmallow task" is the predominant measure, with both wait time and attention deployment scored. Other measures have been developed for young children and for use with adolescents and adults. In the subsequent review, we link performance on these measures to emotion and emotion regulation.

Caregiver Emotion and Responsivity

Given the connection between DG performance and such varied and critical long-term outcomes, the question of how this ability develops becomes important. Of particular relevance for our discussion of DG and emotion is whether there is an association between affective environments in early childhood and the successful development of DG ability later on.

Researchers have found that caregiver variables, including caregiver emotion and caregiver responsivity to children's emotion, are important correlates of subsequent DG development. These variables are often interrelated or measured interchangeably—with the nature of caregivers' expressed emotion serving as a common signal of whether the caregiver is responsive to the child's emotion. In one such study, Eiden, Edwards, and Leonard (2007) coded parental affect from videos of the parent and child interacting at home when the child was age 2. DG was measured 1 year later with a composite of performance on age-appropriate DG tasks, including the snack and gift delay tasks. Children of parents who displayed lower levels of positive affect were worse at DG 1 year later. In a similar study, maternal responsiveness to a child's verbal communications was coded during home visits when the child was age 24 months. The more responsive his or her mother was, the more patient and task-oriented the child was on a gift delay task 1 year later (Olson, Bates, & Bayles, 1990).

Houck and Lecuyer-Maus (2004) had similar results when evaluating the association between maternal responsivity and DG behavior in a narrower context—during child limit setting. During laboratory sessions at child ages 12, 24, and 36 months, parents were asked to prevent their child from touching and playing with an attractive toy. The researchers coded four limit-setting approaches, which varied in the degree of parent responsivity to the child's emotion. Children of parents who predominantly used a power-based limit-setting approach, characterized by little responsivity and high negative affect, waited the least amount of time during the DG task, significantly less than children whose parents' approaches were more responsive to the child's feelings.

Rodriguez and colleagues (2005) also evaluated the role of maternal responsivity in their study linking behaviors during the Strange Situation paradigm (used to measure caregiver–child attachment) when the child was 18 months old to DG ability at age 4 (Rodriguez et al., 2005). Maternal responsiveness during the free-play and reunion episodes was coded using facial and vocal expression, position and body contact, and expression of affection, among other variables. In addition, during the reunion episode, the researchers identified instances when a mother disengaged from her child specifically following the child's expression of negative affect. Children of unresponsive and disengaged mothers waited marginally less time for a larger reward, and spent more time directing their attention toward the rewards and bell during the waiting period, which, again, is an important mechanism underlying DG ability (Rodriguez et al., 1989).

In fact, children's attachment style with their caregivers, a more general reflection of caregiver emotion and responsivity (Shaver

& Mikulincer, 2011), has also been correlated with DG performance. For example, in one study, children who were more securely attached to their caregivers waited the longest on the classic DG task (Jacobsen, Huss, Fendrich, Kruesi, & Ziegenhain, 1997). The greatest difference in wait time was found between children who displayed a secure attachment pattern and those who displayed a disorganized attachment pattern (characterized by fearful and disorganized responses during the reunion phase). For more on attachment and emotion, see Shaver and Mikulincer (this volume).

A variety of studies have corroborated the finding that a caregiver's hostile, overcontrolling behavior is associated with poor DG ability. Jacobsen (1998) coded expressed emotion during a 5-minute speech in which mothers were asked to describe what their child was like. Children of mothers who expressed criticism (resentment of the child's behavior or characteristics) waited less time on the classic DG paradigm. In another study, researchers coded caregivers' behavior for directives (commands) during a free-play time, along with take-overs (completing the game themselves) and contingent positive affect (responding to the child's correct response with affect signaling approval) during a teaching–learning task (Silverman & Ippolito, 1995). Children of mothers who used directives and engaged in take-overs performed worse on age-appropriate versions of the DG task, including the snack and gift delay tasks, 6 months later. Children whose mothers engaged in contingent positive affect were better at DG.

Other studies have provided evidence that a caregiver's approach to emotion more generally relates to DG development. Brophy-Herb, Stansbury, Bocknek, and Horodynski (2011) evaluated the relation between children's DG and a variety of parental *emotion-related socialization behaviors* (ERSBs)—parenting behaviors thought to be critical for a child's socioemotional development. ERSBs were scored during parent–child interactions and with questionnaires. These included positive emotional responsivity and general expressivity in the home, among other measures. Higher scores on the ERSB composite factor (more emotion-related socialization) positively predicted performance on age-appropriate versions of the DG task, including the snack and gift delay tasks.

Collectively, these studies suggest that caregiver emotion and responsivity are associated with the development of DG ability in childhood. But why exactly is a home life characterized by caregiver positive affect, limited hostility, and clear awareness and sensitivity to a child's emotion related to superior DG? How do these affective variables relate to the development of the ability to put off immediate gratification in order to achieve long-term goals? Though there have been little to no studies exploring this question of mechanism, researchers have put forth a few hypotheses, as described below.

Children's negative affect, emerging in the context of stress or frustration, is likely short-circuited by responsive caregivers. These caregivers attend to their child's emotional needs and may provide support, for example, through soothing or distracting behavior (e.g., Garner, 2006; Jahromi & Stifter, 2007). This external source of emotion regulation may serve dual functions. In the immediate, it helps to mitigate children's negative affective states. As further described in the next section, negative emotion can destabilize DG (e.g., Mischel, Ebbesen, & Zeiss, 1972).

At a more global level, the external forms of regulation provided by responsive caregivers serve an important modeling function. Children of responsive caregivers may have more opportunities to observe and learn successful emotion regulation strategies that they themselves can employ at an appropriate age (e.g., Eisenberg, Fabes, & Murphy, 1996; Garner, 2006). These skills may be useful for successful DG, which is in itself a frustrating task. In contrast, children who lack such regulatory models may not develop these skills, therefore becoming more vulnerable to DG failure.

Caregivers' own affective profiles may serve to intensify these processes. Whereas warm caregivers who score high in positive affect likewise engender positive affect in their children, caregivers who score high in negative affect regularly respond to their children's emotions and behaviors in negative ways, thereby maintaining or even escalating their children's negative affect (Malatesta, Culver, Tesman, & Shepard, 1989). Over time, a caregiver's character-

istic affective tone serves as a model for the development of a child's own affective profile—teaching the child which emotional responses are appropriate across a range of situations (e.g., Eisenberg et al., 1999, 2003; Garner, 1995). Children with more chronic negative affect may be regularly diverting resources from DG goals, toward the regulation of their negative affect.

In summary, warm and responsive caregivers may raise children with high positive affect and low negative affect, who are armed with a host of regulation strategies, whereas cold and distant caregivers may raise children with low positive affect and high negative affect, who have fewer regulatory strategies. Future research should evaluate these hypotheses more directly, for example, by linking parental emotion and regulatory profiles to the types of affective reactions and regulatory strategies employed by children during the DG task. For more on the role of family on emotion, see Thompson (this volume).

Affective State and DG

Does an individual's prior affective state impact his or her DG ability? For example, how might children perform if they started the classic DG task right after learning that they received a failing grade on a test? What if they had earned the top score in their class? As briefly mentioned in the previous section, researchers have found that negative affect, in particular, is associated with poor performance on both laboratory measures of DG and during real-world DG scenarios, such as dietary restraint. The evidence on positive affect is more mixed: Some studies indicate that positive affect helps DG; others find that it hurts DG; and still others find that it does not exert an impact all that different from neutral emotional states.

Negative Affect

Analyses conducted with various laboratory measures have collectively supported the prediction that negative emotion is associated with poor DG performance. In an early study, researchers hypothesized that behaviors, or even thoughts, that successfully distract the child from the frustrating nature of the classic DG task will help the child wait for the larger reward (Mischel et al., 1972). Indeed, children directed to think "fun" thoughts during the task (e.g., playing with toys) did wait longer. However, the content of the distraction was found to matter. Directing children to think "sad" thoughts (e.g., crying with no one to help you) actually hurt wait time. Children performed at the same poor level when directed to think sad thoughts, as when they were directed to focus their attention on the rewards themselves, that is, hot attentional focus.

Similar results have been found with choice delay tasks (e.g., Schwarz & Pollack, 1977). For example, in one study, 9-year-old participants drew a picture, and then went through either a positive affect induction—receiving feedback that the drawing was good and would be featured in an art exhibit, or a negative affect induction—receiving feedback that the drawing was not good and would not be featured (Seeman & Schwarz, 1974). Subsequently, they were asked a series of choice delay questions. Participants in the negative affect condition chose large, delayed rewards significantly less often than participants in the positive affect condition. In a similar study, participants, ages 3 to 5 years, directed to think sad thoughts were more likely to choose an immediate, mediocre reward over a desirable, delayed reward than were children in either neutral or positive affect conditions (Moore, Clyburn, & Underwood, 1976). Similar results have been found with adults (Gray, 1999). In one study, participants in a negative affective state (exposed to aversive pictures; in a state of heightened stress about upcoming midterms) showed a preference for short-term gains at the expense of long-term gains, thus performing worse on the task overall.

Individual-difference studies have corroborated this body of experimental work. For example, developmental researchers have argued that temperament (biologically based personality differences; see Rothbart, Sheese, & Posner, 2007, for review) is comprised of three factors: arousal, emotion, and self-regulation. These researchers found that temperamental levels of negative affectivity are inversely related to temperamental levels of self-regulation (Derryberry & Rothbart, 1988). Relatedly, in studies with toddlers,

more frequent negative affect during the separation (Rodriguez et al., 2005; Sethi, Mischel, Aber, Shoda, & Rodriguez, 2000) and reunion (Rodriguez et al., 2005) phases of the Strange Situation procedure is associated with more hot attentional focus (looking toward the rewards and bell) during the classic DG task at ages 4 and 5. Moreover, in a choice delay task, mild and moderately depressed adults were more likely to choose immediate, less desirable rewards over large, delayed rewards than individuals who were not depressed (Wertheim & Schwarz, 1983).

Similar effects have been found in more real-world DG scenarios. For example, dietary restraint, like laboratory measures of DG, requires an individual to put off short-term gratification (e.g., the pleasure derived from eating delicious foods), for the sake of a long-term goal (e.g., losing weight, being healthy). Because restrained eaters are constantly trying to delay gratification to obtain a thinner and healthier body, how their eating changes in response to their current affective state can inform our understanding of how affective states impact DG performance more generally. According to the restraint theory of Herman and Polivy (1975), negative affect increases eating in restrained eaters (the reasons for which we evaluate below).

In an experimental evaluation of this prediction, Frost, Goolkasian, Ely, and Blanchard (1982) had participants, who varied on degree of dietary restraint, go through either a negative, positive, or neutral affect induction. During this affect induction, a bowl of M&M's was placed in front of participants, and they were told to eat freely from the bowl. Restrained eaters in the negative affect condition ate more M&M's than nonrestrained eaters in the same condition, and ate more than restrained eaters in both the positive and neutral affect conditions. Though there was not a significant difference in the eating behavior of nonrestrained eaters across conditions, they appeared to eat the least when in a negative affective state.

Baucom and Aiken (1981) had similar results. Their participants, obese and nonobese, restrained and nonrestrained eaters, were asked to solve a task and received either success feedback (a positive affect induction) or failure feedback (a negative affect induction). Participants were then asked to complete an ostensible taste test study in which they tried different crackers and rated their flavor preferences. Among restrained eaters, participants who received failure feedback ate significantly more crackers than those who received success feedback. In contrast, nonrestrained eaters ate marginally fewer crackers after failure feedback than after success feedback. This pattern of results held for both obese and nonobese participants. The link between negative affect and weakened dietary restraint was also found among participants experiencing clinical depression (characterized by strong negative affect; Polivy & Herman, 1976). In this study, restrained eaters reported a considerable weight gain since the onset of their depression (~6 lbs on average), while nonrestrained eaters reported a considerable weight loss (~5 lbs on average), with the difference between them highly significant.

Collectively, both experimental and individual-difference studies, involving classic laboratory measures and more ecologically valid study designs, suggest that negative affect is associated with worse DG performance. A variety of theories have been posited to explain this association. The most common account argues that negative affective states are incredibly distressing, and as such, relief from them may take precedence over long-term goals (e.g., Heatherton & Baumeister, 1991). Energy or resources initially directed toward putting off immediate gratification may be diverted and used toward emotion regulation instead. With fewer resources aimed specifically at the DG attempt, the ability to wait thus suffers (see Baumeister, Vohs, & Tice, 2007, for review). For example, restrained eaters, when put into a negative affective state, may need to divert the self-regulatory resources they typically use to restrict their caloric intake, to regulate their emotions, resulting in more eating. Note, however, that some researchers have argued that individuals who report dieting may in fact be worse at self-regulation more generally (see Pratt & Wardle, 2012, for review).

Moreover, in some DG scenarios a proximal source of affect relief is available—the smaller, but immediately available, option. For example, a child might experience a reduction in his or her sad feelings as a

result of thinking sad thoughts by enjoying an immediately available, albeit small, treat. At a more global level, this suggests that negative affect may change the subjective reinforcement values of immediate and delayed rewards (e.g., Baucom & Aiken, 1981; Schwarz & Pollack, 1977; Seeman & Schwarz, 1974). For individuals in a negative affective state, the value of the more immediate option, though objectively smaller, may increase, because it has the potential to ameliorate negative affect. On the other hand, the value of the long-term goal, or delayed option, is likely to decrease, because it serves less of a purpose in removing the negative affect and may even exacerbate it by adding additional frustration.

Positive Affect

Although the link between DG and negative affect seems well established, the findings on positive affect are more mixed. Some researchers find that, like negative affect, positive affect is associated with poor DG performance. For example, Cools, Schotte, and McNally (1992) exposed participants to a neutral film, a comedy (i.e., positive affect induction), or a horror film (i.e., negative affect induction). Participants were given a bag of popcorn during the movie and told to eat as much as they liked. In the neutral condition, participants higher in dietary restraint ate less popcorn than those lower in dietary restraint. In both the comedy and horror film conditions, however, participants higher in dietary restraint ate more popcorn.

In many other studies, however, participants in a positive affective state performed better at DG tasks than those in a negative affective state. As previously described, children who received positive feedback regarding the quality of their drawings chose larger, delayed rewards more often than those who received negative feedback (Seeman & Schwarz, 1974). Similarly, children completing the classic DG task performed better when asked to think fun, distracting thoughts than when they were directed to think about the rewards (Mischel et al., 1972). On a choice delay task, children in the positive affect condition tended to choose larger, delayed rewards even more often than those in the neutral affect condition, though these results were not statistically significant (Moore et al., 1976). Studies have also shown that putting restrained eaters in a positive or neutral affective state, in comparison to a negative affective state, is associated with greater DG ability (Baucom & Aiken, 1981; Frost et al., 1982). Positive affective states did not consistently improve DG performance more than neutral states, however (Frost et al., 1982).

In summary, the majority of evidence suggests that being in a positive affective state does not hurt DG the way being in a negative affective state does, and in some cases it may even help DG. Although some researchers' findings implied that there are specific conditions under which positive affect may thwart DG, we suspect that positive affect is likely to serve a facilitative function under most conditions. This assumption is consistent with studies linking positive affect to improved self-regulation more generally (e.g., Shmueli & Prochaska, 2012; Tice, Baumeister, Shmueli, & Muraven, 2007; Tice et al., 2004).

Although there is need for more research to firmly establish the facilitative impact of positive affect on DG, what mechanisms might explain such an effect? As previously described, positive affect has been found to restore self-regulatory resources (Tice et al., 2007), suggesting that individuals in a positive affective state may have additional resources available for DG. Moreover, research in support of Fredrickson's broaden-and-build theory of positive emotion, has found that positive affect increases one's attentional focus, which may help transcend the here and now, and thus DG (see Fredrickson, 2001, for review; cf. Gable & Harmon-Jones, 2011). Relatedly, positive affect appears to mitigate the adverse effects of negative affect (e.g., Fredrickson, 2001; Fredrickson & Levenson, 1998), and perhaps by extension the damaging effect negative affect has on DG performance. Finally, it is possible that positive affect has informational value—affirming that things are safe or certain (see Schwarz, 2011, for review), which may lead individuals to be more willing to wait for the delayed rewards during a DG task. Future work should examine these hypotheses directly.

Emotion Regulation and DG

Since the initial studies, researchers have readily assumed that DG tasks induce emotion, and thus by extension, require emotion regulation—the process of changing the way we think, feel, and express emotions (e.g., Gross & Thompson, 2007). Does the way an individual deals with these task-induced emotions impact his or her ability to wait for the larger reward? What about the reverse? Is there an association between an individual's DG ability and how adept he or she is at regulating emotions? Indeed, a large body of research demonstrates that one's DG ability is predictive of emotion regulation behaviors in a variety of emotionally taxing situations.

Whether, and how, children regulate their emotions during DG tasks likely impacts performance. Here we refer to emotions stimulated by the task itself, above and beyond affective states that precede task onset (as described in the previous section). For example, if a child feels frustration and anger that he or she cannot have the two marshmallows immediately, does down-regulating negative affect help or hurt efforts to wait? On the flip side, if a child's predominant affective reaction to the DG situation is eagerness and excitement, does down-regulating such positive emotions facilitate or undermine DG?

Unfortunately, there has been little to no research that provides clear-cut answers to these questions. This shortage may be due, in part, to the difficulty of disentangling emotional reactivity during DG tasks (e.g., how frustrated–excited the individual feels) and emotion regulation (e.g., how well an individual can regulate frustration–excitement if it indeed emerges). One of the few studies evaluating emotion during DG demonstrates this conundrum. Researchers found that children, ages 4–7, who expressed more anger and sadness during three age-appropriate DG tasks, including the gift delay task and a modified version of the classic task, also focused their attention more on the rewards (an important mechanism underlying poor DG performance; Santucci et al., 2008). If more expressed negative emotion reflects less emotion regulation, the results would suggest that regulating negative emotion during the DG task should help performance. It is also possible, however, that children who performed better were not regulating their negative emotion; rather they were less emotionally reactive—and this is why they displayed fewer expressions of anger and sadness. These possibilities need to be disentangled in future research.

Although research that examines emotion regulation during the DG task is scarce, a growing body of work shows that individuals strong in DG ability are better able to regulate their emotions across a variety of situations. In fact, this literature suggests that individuals at risk for emotion regulatory difficulties may be protected from negative life outcomes if they are also good at DG. To illustrate, in one set of studies, researchers evaluated the link between DG and emotion regulation outcomes in people with high rejection sensitivity (RS; Ayduk et al., 2000). These are individuals who anxiously expect, readily perceive, and overreact to social rejection (e.g., Downey & Feldman, 1996). Prior research indicates that individuals with high RS respond to perceived social rejection with dysregulated emotion, including jealousy (Downey & Feldman, 1996), depressed affect (Ayduk, Downey, & Kim, 2001), and hostility directed toward the source of rejection (Ayduk, Downey, Testa, Yen, & Shoda, 1999).

Ayduk and colleagues (2000) hypothesized that individuals with high RS and strong DG ability would be better able to focus on their longer-term goals, such as relationship maintenance, during perceived rejection, and thus be more likely to regulate their reactive emotional responses. In support of this prediction, inner-city middle school students with high RS were rated by their teachers as more aggressive and less socially accepted than those with low RS, but only if they had poor DG ability, as measured with the classic paradigm 2 years earlier. In fact, participants with high RS and good DG were rated as marginally less aggressive and better liked than their peers with low RS. Correspondingly, in a sample of middle-aged participants, those with high RS were rated by their parents as worse at emotion regulation than were those with low RS (e.g., more likely to go to pieces under stress), but again, only if they performed poorly on the classic DG task as preschoolers (Ayduk et al., 2000).

A similar pattern of results was found with respect to borderline personality disor-

der (BPD) symptoms (Ayduk et al., 2008). A core feature of this disorder is extreme affective lability that bears a resemblance to that of high RS, including intense hostility and depression. Ayduk and colleagues hypothesized, and found, that although there was a positive association between RS and BPD symptoms, this association was weaker in individuals with good DG ability, as measured with the classic DG task in childhood.

A host of other studies corroborate these findings—that individuals with good DG ability seem better able to regulate their emotions in a wide range of situations. For example, researchers have found that good DG ability is related to lower verbal and physical aggression (Mischel et al., 1989), along with less cheating behavior (Mischel & Gilligan, 1964). Another study found that adolescents rated by observers as less likely to wait for a larger, but delayed, payment for study participation were also rated higher in hostility (Funder & Block, 1989). Kochanska, Murray, and Harlan (2000) found that among toddlers (i.e., 22–33 months olds), effortful control (of which DG is a component) was related to better regulation of anger, both concurrently and prospectively. Relatedly, inhibitory control ability (similar to effortful control) among preschoolers, in part measured by a gift delay task, positively correlated with emotion regulation in controlling both positive and negative affect (Carlson & Wang, 2007). For more information on emotion regulation and effortful–inhibitory control, see Eisenberg, Hofer, Sulik, and Spinrad (this volume).

Studies with clinical populations yield similar results. In one study, early adolescents with externalizing disorder (characterized by hostility and aggression) performed worse on a modified DG task that involved a series of choices between pressing a button immediately, which provided a 40% chance of winning a nickel, or waiting to press a button, which provided an 80% chance of winning a nickel (Krueger, Caspi, Moffitt, White, & Stouthamer-Loeber, 1996). Mathias and colleagues (2011) found that adolescent girls' preference for small, immediate rewards on a choice delay task was associated with higher self-reported depression, aggression, impulsivity, and suicide intent. In another study, male parolees were presented with a choice between a small monetary reward available after a short, fixed delay and a large monetary reward available after a longer delay (Cherek, Moeller, Dougherty, & Rhoades, 1997). The number of times parolees chose the immediate, small reward was correlated with the number of aggressive responses they made in another paradigm. In a follow-up study, parolees who were imprisoned for violent crimes (e.g., aggravated assault, manslaughter) displayed a preference for small, immediate rewards significantly more than parolees imprisoned for nonviolent crimes (e.g., theft, forgery) (Cherek & Lane, 1999).

Collectively these studies show that there is a strong association between DG and emotion regulation, and they may suggest that both forms of regulation are drawing on a domain-general regulatory competence or resource that can be utilized in a wide variety of situations. In the next section we present evidence in support of this thesis with work evaluating the neural underpinnings of both emotion regulation and DG processes.

Emotion Regulation and DG in the Brain

Why does an individual's DG ability relate to how well they can regulate their emotions? One theory suggests that DG and emotion regulation are related because both reflect an individual's more general self-regulation ability (e.g., Cohen & Lieberman, 2010; Heatherton & Wagner, 2011)—how well the individual is able to overcome prepotent, automatic, or stimulus-driven responses (e.g., Metcalfe & Mischel, 1999). According to this theory, the ability to overcome an automatic response, be it to grab a marshmallow (in the case of DG) or to become angry and lash out (in the case of emotion regulation), is served by a regulatory "control center" that varies among individuals in its efficiency or strength. Performance on tasks diagnostic of self-regulatory ability in one domain (e.g., DG) are therefore predictive of self-regulatory ability in another domain (e.g., emotion regulation), in that both utilize and reflect the power of this more general regulatory ability.

This control center may emerge at a neural level, with common brain regions or networks serving self-regulation of all

types, including, but not limited to, DG and emotion regulation. From this perspective, individual differences in self-regulation reflect the relative or effective recruitment of these regions. Neuroimaging work supporting this thesis has found that dorsolateral (dlPFC) and ventrolateral (vlPFC) regions of the lateral prefrontal cortex (lPFC) are activated during both DG and emotion regulation tasks (e.g., Cohen & Lieberman, 2010; Kober et al., 2010; Ochsner, Bunge, Gross, & Gabrieli, 2002). This lPFC activation is associated with the down-regulation of activity in subcortical regions, such as those implicated in emotional reactivity in the case of emotion regulation (e.g., amygdala; Kober et al., 2010), and reward-related reactivity in the case of DG (e.g., ventral striatum; Ochsner, Bunge, Gross, & Gabrieli, 2002).

For example, in one study participants presented with aversive pictures were directed to regulate their negative emotion by either increasing or decreasing it (Ochsner et al., 2004). When engaging in both forms of emotion regulation, the dlPFC and vlPFC became active. Moreover, up-regulation of negative emotion was associated with increases in amygdala activation, while down-regulation of negative emotion was associated with decreases in amygdala activation. In another study, participants viewed negative pictures and were instructed to cognitively reappraise (i.e., emotion regulation), or just attend to the pictures (Ochsner et al., 2002). During reappraisal, the lPFC became more active and the amygdala became less active. In fact, there was an inverse correlation between vlPFC and amygdala activation. These results suggest that, during reappraisal, vlPFC may down-regulate activity in the amygdala, a region, again, known for its role in emotion processing. For further review on the neural bases of emotion regulation, see Ochsner and Gross (this volume).

DG studies have similarly found the lPFC is important for self-regulation. For example, in choice delay tasks, both dlPFC (Christakou, Brammer, & Rubia, 2011; McClure et al., 2004) and vlPFC (McClure et al., 2004) activation is associated with delayed, rather than immediate, choices. Additionally, participants who showed low levels of dlPFC activation more quickly discounted delayed rewards (i.e., worse DG performance; Hariri et al., 2006). In a more naturalistic study, cigarette smokers viewed pictures of cigarettes known to induce cravings (Kober et al., 2010). During the NOW condition, participants were directed to focus on the immediate consequences of consuming the substance (how great that cigarette would taste; i.e., immediate gratification), while during the LATER condition, participants were directed to focus on the long-term consequences (risk for heart disease; i.e., delayed gratification). There was greater activation of the dlPFC and vlPFC during LATER trials than during NOW trials. Moreover, dlPFC activation was associated with decreased activation in the ventral striatum. In fact, ventral striatal activation mediated the relationship between dlPFC activation and self-reported craving. This suggests that the dlPFC may down-regulate reward-related activity in the ventral striatum, which then may decrease the subjective craving experience.

Together these studies show that the lPFC, including both dorsolateral and ventrolateral subregions, serve an important role in both DG and emotion regulation tasks. These results suggest that the lPFC may be a critical component of a general self-regulation control center, or network, in the brain. A recent review conducted by Cohen and Lieberman (2010) supports this theory. They found that vlPFC activity, in particular, was associated with self-regulation in a variety of domains, including emotion regulation and DG, as well as motor response inhibition, suppressing risky behavior, memory inhibition, and thought suppression.

Imaging studies also have the potential to clarify the relative contributions of both emotional reactivity and regulation to behavioral indices of DG. For example, Casey and colleagues (2011) found that participants with good DG ability showed greater right lPFC activation when inhibiting responses to appetitive stimuli (happy faces). During these same trials, those with good DG also showed less ventral striatum activation. Importantly, there was no relationship between the lPFC and the ventral striatum, suggesting that activation in each region independently related to DG. These results imply that DG ability may as much reflect diminished emotional reactivity to rewards, here social rewards in the form of smiling faces, as regulatory control. Future

work is needed, however, to more fully disentangle how and when these regions work independently versus inversely in predicting DG performance.

Before concluding this section, it is necessary that we mention a few caveats. First, it is important to note that the lPFC, a relatively large region of the brain, has been related to diverse functions above and beyond self-regulation. Additionally, though we have focused on the lPFC in this review, we by no means suggest that this is the only region involved in self-regulation. For example, activation in the medial prefrontal (e.g., Kober et al., 2010; Ochsner et al., 2002, 2004), anterior cingulate (e.g., Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003; Ochsner et al., 2004) posterior cingulate (e.g., Wittman, Leland, & Paulus, 2007), and parietal (e.g., McClure et al., 2004) cortices is also related to self-regulation in many studies, including some of those mentioned earlier. These regions are activated somewhat less consistently, however, and may thus reflect more specific aspects of regulation, forms of regulation, or even the particulars of the tasks used.

Concluding Remarks

In this chapter, we have reviewed research showing that emotion and emotion regulation are strongly connected to DG. Our analysis focused on four subtopics: (1) the link between early caregiver emotion/responsivity and DG; (2) the role of affective state in DG; (3) the association between emotion regulation and DG abilities; and (4) the common neural foundations of emotion regulation and DG. We found that children best able to DG typically have been raised by caregivers who have high positive affect and low negative affect (especially hostility), and are responsive and attentive to their children's emotional needs. A negative affective state has been found to hinder successful DG attempts, both in laboratory contexts and in a real-world DG scenario—dietary restraint. DG ability appears to support individuals' attempts to navigate emotionally evocative situations. Finally, the literature indicates that both DG and emotion regulation activate or rely on common brain regions, including the lPFC. These findings suggest that performance on both emotion regulation and DG tasks may reflect an individual's domain-general regulatory ability, with regulatory control largely influenced by the lPFC.

Though there has been important research on DG and emotion, many questions remain unanswered. Throughout our review we have highlighted a variety of important directions for future research. For example, given that prior results have been mixed, more studies must be conducted to clarify the role of positive emotion in DG performance. Relatedly, how emotion regulation functions within DG tasks needs to be better understood. In one study, reconstruing temptations as a test of willpower was found to improve self-regulation (Magen & Gross, 2007; see Leroy, Grégoire, Magen, Gross, & Mikolajczak, 2012, for similar results). Therefore, reappraising one's frustration during DG tasks may similarly help performance. However, it is also possible that letting task-induced emotions flow freely to reserve regulatory resources would actually enhance DG performance. Therefore, whether emotion regulation attempts help or hurt DG is an open question that awaits further research.

In addition to emotional reactivity and regulation, other factors, such as environmental conditions, may also relate to DG. For example, Kidd, Palmeri, and Aslin (2013) evaluated performance on a classic DG task in children previously exposed to a reliable experimenter (one who promised new art supplies and delivered them), and those exposed to an unreliable experimenter (one who failed to deliver said art supplies). Children previously exposed to the unreliable environment were less likely to wait for the larger rewards—here, a practical and rational decision. These findings suggest that DG ability may as much reflect the reliability of the environments in which children live as their self-regulatory abilities.

Most of the neuroimaging studies evaluating DG reported here have utilized choice delay tasks in adult populations. Future studies should focus analysis on child populations given that prefrontal regions are among the last to mature (e.g., Casey, Galvan, & Hare, 2005) and may not be reliably utilized by children in their efforts to exert self-regulation. Finally, much of the research outlined here is correlational. More experi-

mental research is needed to answer questions about the causal connection between emotion, emotion regulation, and DG.

References

Ayduk, O., Downey, G., & Kim, M. (2001). Rejection sensitivity and depressive symptoms in women. *Personality and Social Psychology Bulletin, 27*(7), 868–877.

Ayduk, O., Downey, G., Testa, A., Yen, Y., & Shoda, Y. (1999). Does rejection elicit hostility in rejection sensitive women? *Social Cognition, 17*(2), 245–271.

Ayduk, O., Mendoza-Denton, R., Mischel, W., Downey, G., Peake, P. K., & Rodriguez, M. (2000). Regulating the interpersonal self: Strategic self-regulation for coping with rejection sensitivity. *Journal of Personality and Social Psychology, 79*(5), 776–792.

Ayduk, O., Zayas, V., Downey, G., Cole, A. B., Shoda, Y., & Mischel, W. (2008). Rejection sensitivity and executive control: Joint predictors of borderline personality features. *Journal of Research in Personality, 42*(1), 151–168.

Baucom, D. H., & Aiken, P. A. (1981). Effect of depressed mood on eating among obese and nonobese dieting and nondieting persons. *Journal of Personality and Social Psychology, 41*(3), 577–585.

Baumeister, R. F., Vohs, K. D., & Tice, D. M. (2007). The strength model of self-control. *Current Directions in Psychological Science, 16*(6), 351–355.

Brooks, D. (2006, May 7). Marshmallows and public policy. Retrieved from *www.nytimes.com/2006/05/07/opinion/07brooks.html?n=top%2fopinion%2feditorials%20and%20op%2ded%2fop%2ded%2fcolumnists%2fdavid%20brooks.*

Brophy-Herb, H. E., Stansbury, K., Bocknek, E., & Horodynski, M. A. (2011). Modeling maternal emotion-related socialization behaviors in a low-income sample: Relations with toddlers' self-regulation. *Early Childhood Research Quarterly, 27*, 352–364.

Carlson, S. M., & Wang, T. S. (2007). Inhibitory control and emotion regulation in preschool children. *Cognitive Development, 22*(4), 489–510.

Casey, B. J., Galvan, A., & Hare, T. A. (2005). Changes in cerebral functional organization during cognitive development. *Current Opinion in Neurobiology, 15*(2), 239–244.

Casey, B. J., Somerville, L. H., Gotlib, I. H., Ayduk, O., Franklin, N. T., Askren, M. K., et al. (2011). Behavioral and neural correlates of delay of gratification 40 years later. *Proceedings of the National Academy of Sciences USA, 108*(36), 14998–15003.

Cherek, D. R., & Lane, S. D. (1999). Laboratory and psychometric measurements of impulsivity among violent and nonviolent female parolees. *Biological Psychiatry, 46*(2), 273–280.

Cherek, D. R., Moeller, F. G., Dougherty, D. M., & Rhoades, H. (1997). Studies of violent and nonviolent male parolees: II. Laboratory and psychometric measurements of impulsivity. *Biological Psychiatry, 41*(5), 523–529.

Christakou, A., Brammer, M., & Rubia, K. (2011). Maturation of limbic corticostriatal activation and connectivity associated with developmental changes in temporal discounting. *NeuroImage, 54*(2), 1344–1354.

Cohen, J. R., & Lieberman, M. D. (2010). The common neural basis of exerting self-control in multiple domains. In R. R. Hassin, K. N. Ochsner, Y. Trope, R. R. Hassin, K. N. Ochsner, & Y. Trope (Eds.), *Self control in society, mind, and brain* (pp. 141–160). New York: Oxford University Press.

Cools, J., Schotte, D. E., & McNally, R. J. (1992). Emotional arousal and overeating in restrained eaters. *Journal of Abnormal Psychology, 101*(2), 348–351.

Derryberry, D., & Rothbart, M. K. (1988). Arousal, affect, and attention as components of temperament. *Journal of Personality and Social Psychology, 55*(6), 958–966.

Downey, G., & Feldman, S. I. (1996). Implications of rejection sensitivity for intimate relationships. *Journal of Personality and Social Psychology, 70*(6), 1327–1343.

Eiden, R. D., Edwards, E. P., & Leonard, K. E. (2007). A conceptual model for the development of externalizing behavior problems among kindergarten children of alcoholic families: Role of parenting and children's self-regulation. *Developmental Psychology, 43*(5), 1187–1201.

Eigsti, I., Zayas, V., Mischel, W., Shoda, Y., Ayduk, O., Dadlani, M. B., et al. (2006). Predicting cognitive control from preschool to late adolescence and young adulthood. *Psychological Science, 17*(6), 478–484.

Eisenberg, N., Fabes, R. A., & Murphy, B. C. (1996). Parents' reactions to children's negative emotions: Relations to children's social

competence and comforting behavior. *Child Development, 67*(5), 2227–2247.

Eisenberg, N., Fabes, R. A., Shepard, S. A., Guthrie, I. K., Murphy, B. C., & Reiser, M. (1999). Parental reactions to children's negative emotions: Longitudinal relations to quality of children's social functioning. *Child Development, 70*(2), 513–534.

Eisenberg, N., Hofer, C., Sulik, J. & Spinrad, T. L. (this volume, 2014). Self-regulation, effortful control, and their socioemotional correlates. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 157–172). New York: Guilford Press.

Eisenberg, N., Valiente, C., Morris, A. S., Fabes, R. A., Cumberland, A., Reiser, M., et al. (2003). Longitudinal relations among parental emotional expressivity, children's regulation, and quality of socioemotional functioning. *Developmental Psychology, 39*(1), 3–19.

Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist, 56*(3), 218–226.

Fredrickson, B. L., & Levenson, R. W. (1998). Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. *Cognition and Emotion, 12*(2), 191–220.

Frost, R. O., Goolkasian, G. A., Ely, R. J., & Blanchard, F. A. (1982). Depression, restraint, and eating behavior. *Behaviour Research and Therapy, 20*(2), 113–121.

Funder, D. C., & Block, J. (1989). The role of ego-control, ego-resiliency, and IQ in delay of gratification in adolescence. *Journal of Personality and Social Psychology, 57*(6), 1041–1050.

Gable, P. A., & Harmon-Jones, E. (2011). Attentional consequences of pregoal and postgoal positive affects. *Emotion, 11*(6), 1358–1367.

Garner, P. W. (1995). Toddlers' emotion regulation behaviors: The roles of social context and family expressiveness. *Journal of Genetic Psychology, 156*(4), 417–430.

Garner, P. W. (2006). Prediction of prosocial and emotional competence from maternal behavior in African American preschoolers. *Cultural Diversity and Ethnic Minority Psychology, 12*(2), 179–198.

Gray, J. R. (1999). A bias toward short-term thinking in threat-related negative emotional states. *Personality and Social Psychology Bulletin, 25*(1), 65–75.

Green, L., Fry, A. F., & Myerson, J. (1994). Discounting of delayed rewards: A life-span comparison. *Psychological Science, 5*(1), 33–36.

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York: Guilford Press.

Hariri, A. R., Brown, S. M., Williamson, D. E., Flory, J. D., de Wit, H., & Manuck, S. B. (2006). Preference for immediate over delayed rewards is associated with magnitude of ventral striatal activity. *Journal of Neuroscience, 26*(51), 13213–13217.

Hariri, A. R., Mattay, V. S., Tessitore, A., Fera, F., & Weinberger, D. R. (2003). Neocortical modulation of the amygdala response to fearful stimuli. *Biological Psychiatry, 53*, 494–501.

Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. *Psychological Bulletin, 110*(1), 86–108.

Heatherton, T. F., & Wagner, D. D. (2011). Cognitive neuroscience of self-regulation failure. *Trends in Cognitive Sciences, 15*(3), 132–139.

Herman, C. P., & Polivy, J. (1975). Anxiety, restraint, and eating behavior. *Journal of Abnormal Psychology, 84*(6), 666–672.

Houck, G. M., & Lecuyer-Maus, E. A. (2004). Maternal limit setting during toddlerhood, delay of gratification, and behavior problems at age five. *Infant Mental Health Journal, 25*(1), 28–46.

Jacobsen, T. (1998). Delay behavior at age six: Links to maternal expressed emotion. *Journal of Genetic Psychology, 159*(1), 117–120.

Jacobsen, T., Huss, M., Fendrich, M., Kruesi, M. J. P., & Ziegenhain, U. (1997). Children's ability to delay gratification: Longitudinal relations to mother–child attachment. *Journal of Genetic Psychology, 158*(4), 411–426.

Jahromi, L. B., & Stifter, C. A. (2007). Individual differences in the contribution of maternal soothing to infant distress reduction. *Infancy, 11*(3), 255–269.

Kidd, C., Palmeri, H., & Aslin, R. N. (2013). Rational snacking: Young children's decision-making on the marshmallow task is moderated by beliefs about environmental reliability. *Cognition, 126*, 109–114.

Kober, H., Mende-Siedlecki, P., Kross, E. F., Weber, J., Mischel, W., Hart, C. L., et al. (2010). Prefrontal–striatal pathway underlies cognitive regulation of craving. *Proceedings of the National Academy of Sciences USA, 107*(33), 14811–14816.

Kochanska, G., & Knaack, A. (2003). Effort-

ful control as a personality characteristic of young children: Antecedents, correlates, and consequences. *Journal of Personality, 71*(6), 1087–1112.

Kochanska, G., Murray, K. T., & Harlan, E. T. (2000). Effortful control in early childhood: Continuity and change, antecedents, and implications for social development. *Developmental Psychology, 36*(2), 220–232.

Krueger, R. F., Caspi, A., Moffitt, T. E., White, J., & Stouthamer-Loeber, M. (1996). Delay of gratification, psychopathology, and personality: Is low self-control specific to externalizing problems? *Journal of Personality, 64*(1), 107–129.

Lehrer, J. (2009, May 18). Don't!: The secret of self-control. *The New Yorker.* Retrieved from *www.newyorker.com/reporting/2009/05/18/090518fa_fact_lehrer.*

Leroy, V., Grégoire, J., Magen, E., Gross, J. J., & Mikolajczak, M. (2012). Resisting the sirens of temptation while studying: Using reappraisal to increase focus, enthusiasm, and performance. *Learning and Individual Differences, 22*(2), 263–268.

Magen, E., & Gross, J. J. (2007). Harnessing the need for immediate gratification: Cognitive reconstrual modulates the reward value of temptations. *Emotion, 7*(2), 415–428.

Malatesta, C. Z., Culver, C., Tesman, J. R., & Shepard, B. (1989). The development of emotion expression during the first two years of life. *Monographs of the Society for Research in Child Development, 54*(1–2), 1–104.

Mathias, C. W., Dougherty, D. M., James, L. M., Richard, D. M., Dawes, M. A., Acheson, A., et al. (2011). Intolerance to delayed reward in girls with multiple suicide attempts. *Suicide and Life-Threatening Behavior, 41*(3), 277–286.

McClure, S. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. (2004). Separate neural systems value immediate and delayed monetary rewards. *Science, 306*(5695), 503–507.

Metcalfe, J., & Mischel, W. (1999). A hot/cool-system analysis of delay of gratification: Dynamics of willpower. *Psychological Review, 106*(1), 3–19.

Miller, E. M., Rodriguez, C., Kim, B., & McClure, S. M. (this volume, 2014). Delay discounting: A two-systems perspective. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 93–110). New York: Guilford Press.

Mischel, W., & Ayduk, O. (2011). Willpower in a cognitive-affective processing system: The dynamics of delay of gratification. In K. D. Vohs & R. F. Baumeister (Eds.), *Handbook of self-regulation: Research, theory, and applications* (2nd ed., pp. 83–105). New York: Guilford Press.

Mischel, W., Ebbesen, E., & Zeiss, A. (1972). Cognitive and attentional mechanisms in delay of gratification. *Journal of Personality and Social Psychology, 21*(2), 204–218.

Mischel, W., & Gilligan, C. (1964). Delay of gratification, motivation for the prohibited gratification, and responses to temptation. *Journal of Abnormal and Social Psychology, 69*(4), 411–417.

Mischel, W., Shoda, Y., & Rodriguez, M. L. (1989). Delay of gratification in children. *Science, 244*(4907), 933–938.

Moore, B. S., Clyburn, A., & Underwood, B. (1976). The role of affect in delay of gratification. *Child Development, 47*(1), 273–276.

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14*(8), 1215–1229.

Ochsner, K. N., & Gross, J. J. (this volume, 2014). The neural bases of emotion and emotion regulation: A valuation perspective. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 23–42). New York: Guilford Press.

Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D., et al. (2004). For better or for worse: Neural systems supporting the cognitive down-and up-regulation of negative emotion. *NeuroImage, 23*, 483–499.

Olson, S. L., Bates, J. E., & Bayles, K. (1990). Early antecedents of childhood impulsivity: The role of parent–child interaction, cognitive competence, and temperament. *Journal of Abnormal Child Psychology, 18*(3), 317–334.

Polivy, J., & Herman, C. P. (1976). Clinical depression and weight change: A complex relation. *Journal of Abnormal Psychology, 85*(3), 338–340.

Pratt, F. J., & Wardle, J. (2012). Dietary restraint and self-regulation in eating behavior. *International Journal of Obesity, 36*, 665–674.

Rodriguez, M., Ayduk, O., Aber, J. L., Mischel, W., Sethi, A., & Shoda, Y. (2005). A contextual approach to the development of self-regulatory competencies: The role of maternal unresponsivity and toddlers' negative affect

in stressful situations. *Social Development, 14*(1), 136–157.

Rodriguez, M. L., Mischel, W., & Shoda, Y. (1989). Cognitive person variables in the delay of gratification of older children at risk. *Journal of Personality and Social Psychology, 57*(2), 358–367.

Rothbart, M. K., Sheese, B. E., & Posner, M. I. (2007). Executive attention and effortful control: Linking temperament, brain networks, and genes. *Child Development Perspectives, 1*(1), 2–7.

Santucci, A. K., Silk, J. S., Shaw, D. S., Gentzler, A., Fox, N. A., & Kovacs, M. (2008). Vagal tone and temperament as predictors of emotion regulation strategies in young children. *Developmental Psychobiology, 50*(3), 205–216.

Schwarz, J. C., & Pollack, P. R. (1977). Affect and delay of gratification. *Journal of Research in Personality, 11*(2), 147–164.

Schwarz, N. (2011). Feelings-as-information theory. In P. A. M. Van Lange, A. W. Kruglanski, & E. T. Higgins (Eds.), *Handbook of theories of social psychology* (pp. 289–308). Thousand Oaks, CA: Sage.

Seeman, G., & Schwarz, J. C. (1974). Affective state and preference for immediate versus delayed reward. *Journal of Research in Personality, 7*(4), 384–394.

Sethi, A., Mischel, W., Aber, J. L., Shoda, Y., & Rodriguez, M. L. (2000). The role of strategic attention deployment in development of self-regulation: Predicting preschoolers' delay of gratification from mother–toddler interactions. *Developmental Psychology, 36*(6), 767–777.

Shaver, P. R., & Mikulincer, M. (this volume, 2014). Adult attachment and emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 237–250). New York: Guilford Press.

Shaver, P. R., & Mikulincer, M. (2011). Attachment theory. In P. A. M. Van Lange, A. W. Kruglanski, & E. T. Higgins (Eds.), *Handbook of theories of social psychology* (pp. 160–179). Thousand Oaks, CA: Sage.

Shmueli, D., & Prochaska, J. J. (2012). A test of positive affect induction for countering self-control depletion in cigarette smokers. *Psychology of Addictive Behaviors, 26*(1), 157–161.

Shoda, Y., Mischel, W., & Peake, P. K. (1990). Predicting adolescent cognitive and self-regulatory competencies from preschool delay of gratification: Identifying diagnostic conditions. *Developmental Psychology 26*(6), 978–986.

Silverman, I. W., & Ippolito, M. F. (1995). Maternal antecedents of delay ability in young children. *Journal of Applied Developmental Psychology, 16*(4), 569–591.

Thompson, R. A. (this volume, 2014). Socialization of emotion and emotion regulation in the family. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 173–186). New York: Guilford Press.

Tice, D. M., Baumeister, R. F., Shmueli, D., & Muraven, M. (2007). Restoring the self: Positive affect helps improve self-regulation following ego depletion. *Journal of Experimental Social Psychology, 43*(3), 379–384.

Tice, D. M., Baumeister, R. F., & Zhang, L. (2004). The role of emotion in self-regulation: Differing role of positive and negative emotions. In P. Philippot & R. S. Feldman (Eds.), *The regulation of emotion* (pp. 213–226). Mahwah, NJ: Erlbaum.

Wertheim, E. H., & Schwarz, J. C. (1983). Depression, guilt, and self-management of pleasant and unpleasant events. *Journal of Personality and Social Psychology, 45*(4), 884–889.

Wittman, M., Leland, D. S., & Paulus, M. P. (2007). Time and decision making: Differential contribution of the posterior insular cortex and the striatum during a delay discounting task. *Experimental Brain Research, 179*(4), 643–653.

CHAPTER 8

Emotion Regulation Choice: Theory and Findings

Gal Sheppes

A few months ago, I had to take a long, 12-hour international flight in order to get home. Having more than enough time to spend, I started looking around me. While some people were sleeping (in extremely creative body postures), or watching movies (with delight that they finally had enough time to watch Tolkien's full trilogy), others appeared quite alert and fearful, especially when the captain announced that the plane had started its final descent. While appearing fearful, luckily these individuals were able to choose between different regulatory options to control their fear. For example, they could choose whether they want to deal with their flight phobia by disengagement, closing their window shades while immersing themselves in unrelated yet demanding conversations. Alternatively, they could choose to look out the window while explaining some facts, including a decline in crash statistics since autopilots took charge.

Emotion regulatory choices are an integral part of our daily lives in a dynamically changing affective world. By *emotion regulation choice* I mean the choices individuals make as to how they should regulate their emotions in a particular context when regulation is warranted and more than one regulatory option is active. Furthermore, given the abundance of knowledge on explicit emotion regulation strategies and the relative lack of evidence on implicit emotion regulation, I limit this chapter to deliberate and explicit forms of emotion regulation choice.

Can we consistently predict the regulatory choices individuals make to deal with their emotions? Are there specific emotional, cognitive, and motivational influences that systematically affect these regulatory preferences? What are the major factors underlying the regulatory choice process? And what are the broad implications of regulatory choices? In this chapter I try to answer these important questions. Specifically, I begin by introducing the importance of emotion regulation choice and its relative lack of empirical investigation. I then introduce a recent conceptual model to explain dominant emotional, cognitive, and motivational determinants, and underlying mechanisms of emotion regulation choice between dominant cognitive emotion regulation categories. I close by describing broad implications and future directions.

The Importance of Emotion Regulation Choice

The different ways individuals can go about controlling their emotions has attracted scholars for centuries. Nevertheless, emotion regulation has become an independent

field only recently (Gross, 1998, 2007, 2010; Koole, 2009; Tamir, 2011). From its early days a central question in this field has been whether different forms of emotion regulation have different consequences. However, in a first generation of studies, different forms of emotion regulation have generally been considered to be either adaptive or maladaptive.

Of two well-known examples, consider first Nolen-Hoeksema's influential response styles theory contrasting maladaptive rumination with distraction (Nolen-Hoeksema, 1991; see Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008, for reviews). Multiple studies have widely established that ruminating on negative attributes of one's self rather than distracting attention away from them is strongly related to the development, maintenance, and recurrence of depressive episodes. As a second example, consider Gross's canonical process model and supporting evidence from multiple studies showing the relative superiority of reappraising the meaning of negative events over suppression of affective expressions, with respect to a wide range of affective, cognitive, and social indicators of adaptive functioning (Gross, 2002; see Gross & Thompson, 2007, for reviews). The centrality of the alleged dichotomy between "adaptive" and "maladaptive" forms of emotion regulation is captured by a recent meta-analysis that summarizes a decade of work on the relationship between some regulation strategies (rumination, suppression) and psychopathology, and other strategies (reappraisal, problem solving) and resilience (Aldao, Nolen-Hoeksema, & Schweizer, 2010).

There is no doubt that the first generation of studies has enormously advanced the field of emotion regulation. However, with its rapid growth a second generation of studies to emerge has begun to find inconsistencies in the formerly unconditional maladaptive–adaptive label given to different strategies. For example, the ostensibly maladaptive strategy of rumination was found to have both adaptive and maladaptive forms (Watkins, 2008). For example, rumination was found to be advantageous in situations in which a single goal needs to be maintained in the face of distractors (Altamirano, Miyake, & Whitmer, 2010). Similarly, the ostensibly maladaptive strategy of suppression was shown to be beneficial in extremely adverse situations (e.g., Bonanno & Keltner, 1997). At the same time, the ostensibly adaptive strategy of distraction was found to be maladaptive when long-term adjustment is required (Kross & Ayduk, 2008), and the ostensibly adaptive strategy of reappraisal was found to be less effective when dealing with particularly high-intensity emotional situations (Sheppes, Catran, & Meiran, 2009; Sheppes & Meiran, 2007, 2008).

The main conclusion derived from the second generation of studies is that emotion regulation strategies have different consequences in different contexts. This means that healthy adaptation is the result of flexibly choosing between regulation strategies to adapt to differing situational demands (e.g., Bonanno, 2005; Kashdan & Rottenberg, 2010; see Troy & Mauss, 2011, for reviews). To illustrate, Kashdan and Rottenberg (2010) argue that different forms of psychopathology can be characterized by different ways in which flexible regulation choice breaks down, and Troy and Mauss's (2011) and Bonanno's (2005) influential accounts highlight how flexible regulation choice promotes resilience in the face of stress and trauma.

Although emotion regulation choice is now viewed as a crucial element in healthy adaptation, until recently it has only been studied indirectly. Two forms of important yet indirect evidence come from correlational studies involving self-report questionnaires that assess individual differences in people's frequency of use of different regulatory strategies across situations (e.g., Aldao & Nolen-Hoeksema, 2012a; Garnefski et al., 2001; Gross & John, 2003; John & Gross, 2007) and from laboratory experiments that evaluate spontaneous use of emotion regulation strategies in emotion-inducing situations (e.g., Aldao & Nolen-Hoeksema, 2012b; Ehring, Tuschen-Caffier, Schnülle, Fischer, & Gross, 2010; Gruber, Harvey, & Gross, 2012). While showing important links to well-being and various forms of psychopathology (see Aldao et al., 2010, for a review), these studies do not assess the factors that influence individuals predominantly to prefer using one particular regulatory strategy over another. Even more conventional studies of emotion regulation have not examined which regulation strategies are chosen

in different emotional contexts, because the experimental design involved directly instruct participants to use regulation strategies in different situations. Consider, for example, the most direct and convincing evidence of the importance of flexible regulation patterns, which has shown that the ability to alternate flexibly between enhancing and suppressing emotion strongly predicts healthy adaptation (Bonanno, Papa, Lalande, Westphal, & Coifman, 2004) over an extended time period (Westphal, Seivert, & Bonanno, 2010), and that flexible regulation can protect people from complicated grief patterns in bereavement (Gupta & Bonanno, 2011). In these and other studies, the regulation strategies employed by participants were determined by the experimenter, leaving the important topic of determinants and consequences of emotion regulation choice unexplored.

Conceptualizing Emotion Regulation Choice

Recently my colleagues and I developed a conceptual framework to explain (1) major determinants and (2) underlying mechanisms of emotion regulation choice (Sheppes, Scheibe, Suri, Radu, Blechert, & Gross, 2012). A central working hypothesis in this framework is that healthy individuals would be sensitive to the central costs and benefits associated with the implementation of each regulatory option under different contexts. With this working hypothesis in mind, certain emotional, cognitive, and motivational contextual factors are likely to bias regulatory choices in ways that are congruent with the differential consequences of implementing these strategies under these conditions. In illuminating the underlying mechanisms of emotion regulation choice a further argument suggests that healthy regulation choice should require in some contexts an ability to recruit deliberate executive control processes that can override contrasting associative emotional processes. This interplay of higher control processes that can override opposing associative processes is at the heart of many models of self-regulation (e.g., Muraven & Baumeister, 2000). Moreover, the regulation choice process gives a central weight to differences between strategies' underlying engagement–disengagement dimension relative to other potent factors, such as cognitive effort. To help us better appreciate the broader context of this emotion regulation choice conceptual framework I elaborate it below.

The starting point of this conceptual framework is that individuals' limited cognitive capacity poses permanent processing constraints. These constraints result in a constant competition between emotion generation and emotion regulation processes (Gross, Sheppes, & Urry, 2011a, 2011b) for dominance over behavior. Our conceptual account draws on major information-processing theories (e.g., Hübner, Steinhauser, & Lehle, 2010; Pashler, 1998) and the process model of emotion regulation (Gross & Thompson, 2007) to suggest that regulating one's emotions involves recruiting *deliberate executive control mechanisms* that try to modify the nature of emotional information processing at two major cognitive stages (Sheppes & Gross, 2011, 2012), which include an early attentional selection stage and a late semantic meaning stage. Specifically, regulation can be achieved via early *disengagement* from emotional processing at the attention selection stage, or via an *engagement* with emotional processing that is modulated at a late semantic meaning stage (e.g., Johnston & Heinz, 1978; Lehle & Hübner, 2008). This conceptual framework focuses on two particular regulatory strategies that are frequently used in everyday life and have their major influence in each of these two cognitive stages of information processing.

Incoming emotional information can be regulated at an early attentional selection processing stage by disengaging from emotional information processing before it undergoes elaborated processing. A classic early selection strategy is *distraction*, which involves disengaging attention from emotional processing before it is represented in working memory by producing neutral thoughts that are independent from and not in conflict with emotional information (e.g., van Dillen & Koole, 2007).

Engagement with incoming emotional information that passes the early attentional selection stage can still be regulated at a late semantic meaning processing stage before it affects behavior. A classic late

selection regulation strategy is *reappraisal*, which involves engaging with and elaborating emotional information prior to changing its meaning in a late processing stage (e.g., Gross, 2002). In reappraisal, the neutral reinterpretation is semantically dependent and in direct conflict with the original emotional information. According to our conceptual framework, these underlying characteristics of disengagement distraction and engagement reappraisal result in a differential cost–benefit tradeoff (Sheppes & Gross, 2012). Specifically, the benefits of blocking emotional information early, before it gathers force via distraction, are that *emotionally* high-intensity information can be successfully modulated. *Cognitively*, this successful modulation involves relatively simple processes, because the generation of regulatory neutral thoughts in distraction is independent from and does not conflict with the original emotional information. Nevertheless, the major cost of distraction is that because it does not allow processing, evaluating, and remembering of emotional information, *motivationally* it is less beneficial for one's long-term goals and adaptation (see Wilson & Gilbert, 2008, for a review). Specifically, distraction does not allow one to attend to emotional events that are being repeatedly encountered and be provided with adequate explanation, a requirement that is at the heart of many long-term goals in which an individual has to face difficulties in order to adapt.

The underlying characteristics of engagement reappraisal result in a different set of costs and benefits. Specifically, the elaborated semantic processing that occurs prior to late modulation should be *emotionally* costly, because it can less successfully block high-intensity emotional information. *Cognitively*, reappraisal engages relatively complex processes, because the generation of alternative construals is dependent on and in conflict with the original emotional information. Nevertheless, the major benefit of engaging with emotional information is that *motivationally* it allows one to process, evaluate, and remember emotional information, which are crucial for long-term goals and for adaptation.

Direct empirical support for the costs and benefits of employing (rather than freely choosing) distraction and reappraisal comes from behavioral and electrophysiological studies. Specifically, several behavioral studies showed that employing early disengagement distraction in high-sadness *emotionally* intense situations resulted in successful regulation (Sheppes & Meiran, 2007), and did not result in an increased expenditure of *cognitive* resources (Sheppes et al., 2009; Sheppes & Meiran, 2008). At the same time, distraction's lack of emotional processing was demonstrated in an impaired memory for emotional information (Sheppes & Meiran, 2007, 2008), and distraction's *motivational* costs were evident in a lack of long-term attenuation of the intensity or quality of important autobiographical, negative emotional events (Kross & Ayduk, 2008). By contrast, these studies showed that while employing late engagement reappraisal in low-sadness *emotionally* intense situations was successful, high-sadness emotionally intensity situations resulted in less successful modulation and in an increased expenditure of *cognitive* resources. The elaborated emotional processing was demonstrated in intact memory for emotional information (see also Dillon, Ritchey, Johnson, & LaBar, 2007; Richards & Gross, 1999, 2000), and reappraisal's *motivational* benefits evinced in adaptation to distressing events that are important for one's long-term goals and functioning.

In two recent electrophysiological studies, my colleagues and I took advantage of the excellent temporal resolution of electroencephalography (EEG) and event-related potentials (ERPs) to provide further support for the differential underlying cognitive mechanisms and consequences of employing distraction and reappraisal (Blechert, Sheppes, Di Tella, Williams, & Gross, 2012; Thiruchselvam, Blechert, Sheppes, Rydstrom, & Gross, 2011). Recent ERP studies in emotion regulation showed that distraction (e.g., Dunning & Hajcak, 2009; Hajcak, Dunning, & Foti, 2009) and reappraisal (e.g., Foti & Hajcak, 2008; Hajcak & Nieuwenhuis, 2006) modulate the late positive potential (LPP)—an electrocortical component that is enhanced during emotionally arousing viewing and that reflects enhanced attentional and semantic meaning processing of emotionally salient information (Hajcak, MacNamara, & Olvet, 2010).

Consistent with the framework, we found

that implementing distraction resulted in a strong modulation of an initial phase of the LPP that represents early disengagement of emotional information before it is represented in working memory, and reappraisal only modulated the late phase of the LPP representing engagement and elaborated meaning prior to late modulation (Thiruchselvam et al., 2011). In that same study, we also tested our prediction that, motivationally, distraction relative to reappraisal cannot be in accord with major long-term goals. Specifically, distraction does not allow attending to and explaining emotional information, which result in subsequent attenuation of emotional responses relative to novel emotional situations (Wilson & Gilbert, 2008). To that end, our participants were reexposed to emotional materials they had previously distracted or reappraised. Consistent with our prediction, we found that emotional materials with a distraction but not reappraisal history demonstrated an enhanced LPP during reexposure, which represents an extended influence of negative emotional processing beyond the regulatory episode. Prolonged influence of emotional events is considered maladaptive, with many long-term goals that require dealing with emotional events that are repeatedly encountered (see also MacNamara, Ochsner, & Hajcak, 2011). In a similar vein, we recently showed that repeated reappraisal efforts with biologically significant emotional stimuli (i.e., angry facial expressions) resulted in a gradual change to the basic evaluation and thus representation of these emotional stimuli (Blechert et al., 2012).

Emotional, Cognitive, and Motivational Determinants of Emotion Regulation Choice

In a recent set of studies my colleagues and I have evaluated central determinants of emotion regulation choice (Sheppes, Scheibe, Suri, & Gross, 2011; Sheppes et al., 2012). Prior to discussing these studies I wish briefly to describe the novel paradigm we developed to enable the evaluation of emotion regulation choice. In short, participants that come to the laboratory initially undergo a learning phase, followed by a training phase in which they are taught the differences between distraction and reappraisal, as well as how to implement each. In the actual task, participants are exposed to series of trials that involve a brief presentation of an emotional stimulus followed by a choice screen, where they decide their preferred regulatory strategy. Following a short preparation period, the emotional stimulus reappears and participants are instructed to implement their chosen strategy and to rate how they feel. In general, participants are instructed to choose freely between distraction and reappraisal, and to base their choice on the strategy they think will make them feel less negative. The main dependent measure is the proportion of choice of each regulatory option. To evaluate adherence of participants' reports of their chosen regulatory strategies (achieved via which button they pressed during the choice screen), we either asked participants to talk out loud about their chosen strategies during strategy implementation or we administered a surprise memory test for emotional materials that were presented (see Sheppes et al., 2011). These two methods have proven to be satisfactory. Specifically, adherence levels were evident in high agreement between participants' button responses and their talk-out-loud protocols, and in the finding that in trials on which participants indicated that they chose to distract, memory was worse (and not significantly different from chance) than that in trials of participants who indicated that they chose to reappraise (Sheppes et al., 2011).

The first determinant of regulation choice examined is *emotional* intensity, which is a key dimension of variation across emotional contexts (Sheppes et al., 2011). Based on the emotion regulation choice conceptual framework, we predicted that under low-negative emotionally intense situations, individuals would prefer to choose late selection engagement reappraisal over early selection disengagement distraction, because reappraisal can both successfully modulate emotional responding and provide long-term affective adaptation. However, we predicted that under high-negative intensity situations, participants would switch, to prefer choosing early disengagement distraction over reappraisal, because only distraction can successfully block emotional information early before it gathers force.

To test our predictions, we manipulated emotional intensity with emotional images or unpredictable electric stimulation and had participants choose between distraction and reappraisal (Sheppes et al., 2011). The results strongly supported our predictions in both emotional contexts. Specifically, participants preferred to reappraise their emotional reactions to low-negative-intensity pictures and to a threat of low-intensity electric shocks, but they preferred to distract from their emotional reactions to high-negative-intensity pictures and to a threat of high-intensity electric shocks.

In a follow-up study we wanted to examine the robustness of the effect of emotional intensity on regulation choice (Sheppes et al., 2012). To that end, we examined whether individuals would keep their regulatory preferences under different emotional intensities even when offered a potent reinforcement (monetary incentive) to engage in a counter preference regulatory option. Although we found that monetary incentives influenced regulatory choices in the expected direction, the basic preference to reappraise low-intensity emotional situations and to distract high-intensity emotional situations remained evident, even when participants were paid high monetary amounts to choose the contrasting strategy. These results suggest that emotional intensity strongly influences regulatory preferences.

The second determinant we examined was the *cognitive* complexity of generating an emotion regulation strategy (Sheppes et al., 2012). Emotion regulation can be viewed as involving several sequential cognitive processes that involve generation, implementation, and maintenance (Kalisch, 2009; Ochsner & Gross, 2008). Generation involves finding an adequate regulatory option that can replace emotional information processing. Implementation involves activating a regulatory strategy, and maintenance involves holding it in an active state as long as regulation is required. Because generation operates early in the sequence of an emotion regulation episode, it is likely to affect emotion regulation choice. In distraction, the generation process is simple, because the neutral thoughts that are produced can be of any content as long as they are absorbing. However, in reappraisal, the generation process is complex, because the formation of an alternative neutral reinterpretation depends on the original emotional information. To examine this disparity we showed that when the generation process was simplified, by providing participants concrete regulatory options for distraction and reappraisal, reappraisal was more frequently chosen.

The third determinant of emotion regulation choice involved investigating the influence of *motivational* goals (Sheppes et al., 2012). An important motivational goal for choosing a regulatory strategy involves evaluating whether an emotional stimulus will be encountered once or multiple times. While an emotional stimulus that is encountered once can be regulated with strategies such as distraction, which provide short term relief, emotional stimuli that are encountered multiple times can be better regulated with strategies such as reappraisal, which involves engaging with emotional processing that results in gradual adaptation (e.g., Wilson & Gilbert, 2008; Blechert et al., 2012). As we therefore expected and found, when participants were told that they would encounter emotional stimuli more than once, they preferred to reappraise more than they did when they expected to encounter an emotional stimulus once.

Underlying Mechanisms of Emotion Regulation Choice

The previous section established that central emotional, cognitive, and motivational factors strongly influence individuals' preferences between two regulation strategies that modulate emotional responding at an early attentional stage (distraction) or a late semantic meaning stage (reappraisal). In this section I turn to the issue of the mechanisms at the core of emotion regulation choice.

According to our conceptual framework, emotion regulation choice should involve a general ability of deliberate executive control processes to override competing associative emotional processes. In line with central models of self control (e.g., Muraven & Baumeister, 2000), an ability to recruit central control processes that can moderate the influence of drives and emotions is crucial for daily functioning. In addition, the actual regulatory choice process is heavily influenced by the engagement–disengagement

dimension where people are weighting their preference to employ early attentional *disengagement* from emotional processing (distraction) versus *engagement* with emotional processing prior to late modulation at the semantic meaning processing stage (reappraisal).

Provision of supporting evidence for the involvement of deliberate executive control processes that override associative emotional processes in emotion regulation choice is important given a potential alternative, more parsimonious account. According to this more parsimonious account, emotion regulation choice can be fully explained by a direct influence from simple associative emotional processes (e.g., Bradley, Codispoti, Cuthbet, & Lang, 2001). Specifically, according to an associative emotional process account, as negative emotional intensity increases, it directly activates a basic defensive system to shift from an engagement (or sensory intake) preference, which leads to preferring reappraisal, to a disengagement (or sensory rejection) preference, which may result in an increased preference to distract. Alternatively, according to the emotion regulation choice account, individuals can use deliberate executive processes in preferring to reappraise in low-negative intensity situations and to distract in high-negative intensity situations. Because both accounts lead to the same regulatory choice prediction in negative emotional situations, in order to determine between them we investigated down-regulation of positive emotional situations—an emotional context where the two accounts diverge. Specifically, the associative emotional process account would argue that as positive emotional intensity increases, it directly activates a basic appetitive system that would lead to an increased preference to engage. By contrast, the emotion regulation choice account would predict that the operation of deliberate control processes, whose goal is to provide down-regulation of positive emotional situations, would involve overriding the associative tendency to engage, resulting in an increased preference to disengage as positive emotional intensity increases. The results, which showed participants' clear tendency to disengage via distraction as positive emotional intensity increased, clearly support the involvement of deliberate executive control processes that override opposing associative emotional processes originating from the appetitive system (Sheppes et al., 2012).

While these results support the involvement of deliberate executive control processes in emotion regulation choice, an important question remains: What are the dimensions that receive central weight in the choice between distraction and reappraisal? One central dimension in my conceptual account is engagement–disengagement, in which people who prefer to reappraise may wish to engage in emotional processing, and when they want to distract may want to disengage from emotional processing. However, in a second potential key dimension, the amount of cognitive effort people wish to exert (e.g., Chajut & Algom, 2003; Kool, McGuire, Rosen, & Botvinick, 2010; Muraven & Baumeister, 2000), when people prefer to reappraise they are willing to exert considerable effort, and when they want to distract they wish to preserve cognitive effort. Accordingly, when people prefer to distract in high-negative emotionally intense situations, do they prefer to disengage from emotional processing or do they prefer to reserve their cognitive resources and go with the easy regulatory option?

To determine between these two options, we had participants choose between two types of distractions that involve making mathematical computations (e.g., see Erber & Tesser, 1992; van Dillen & Koole, 2007 for similar operationalization of attentional distractions): One regulatory option was cognitively simple and involved minor disengagement from emotional processing (subtract 2's) and a second regulatory option was cognitively effortful yet highly disengaged from emotional processing (subtract 7's). The logic was that if the cognitive effort dimension is central in regulatory choice, then one should expect that the preference to use the more simple subtract 2's distraction would increase as negative emotional intensity increases. However, if the engagement–disengagement dimension is central in regulatory choice, we should expect that the preference to use the more disengaging (despite it being also more effortful) subtract 7's distraction would increase as negative emotional intensity increases.

Results strongly supported the centrality of the engagement–disengagement dimension, where participants preferred to disengage from emotional processing in the high-

intensity condition despite the fact that this regulatory option was clearly more effortful (Sheppes et al., 2012). In addition, when participants were performing the subtract 7's option, their actual mathematical performance was interfered with less by the intensity of emotional stimuli relative to performing subtract 2's. This result indicates that the more effortful subtract 7's option indeed resulted in a stronger disengagement from emotional processing relative to subtracting 2's.

In a complementary fashion we wished to show that the engagement–disengagement dimension is central within the reappraisal category. Although, reappraisal is generally considered to be an engagement strategy (Gross & Thompson, 2007; Sheppes & Gross, 2011), there is also a considerable variation in this broad regulation category (Ochsner et al., 2004; McRae, Ciesielski, & Gross, 2012). Specifically, in a classic situation-focused reappraisal, engagement with emotional processing is central in order to reinterpret the meaning of ongoing events. However, other forms of reappraisal, such as reality challenge, involve a disengagement element in which emotional consequences are simply not considered and the basic authenticity of the event is being questioned. In a regulation choice study we showed that whereas participants preferred to engage via situation-focused reappraisal under a low-intensity condition, they preferred to disengage via reality challenge reappraisal under a high-intensity condition (Sheppes et al., 2012).

Broader Implications

In the two previous sections, I tried to establish key emotional, cognitive, and motivational determinants, and underlying mechanisms of emotion regulation choice. In this section I zoom out in order to provide broader implications of emotion regulation choice. In doing so I concentrate on implications for emotion regulation science, decision making, and clinical science.

Implications for Emotion Regulation

Emotion regulation choice provides an important extension to the general field of emotion regulation. Previous studies of emotion regulation have almost exclusively focused on the consequences of implementing different emotion regulation strategies (Gross, 2007; see Koole, 2009, for reviews). Multiple studies have instructed participants to engage with different emotion regulation strategies and have examined the costs and benefits associated with successful implementation. Understanding the consequences of employing different regulation strategies is a crucial and important step toward an understanding of the basic elements of emotion regulation strategies. Nevertheless, the emotion regulation choice findings extend this work by illuminating a step that precedes the implementation of emotion regulation. Specifically, when an emotion regulation goal has been activated, individuals need to select a regulation strategy out of all the available options. Our conceptual account has highlighted some emotional, cognitive, and motivational factors that can strongly influence the regulatory choices individuals are inclined to make in any particular context.

Combining the recent knowledge on emotion regulation choice with existing knowledge on the consequences of implementing emotion regulation strategies leads to important conceptual extensions. For example, individuals' regulatory preferences as indexed by their regulatory choices should be considered when evaluating individuals' ability to implement different strategies. Specifically, initiating a particular strategy may require not only generation, implementation, and maintenance but also overriding a default regulatory preference. To give laboratory and real-life analogues, it may be that when participants in the laboratory or patients in the clinic are asked to engage via reappraisal when dealing with high-intensity situations, in addition to generating, implementing, and maintaining reinterpretation of an emotional situation, they also need to override their strong default preference to disengage via distraction.

Implications for Decision Making

Emotion regulation choice is an instance of general choice behavior, and as such it is important to place it under the broad umbrella of general decision making. Classic examples of choice behavior involve deciding between different external outcomes in

one's environment. For example, in intertemporal choice paradigms, such as temporal discounting (Reynolds, 2006), individuals choose between receiving smaller monetary rewards sooner (e.g., $5 today) and receiving bigger monetary rewards later (e.g., $8 tomorrow). Other examples of choice behavior involve deciding between different internal processes to deal with external demands, such as the mathematical strategies children choose in order to solve math problems (Siegler, 2005) or the strategies adults choose to solve chess problems (de Groot, 1978). Emotion regulation choice is a special case of decision making, because it involves choosing between internal cognitive processes to control one's internal emotional environment.

Although emotion regulation choice appears to be unique in some ways, it shares with other theories basic assumptions about strategy choice. Like classic theories in decision sciences (e.g., Payne, Bettman, & Johnson, 1988, 1993), the emotion regulation choice account argues that individuals are sensitive to central factors (emotional, cognitive, and motivational factors in the case of emotion regulation choice) when making their regulatory selections. In addition, just as other models highlight the role of learning (e.g., Rieskmap, 2006; Rieskmap & Otto, 2006), in the emotion regulation choice account regulatory decisions are to some extent based on prior knowledge, with the consequences of implementing different strategies in different contexts.

Implications for Clinical Psychology

Emotion regulation choice also has important clinical implications. Central conceptual accounts argue that psychological well-being requires flexibly adapting emotion regulation strategies to fit with differing situational demands (Gross, 2007; Kashdan & Rottenberg, 2010; Watkins, 2011). The flip side of flexible regulatory choice is a rigid and maladaptive regulation choice that may be related to various forms of psychopathology.

The recent empirical evidence maps emotional, cognitive, and motivational determinants of regulation choice in healthy adults; thus, deviations from healthy regulatory choice can be used to understand different forms of psychopathology.

Consider first the emotional intensity one is facing. As previously described, healthy individuals prefer to use reappraisal in low-intensity emotional situations and distraction in high-intensity situations. Regulatory preferences that deviate from the flexible regulation choice observed in healthy individuals might be related to different psychopathologies. Specifically, deviation from a preference to choose to disengage from very high-intensity stimuli is expected from individuals who are prone to develop major depression. According to the response style theory, rumination involves engaging with strong emotional experiences, and repeatedly thinking about their causes and consequences in an abstract and repetitive way (e.g., Nolen-Hoeksema, 1991; see Nolen-Hoeksema et al., 2008, for reviews). Although rumination has been proven to be related to onset maintenance and relapse of depression, depressed individuals who prefer to use positive distractions when dealing with strong emotional experiences show a better prognosis (e.g., Nolen-Hoeksema & Morrow, 1991, 1993). Emotion regulation choice is likely to be an important target in the context of depression, because empirical studies have shown that although depressed individuals are able to implement distraction effectively when instructed to do so (e.g., Joormann & Siemer, 2004; Joormann, Siemer, & Gotlib, 2007), they hold a favorable view of rumination, believing that it helps them understand better the reasons for depressed mood. Nevertheless, an overgeneralized preference to engage with emotional contents may also depend on impaired ability to shift attention away from negative contents (e.g., Joormann & Gotlib, 2008).

Another type of deviation from the healthy regulatory choice patterns involves diverging from engagement with low-intensity emotional stimuli. Common to several anxiety disorders is a tendency to overgeneralize a disengagement or avoidance regulatory response (for reviews, see Campbell-Sills & Barlow, 2007; Foa & Kozak, 1986). Avoidance usually starts in response to high-intensity emotional stimuli, but over time, it ends up spilling over to seemingly low-intensity stimuli. As pointed in our conceptual model, while disengagement strategies are helpful in providing short-term relief, they are maladaptive in the long run and can perpetuate anxiety and fears.

The cognitive factor highlighted is the ease with which a regulation strategy is generated. Specifically, aiding the generation process resulted in increased reappraisal choice. These results are relevant for many cognitive-behavioral therapies in which patients are encouraged to reappraise strong emotional events (e.g., Campbell-Sills & Barlow, 2007). While the final objective is to have patients generate their own regulation strategies, therapists should be mindful that their patients might need aid in generating alternative ways to think about upsetting events, until they can gradually build their skill. Complementarily, one can expect that with continued practice in reappraisal, patients would improve its generation and choose to use it more frequently when it's adaptive.

The motivational factor highlighted individuals' goals when choosing to regulate their emotions. It was shown that when participants expect to encounter emotional events repeatedly, they increase reappraisal choice, which offers long-term benefits. The ability to override regulatory preferences that offer short-term relief (i.e., distraction) in favor of regulatory preferences that offer long-term benefits (i.e., reappraisal) is likely to require self-control, and impairments in self-control ability have been linked to various psychopathologies, including addictions and eating disorders such as bulimia (for comprehensive reviews, see Heatherton & Baumeister, 1991; Vohs & Baumeister, 2011).

Future Directions

Despite promising preliminary results and clear, broad implications, research in emotion regulation choice has only begun to develop. In closing I wish to point out several potential future research directions.

First, to date, researchers have examined the influence of only one emotional (emotional intensity), one cognitive (generation of a strategy), and one motivational (short- vs. long-term goals) determinant of emotion regulation choice. While these factors are important, future studies should evaluate the influence of the many additional factors that likely influence regulatory preference. To give just one example, the availability of cognitive resources is likely to influence individuals' regulatory choices. Specifically, a temporary state of self-control resource depletion (e.g., Baumeister, Vohs, & Tice, 2007; Muraven & Baumeister, 2000) is likely to lead individuals to prefer strategies such distraction, which provide short-term relief. Therefore, studying how various novel factors affect regulatory choice is an important future research direction.

Second, while distraction and reappraisal are considered classic disengagement and engagement strategies (Parkinson & Totterdell, 1999), and they are widely used in everyday life, studies of choices between other emotion regulation strategies are urgently needed. In everyday life, individuals choose from many more regulatory options that should be studied in the future. Several promising avenues involve allowing people to choose from regulatory options that are considered less adaptive in certain contexts (e.g., suppression and rumination). Another option involves investigating how individuals switch their regulatory choices when dealing with dynamic and prolonged emotional events that constitute many of our daily experiences (see Aldao & Nolen-Hoeksema, 2012c, for a related discussion). Relatedly, a crucial test of the applicability of the emotion regulation choice paradigm and its supporting results should involve studying regulatory choices in daily emotional experiences, such as when patients wait to receive potentially stressful news in medical settings, or when clients face a stimulus about which they feel anxious. Although distraction and reappraisal are widely used in real-life situations, it remains unclear whether we would observe somewhat similar preferences when individuals spontaneously choose between regulatory options in daily emotional situations.

Third, the goals of this study were generally to characterize the influence of different factors on emotion regulatory choice. Nevertheless, it is quite clear that studying individual differences in emotion regulation choice is crucial. Recent relevant studies have shown that individual differences in the ability to modify emotions are tightly linked to long-term adaptation (e.g., Bonanno et al., 2004; Westphal et al., 2010). Therefore, future studies should evaluate how multiple individual differences can moderate the influence of central factors on emotion regulation choice. One promising venue,

which I mentioned in discussing the clinical implications, involves studying impairments in regulatory preferences of individuals with mood, anxiety, and personality disorders, whose psychopathology revolves around emotion dysregulation.

Fourth, according to the emotion regulation choice account, healthy individuals are sensitive to central costs and benefits of different regulation strategies when trying to choose between regulatory options, and in general these individuals showed adaptive regulatory choice profiles. Nevertheless, multiple demonstrations in general decision-making studies have shown important limitations in human reasoning (e.g., Tversky & Kahneman, 1974); therefore, future studies should investigate situations in which individuals' regulatory choices would not necessarily lead them to the best outcome. In a related vein, while conscious regulation strategies have been a major focus in the field of emotion regulation, and an integral part of many cognitive-behavioral therapies targeting emotion dysregulation (e.g., Linehan, 1993), many emotion regulation choices are likely to be determined implicitly and without deliberate control.

Finally, until now, emotion regulation choice has been investigated separately from studies on the consequences of implementing regulation strategies. Future studies should make stronger connections between the developing studies on emotion regulation choice and the well-established studies on the consequences of regulation implementation. For example, studies should evaluate whether the effectiveness of implementing a given strategy is moderated by an ability to override default regulatory choice preferences. At the same time, future studies in emotion regulation choice should utilize multiple levels of analysis that combine the concurrent assessment of the effectiveness of a chosen regulatory strategy.

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References

Aldao, A., & Nolen-Hoeksema, S. (2012a). When are adaptive strategies most predictive of psychopathology? *Journal of Abnormal Psychology, 121*, 276–281.

Aldao, A., & Nolen-Hoeksema, S. (2012b). The influence of context on the implementation of adaptive emotion regulation strategies. *Behaviour Research and Therapy, 50*, 493–501.

Aldao, A., & Nolen-Hoeksema, S. (2012c). One versus many: Capturing the use of multiple emotion regulation strategies in response to an emotion-eliciting stimulus. *Cognition and Emotion*. [E-publication ahead of print]

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*, 217–237.

Altamirano, L. J., Miyake, A., & Whitmer, A. J. (2010). When mental flexibility facilitates cognitive control: Beneficial side effects of ruminative tendencies on goal maintenance. *Psychological Science, 21*, 1377–1382.

Baumeister, R. F., Vohs, K. D., & Tice, D. M. (2007). The strength model of self-control. *Current Directions in Psychological Science, 16*, 396–403.

Blechert, J., Sheppes, G., Di Tella, C., Williams, H., & Gross, J. J. (2012). See what you think: Reappraisal modulates behavioral and neural responses to social stimuli. *Psychological Science, 4*, 356–363.

Bonanno, G. A. (2005). Resilience in the face of potential trauma. *Current Directions in Psychological Science, 14*, 135–138.

Bonanno, G. A., & Keltner, D. (1997). Facial expressions of emotion and the course of conjugal bereavement. *Journal of Abnormal Psychology, 106*, 126–137.

Bonanno, G. A., Papa, A., Lalande, K., Westphal, M., & Coifman, K. (2004). The importance of being flexible: The ability to both enhance and suppress emotional expression predicts long term adjustment. *Psychological Science, 15*, 482–487.

Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and motivation I: Defensive and appetitive reactions in picture processing. *Emotion, 1*, 276–298.

Bradley, M. M., Codispoti, M., Sabatinelli, D., & Lang, P. J. (2001). Emotion and motivation II: Sex differences in picture processing. *Emotion, 1*, 300–319.

Campbell-Sills, L., & Barlow, D. H. (2007). Incorporating emotion regulation into conceptualizations and treatments of anxiety and mood disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 542–559). New York: Guilford Press.

Chajut, E., & Algom, D. (2003). Selective attention improves under stress: Implications for theories of social cognition. *Journal of Personality and Social Psychology, 85*, 231–248.

de Groot, A. D. (1978). *Thought and choice in chess* (2nd ed.). The Hague: Mouton.

Dillon, D. G., Ritchey, M., Johnson, B. D., & LaBar, K. S. (2007). Dissociable effects of conscious emotion regulation strategies on explicit and implicit memory. *Emotion, 7*, 354–365.

Dunning, J. P., & Hajcak, G. (2009). See no evil: Directed visual attention modulates the electrocortical response to unpleasant images. *Psychophysiology, 46*, 28–33.

Ehring, T., Tuschen-Caffier, B., Schnülle, J., Fischer, S., & Gross, J. J. (2010). Emotion regulation and vulnerability to depression: Spontaneous versus instructed use of emotion suppression and reappraisal. *Emotion, 10*, 563–572.

Erber, R., & Tesser, A. (1992). Task effort and the regulation of mood: The absorption hypothesis. *Journal of Experimental Social Psychology, 28*, 339–359.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99*, 20–35.

Foti, D., & Hajcak, G. (2008). Deconstructing reappraisal: Descriptions preceding arousing pictures modulate the subsequent neural response. *Journal of Cognitive Neuroscience, 20*, 977–988.

Garnefski, N., Kraaij, V., & Spinhoven, P. (2001). Negative life events, cognitive emotion regulation and emotional problems. *Personality and Individual differences, 30*, 1311–1327.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*, 271–299.

Gross, J. J. (2001). Emotion regulation in adulthood: Timing is everything. *Current Directions in Psychological Science, 10*, 214–219.

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*, 281–291.

Gross, J. J. (Ed.). (2007). *Handbook of emotion regulation*. New York: Guilford Press.

Gross, J. J. (2010). The future's so bright, I gotta wear shades. *Emotion Review, 2*, 212–216.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*, 348–362.

Gross, J. J., Sheppes, G., & Urry, H. L. (2011a). Emotion generation and emotion regulation: A distinction we should make (carefully). *Cognition and Emotion, 25*, 765–781.

Gross, J. J., Sheppes, G., & Urry, H. L. (2011b). Taking one's lumps while doing the splits: A big tent perspective on emotion generation and emotion regulation. *Cognition and Emotion, 25*, 789–793.

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York: Guilford Press.

Gruber, J., Harvey, A. G., & Gross, J. J. (2012). When trying is not enough: Emotion regulation and the effort-success gap in bipolar disorder. *Emotion, 12*(5), 997–1003.

Gupta, S., & Bonanno, G. A. (2011). Complicated grief and deficits in emotional expressive flexibility. *Journal of Abnormal Psychology, 120*, 635–643.

Hajcak, G., Dunning, J. P., & Foti, D. (2009). Motivated and controlled attention to emotion: Time-course of the late positive potential. *Clinical Neurophysiology, 120*, 505–510.

Hajcak, G., MacNamara, A., & Olvet, D. M. (2010). Event-related potentials, emotion, and emotion regulation: An integrative review. *Developmental Neuropsychology, 35*, 129–155.

Hajcak, G., & Nieuwenhuis, S. (2006). Reappraisal modulates the electrocortical response to negative pictures. *Cognitive, Affective, and Behavioral Neuroscience, 6*, 291–297.

Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. *Psychological Bulletin, 110*, 86–108.

Hübner, R., Steinhauser, M., & Lehle, C. (2010). A dual-stage two-phase model of selective attention. *Psychological Review, 117*, 759–784.

John, O. P., & Gross, J. J. (2007). Individual differences in emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 351–372). New York: Guilford Press.

Johnston, W. A., & Heinz, S. P. (1978). Flexibility and capacity demands of attention. *Journal of Experimental Psychology: General, 107*, 420–435.

Joormann, J., & Gotlib, I. H. (2008). Updating

the contents of working memory in depression: Interference from irrelevant negative material. *Journal of Abnormal Psychology, 117*, 182–192.

Joormann, J., & Siemer, M. (2004). Memory accessibility, mood regulation and dysphoria: Difficulties in repairing sad mood with happy memories? *Journal of Abnormal Psychology, 113*, 179–188.

Joormann, J., Siemer, M., & Gotlib, I. H. (2007). Mood regulation in depression: Differential effects of distraction and recall of happy memories on sad mood. *Journal of Abnormal Psychology, 116*, 484–490.

Kalisch, R. (2009).The functional neuroanatomy of reappraisal: Time matters. *Neuroscience Biobehavioral Reviews, 33*, 1215–1226.

Kashdan, T. B., & Rottenberg, J. (2010). Psychological flexibility as a fundamental aspect of health. *Clinical Psychology Review, 30*, 865–878.

Kool, W., McGuire, J. T., Rosen, Z., & Botvinick, M. M. (2010). Decision making and the avoidance of cognitive demand. *Journal of Experimental Psychology: General, 139*, 665–682.

Koole, S. L. (2009). The psychology of emotion regulation: An integrative review. *Cognition and Emotion, 23*, 4–41.

Kross, E., & Ayduk, O. (2008). Facilitating adaptive emotional analysis: Distinguishing distanced-analysis of depressive experiences from immersed-analysis and distraction. *Personality and Social Psychology Bulletin, 34*, 924–938.

Lehle, C., & Hübner, R. (2008). On-the-fly adaptation of selectivity in the flanker task. *Psychonomic Bulletin and Review, 15*, 814–818.

Linehan, M. M. (1993). *Skills training manual for treating borderline personality disorder.* New York: Guilford Press.

MacNamara, A., Ochsner, K. N., & Hajcak, G. (2011). Previously reappraised: The lasting effect of description type on picture-elicited electrocortical activity. *Social, Cognitive, and Affective Neuroscience, 6*, 348–358.

Mancebo, M. C., Eisen, J. L., Grant, J. E., & Rasmussen, S. A. (2005). Obsessive–compulsive personality disorder and obsessive–compulsive disorder: Clinical characteristics, diagnostic difficulties, and treatment. *Annals of Clinical Psychiatry, 17*, 197–204.

McRae, K., Ciesielski, B. R. G., & Gross, J. J. (2012). Unpacking cognitive reappraisal: Goals, tactics and outcomes. *Emotion, 12*, 250–255.

Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin, 126*, 247–259.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100*, 569–582.

Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and post-traumatic stress symptoms following a natural disaster: The 1989 Loma Prieta Earthquake. *Journal of Personality and Social Psychology, 61*, 115–121.

Nolen-Hoeksema, S., & Morrow, J. (1993). Effects of rumination and distraction on naturally-occurring depressed mood. *Cognition and Emotion, 7*, 561–570.

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3*, 400–424.

Ochsner, K. N., & Gross, J. J. (2008). Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. *Current Directions in Psychological Science, 17*, 153–158.

Ochsner, K. N., Ray, R. D., Robertson, E. R., Cooper, J. C., Chopra, S., Gabrieli, J. D. E., et al. (2004). For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *NeuroImage, 23*(2), 483–499.

Parkinson, B., & Totterdell, P. (1999). Classifying affect regulation strategies. *Cognition and Emotion, 13*, 277–303.

Pashler, H. (1998). *The psychology of attention.* Cambridge, MA: MIT Press.

Payne, J. W., Bettman, J. R., & Johnson, E. J. (1988). Adaptive strategy selection in decision making. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 14*, 534–552.

Payne, J. W., Bettman, J. R., & Johnson, E. J. (1993). *The adaptive decision maker.* New York: Cambridge University Press.

Reynolds, B. (2006). A review of delay-discounting research with humans: Relations to drug use and gambling. *Behavioural Pharmacology, 17*, 651–667.

Richards, J. M., & Gross, J. J. (1999). Compo-

sure at any cost?: The cognitive consequences of emotion suppression. *Personality and Social Psychology Bulletin, 25*, 1033–1044

Richards, J. M., & Gross, J. J. (2000). Emotion regulation and memory: The cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology, 79*, 410–424.

Rieskamp, J. (2006). Perspectives on probabilistic inferences: Reinforcement learning and an adaptive network compared. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 32*, 1355–1370.

Rieskamp, J., & Otto, P. E. (2006). SSL: A theory of how people learn to select strategies. *Journal of Experimental Psychology: General, 135*, 207–236.

Sheppes, G., Catran, E., & Meiran, N. (2009). Reappraisal (but not distraction) is going to make you seat: Physiological evidence for self control effort. *International Journal of Psychophysiology, 71*, 91–96.

Sheppes, G., & Gross, J. J. (2011). Is timing everything?: Temporal considerations in emotion regulation. *Personality and Social Psychology Review, 15*, 319–331.

Sheppes, G., & Gross, J. J. (2012). Emotion regulation effectiveness: What works when. In H. A. Tennen & J. M. Suls (Eds.), *Handbook of psychology* (2nd ed., pp. 391–406). Indianapolis, IN: Wiley-Blackwell.

Sheppes, G., & Meiran, N. (2007). Better late than never?: On the dynamics of on-line regulation of sadness using distraction and cognitive reappraisal. *Personality and Social Psychology Bulletin, 33*, 1518–1532.

Sheppes, G., & Meiran, N. (2008). Divergent cognitive costs for online forms of reappraisal and distraction. *Emotion, 8*, 870–874.

Sheppes, G., Scheibe, S., Suri, G., & Gross, J. J. (2011). Emotion-regulation choice. *Psychological Science, 22*, 1391–1396.

Sheppes, G., Scheibe, S., Suri, G., Radu, P., Blechert, J., & Gross, J. J. (2012). Emotion regulation choice: A conceptual framework and supporting evidence. *Journal of Experimental Psychology: General*. [E-publication ahead of print]

Siegler, R. S. (2005). Children's learning. *American Psychologist, 60*, 769–778.

Tamir, M. (2011). The maturing field of emotion regulation. *Emotion Review, 3*, 3–7.

Thiruchselvam, R., Blechert, J., Sheppes, G., Rydstrom, A., & Gross, J. J. (2011). The temporal dynamics of emotion regulation: An EEG study of distraction and reappraisal. *Biological Psychology, 87*, 84–92.

Troy, A. S., & Mauss, I. B. (2011). Resilience in the face of stress: Emotion regulation ability as a protective factor. In S. Southwick, B. Litz, D. Charney, & M. Friedman (Eds.), *Resiliency and mental health* (pp. 30–44). Cambridge, UK: Cambridge University Press.

Tversky, A., & Kahneman, D. (1974). Judgment under uncertainty: Heuristics and biases. *Science, 185*, 1124–1131.

van Dillen, L. F., & Koole, S. L. (2007). Clearing the mind: A working memory model of distraction from negative mood. *Emotion, 7*, 715–723.

Vohs, K. D., & Baumeister, R. (Eds.). (2011). *Handbook of self-regulation: Research, theory, and applications* (2nd ed.). New York: Guilford Press.

Vohs, K. D., Baumeister, R. F., Schmeichel, B. J., Twenge, J. M., Nelson, N. M., & Tice, D. M. (2008). Making choices impairs subsequent self-control: A limited resource account of decision making, self-regulation, and active initiative. *Journal of Personality and Social Psychology, 94*, 883–898.

Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin, 134*, 163–206.

Watkins, E. (2011). Dysregulation in level of goal and action identification across psychological disorders. *Clinical Psychology Review, 31*, 260–278.

Westphal, M., Seivert, N. H., & Bonanno, G. A. (2010). Expressive flexibility. *Emotion, 10*, 92–100.

Wilson, T. D., & Gilbert, D. T. (2008). Explaining away: A model of affective adaptation. *Perspectives on Psychological Science, 3*, 370–386.

CHAPTER 9

Emotion Regulation and Decision Making

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Emotion and Decision Making

Definition of the Field

Recent years have seen a notable increase in interest in examining the processes that underlie human decision making, with one of the more energetic new directions being that of how emotions can impact our decisions and choices (Sanfey, 2007). Though a relatively recent development, this research area has already uncovered some compelling findings with regard to the emotional and social factors involved in how we make decisions. Indeed, it is now commonly argued that the principal way in which actual decision-making behavior deviates from the choices prescribed by economic models of decision making may be in large part due to emotional factors that weigh heavily on our actual decision but are seldom taken into account by the standard models. Hence, greater knowledge of the exact role that emotions play in decision making is invaluable in building complete, accurate, models of choice.

The Role of Emotion in Decision Making

Historically, the study of how we make decisions has been tremendously influenced by economic theories grounded in mathematical models of behavior. These models typically strive to outline what the correct decision might be when selecting from a specified choice set, with these models generally assuming that decisions are based on a "rational," cognitive evaluation of their consequences (Sanfey, 2007). While the explicit and analytically tractable solutions have certainly been of use in examining how we make decisions, and in particular perhaps how we *should* make decisions, factors such as emotions, mood, and social cues have typically been excluded from these models. However, in recent decades there has been a welcome increased focus on the roles these factors play in decision making, and experimental evidence has begun to clarify greatly how emotions impact our decision behavior.

Emotions can affect individual's goals, attitudes, and social decision making (Forgas, 2003; Zajonc, 2000). For example, unpleasant emotions are associated with lower confidence and a more risk-averse and vigilant processing style (Clark & Isen, 1982; Isen & Daubman, 1984).

On the other hand, pleasant emotions have been associated with higher confidence, more optimistic framing, and greater cooperation (Forgas, Bower, & Moylan, 1990). More related to economic decisions is the work done by researchers in the field of neuroeconomics. One of the first well-

characterized demonstrations of the relationship between emotions and economic choices famously showed that damage to particular neural areas can radically bias decision making, with the lesioned areas of interest thought to contribute important emotional information to choice (Damasio, 1994; Sanfey, Rilling, Aronson, Nystrom, & Cohen, 2003). Continuing in this vein, many other basic decision-making studies have now shown that physiological indices, such as skin conductance, correlate with other decision-relevant information, such as imminent losses in a gambling task (Bechara, Damasio, Tranel, & Damasio, 1997) and anger during negative interpersonal interactions (Van't Wout, Kahn, Sanfey, & Aleman, 2006).

Other studies have looked at how emotional responses to the stimuli themselves can directly impact decision making. For example, in a functional magnetic resonance imaging (fMRI) experiment, Sanfey et al. (2003) showed that activation of the anterior insula, known to be associated with disgust and anger (Phillips et al., 1997), correlated with the rejection of inequitable financial offers made by an opponent in an economic task known as the Ultimatum Game. The interpretation given was that insula activation reflected negative emotional reactivity in response to the unfair offers (Sanfey et al., 2003). This hypothesis was tested in a follow-up study (Van't Wout et al., 2006), which showed that skin conductance responses, a measure for emotional arousal, were higher for unfair offers, and this measure discriminated between acceptances and rejections of these offers.

Researchers have also carefully manipulated emotions either before or during choices to demonstrate the causality of the relationship (Lerner, Small, & Loewenstein, 2004; Winkielman, Berridge, & Willbarger, 2005; Harlé & Sanfey, 2007; Andrade & Ariely, 2009; Sokol-Hessner, Camerer, & Phelps, 2013). Recent research in this vein suggests that task-irrelevant mood states may bias decision making (Harlé, Chang, Van't Wout, & Sanfey, 2012). For example, Harlé and Sanfey (2007) showed that negative emotion is associated more with lower acceptance rates of unfair monetary. What rational models consider suboptimal (i.e., turning down monetary gain) therefore appears likely to reflect the activation of emotional variables that can be additionally increased by subtle incidental mood states (Harlé & Sanfey, 2007), and, given that the role of emotions in human social and individual decision making has been widely demonstrated (Slovic, Finucane, Peters, & MacGregor, 2004), suggests that the existing models of behavior need to incorporate affective factors to arrive at a more comprehensive accounting of human decision-making behavior.

As a result of this understanding, researchers have begun to incorporate emotional influences into decision process models by assuming the reciprocal modulation of affective and more deliberative modes of thought (Harlé & Sanfey, 2007). One useful framework to interpret the role of emotions in decision making suggests that our choices may be best understood as the operation of multiple underlying systems that interact to form our decisions (Sanfey & Chang, 2008).

There is a long tradition in psychology for a dual-systems approach in the understanding of cognitive processes, and one aspect of dual systems that is relevant for this chapter is the distinction between emotion-based and cognitive-based judgments. Emotions are rapid and automatic responses to specific stimuli (Sanfey & Chang, 2008); however, in some circumstances we are required to adopt more flexible and context-specific solutions. At a neural level, emotional processes activate brainstem reward processing regions, such as the basal ganglia; ventral parts of the brain, such as the ventromedial frontal, orbitofrontal, and anterior cingulate cortex; and amygdala and insular cortex (Dalgleish, 2004; Sanfey & Chang, 2008). Deliberative processing engages anterior and dorsolateral and posterior regions of the brain (Duncan, Emslie, Williams, Johnson, & Freer, 1996; Smith & Jonides, 1999; Miller & Cohen, 2001; Stuss & Knight, 2002), whereas automatic processes activate more parietal and subcortical systems. It seems evident that emotions play a role in decision making in general; however, it is still largely unknown how these processes interact with deliberative systems and whether they can be controlled in a "top-down" fashion (Grecucci, Giorgetta, Bonini, & Sanfey, 2013a, 2013b). One proposal on how the two systems may interact comes from Evans (2009) who conceptualized the

conflict between these two styles of thinking as a "cognitive control problem," referring to the fundamental question of the mechanism by which control over the answer is ultimately allocated, and suggested how this conflict could be resolved. Evans pointed out that when confronted with a decision, automatic processing produces default intuitive responses, unless slower and more reflective deliberative processing intervenes. Importantly, such an intervention may or may not lead to a different decision that in turn may or may not be the correct one. However, the linkage between emotions and decision making may be more complex and multifaceted, as suggested by the "fourfold classification of emotions and decision-making" model of Pfister and Böhm (2008), according to which emotions provide information about pleasure and pain during preference construction, enable rapid choices under time pressure, lead attention on relevant aspects of a decision problem, and may generate commitment concerning morally and socially significant decisions.

Emotion Regulation and Decision Making

Overview

One useful approach to investigate how emotion and cognition interact is to examine whether regulatory strategies applied to decision-making tasks have the ability to alter subjects' decisions by modulating the ongoing emotions (Van't Wout, Chang, & Sanfey, 2010). Emotion regulation refers to a set of different strategies by which individuals influence which emotions they have, when they have them, and how they experience and express them. Emotions can intervene in different ways in our decisions, starting from the assessment of potential alternative options, the evaluation of a choice, the various possible outcomes, and so on. Although there is surprisingly little empirical work on the link between top-down control of emotions and the act of decision making, the experimental use of emotion regulation strategies has great potential to elucidate this relationship (Van't Wout et al., 2010), that is, to clarify how emotions and decision making interact. Broadly speaking, emotion regulation strategies applied to decision making have two notable advantages as compared to basic emotion regulation studies: (1) It provides the opportunity to see how these regulatory strategies can actually affect behavioral responses themselves and not only emotional perception per se, and (2) it allows for the study of complex emotions that are often difficult to elicit via standard sets of simple perceptual stimuli.

Though there has been much important recent progress on understanding emotion regulation, most studies to date have mainly measured the ability to regulate negative emotions induced by the viewing of images of valenced and arousing scenes (see Ochsner & Gross, 2008, for a review). However, in daily life we are confronted with an often bewildering range of decisions, each containing an evaluation of gains, losses, and associated risks, and usually laden with emotional value. Therefore, investigating whether emotion regulation strategies can have an effect on actual decisions provides an exciting opportunity to extend emotion regulation research beyond the modulation of emotional responses per se.

The effectiveness of emotion regulation strategies is dependent on both the specific type of stimulus and the emotion to be regulated (Grecucci, 2012; Ochsner & Gross, 2008), and the emotions elicited by decision-making situations are substantially different than those experienced while passively viewing emotional images (Van't Wout et al., 2010). In addition to the benefits of better understanding the processes of emotional regulation by examining these mechanisms in concert with decision making, the field of decision making itself also stands to benefit from this research program. Decision making and emotion regulation are linked by the role that emotion plays in both (Mitchell, 2011), and emotions appear to play a fundamental role in choices associated with reward or punishment. Moreover, psychiatric disorders are often associated with emotion regulation deficits and abnormal decision making (Mitchell, 2011). For example, neuropsychological studies have shown that damage in brain areas implicated in emotional regulation (i.e., ventromedial cortex) often result in suboptimal decision making (Koenigs & Tranel, 2007; Moretti, Dragone, & di Pelegrino, 2009). A recent line of research has studied how emotion regulation

strategies can affect decision making directly and bias our decision-making behavior as presented in the following sections.

Individual Decision Making

One useful organizational distinction when talking about decision making is to separate the field into two main branches: individual decision making and social decision making. This categorization is not trivial, and it has particular implications for emotion regulation, because the affective experience associated with these two kinds of decision can be quite different. Individual decision making refers to situations in which the decision makers assess the available options, then choose the best course of action for themselves based on their own value and beliefs, for example, when choosing to play a risky bet. Factors typically considered here include risk evaluation, reward, loss aversion, regret, and quantity evaluation. Social decision scenarios also require that these factors be taken into account, but what distinguishes these situations is the presence of another (involved) agent. A good example of this is making decisions in a business negotiation context. These contexts additionally involve evaluations of motivations such as fairness, equity, cooperation, and in general more socially driven emotions. Here, we first examine the experimental evidence of emotion regulation on individual decision making, before turning our attention to the social contexts.

The Modulation of Risk Evaluation and Risk-Seeking Behavior

One method to study the interaction between emotion and decision making is examining risky choices, for example, when there is uncertainty about the exact outcome of the decision and, typically, a safe, relatively low payout or low-cost option is weighed against an option with both higher costs and benefits. A canonical example would be a choice between a sure gain of $10 and a risky choice, with a 50% chance of paying out $25. The importance of studying the effect of emotion regulation strategies in modulating risky behaviors becomes particularly salient when considering that many pathological behaviors stem from seemingly irrational evaluation of the risk associated with a given choice (pathological gambling, substance abuse, etc.).

Two recent studies have manipulated people's emotion regulation strategies when making risky decisions. One study from Heilman, Crişan, Houser, Miclea, and Miu (2010) used cognitive reappraisal (an antecedent-focused strategy that changes the meaning of the situation), expressive suppression (a response-focused strategy that involves inhibiting behavioral expressions of emotions), and a control baseline condition in a between-subjects design (Study 1). Subjects were required to apply these strategies while playing the Balloon Analogue Risk Task (BART; Lejuez et al., 2002), as well as the Iowa Gambling Task (IGT; Bechara, Damasio, Damasio, & Anderson, 1994). The BART provides a vivid measure of risk taking by giving players the opportunity to progressively earn money by pumping up computerized balloons (Lejuez et al., 2002). While each additional pump provides an extra monetary payoff, the balloons can explode without warning, in which case the participant loses everything. Therefore, the task assesses the degree to which players are prepared to risk an extra pump (for an extra reward) at the risk of a total loss (Lejuez et al., 2002). The well-known IGT is a computerized card-searching game that measures the degree to which individuals shift from choices with large immediate gains (which, however, are also associated with even larger losses in the long term) toward the more prudent and eventually more rewarding choices with small, immediate gains (Bechara et al., 1994).

Heilman et al. (2010) found that when compared with the control group, the group of reappraisers reported fewer negative emotions and demonstrated reduced risk aversion; that is, they made more risky decisions in the BART task, operationalized as a greater number of pumps on average per unexploded balloon. In the IGT, reappraisers demonstrated improved performance in the prehunch/hunch period of the task, in which players are endeavoring to discover the exact traits of the various cards; therefore, they yielded significantly higher earnings at the end of the game. Importantly, suppressors and controls showed no significant difference in both tasks in terms

of emotional ratings and decision-making behavior. Overall, then, this study showed that cognitive reappraisal appears to lead to increased risk taking by reducing the experience of negative emotions elicited by the decision scenario.

Another study (Martin & Delgado, 2011) explored how emotion regulation strategies influence participants' preferences when choosing between two monetary options: a gamble (risky option) and a guaranteed amount (safe option). Here, participants engaged in imagery-focused regulation strategies during the presentation of a cue that preceded an economic decision. The emotion regulation strategies placed subjects in the following experimental conditions: "Look," "Relax," or "Excite." When instructed to "Relax," participants were asked to imagine a calming scene; when in the "Excite" condition, participants were to imagine an exciting scene, in order to increase their reactivity. After the imagery phase, participants made a choice between a risky and a safe monetary lottery while undergoing fMRI. Participants who successfully used the emotion regulation strategy "Relax" made fewer risky choices in comparison to the "Look" condition. The "Excite" condition was not effective in producing any change in decision behavior. To better understand the effect of the "Relax" strategy on decision making, the authors divided participants into two groups on the basis of their success at implementing the strategy. Regulators were then compared with nonregulators, and the former showed a significant proportion of fewer risky choices as compared with "Look" trials, suggesting that emotion regulation strategies are able to modulate risky decisions (Martin & Delgado, 2011). Moreover, at the neural level, responses in the striatum, a key region associated with risky choice, were attenuated during decision making when participants adopted the "Relax" emotion regulation strategy (Martin & Delgado, 2011). In contrast, participants who did not effectively use emotion regulation strategies (i.e., the nonregulator group) failed to show neural differences during decision making. In conclusion, the authors argued that imagery-focused strategies "can foster more goal-directed behavior and promote safer, compared with riskier, decision-making."

Anticipation and Processing of Losses: The Regulation of Loss Aversion

Kahneman and Tversky (1979) famously codified the notion that losses loom larger than their equivalent gains, terming this phenomenon "loss aversion." Experimental studies over the past 30 years indicate that people demonstrate loss aversion for objects (Kahneman, Knetsch, & Thaler, 1990) and money (Tversky & Kahneman, 1992). Two studies from Sokol-Hessner and his colleagues (2009, 2013) showed that applying emotion regulation strategies to decision making can affect the experience of loss aversion. In the first study (Sokol-Hessner et al., 2009), participants were asked to reappraise, by taking a different perspective, about a choice between a binary gamble and a guaranteed amount. Outcomes were displayed following the decision phase (e.g., "You won" or "You lost"). For half of the trials, participants applied an "Attend" strategy, which considered each choice in isolation, and for the other half, a "Regulate" strategy, which emphasized choices in their broader context, such as "thinking like a trader." In other words, they were asked to imagine themselves to be experienced professional stock traders. Risk aversion and loss aversion indices were then computed, based on participants' actual choice behavior. The authors were able to show that the "Regulate" strategy had a strong effect in decreasing individuals' initial levels of loss aversion but not risk aversion. Importantly, to identify the degree of change in loss aversion according to differences in regulation success, subjects were divided into "regulators" and "nonregulators." Reduced loss aversion was also associated with reduced physiological arousal responses to loss outcomes, further showing that the reappraisal strategy was also associated with decreased emotional reaction to bad outcomes.

In the second study, using a similar paradigm, Sokol-Hessner et al. (2013) found that success in regulating loss aversion by using emotion regulation strategies (reappraisal), was associated with a reduction in amygdala responses to losses but not to gains. Moreover, the application of the reappraisal strategy increased the baseline activity in prefrontal cortices and the striatum, in line with previous studies (Ochsner, Bunge,

Gross, & Gabrieli, 2002; Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Eippert et al., 2007; Delgado, Gillis, & Phelps, 2008; Delgado, Nearing, LeDoux, & Phelps, 2008). These results support the idea that different modes of thinking can alter both choices and arousal responses related to loss aversion.

Regulation of Rewards, Expected Values, and Prediction Error

Another line of research has involved the study of rewards in an economic decision-making context. A *reward* can be defined as "an object or event that generates approach and consummatory behavior, represents positive outcomes of decisions and may engage positive emotions" (cf. Schultz, 2009, p. 323). In other words, rewards are positive outcomes that occur after specific events.

The importance of studying emotion regulation strategies of reward stems from the fact that dysregulation of reward seems to be associated with pathological addiction syndromes (Heinz, 2002; Robinson & Berridge, 2003; Volkow & Wise, 2005; Wrase et al., 2007). At the neural level, when the reward is greater than expected, there is a positive prediction error (greater ventral striatal activity), and when the reward is less than expected (or missing altogether), there is a negative prediction error (less ventral striatal activity) (Hollerman & Schultz, 1998; McClure, Berns, & Montague, 2003; Staudinger, Erk, Abler, & Walter, 2009). Prediction errors have been interpreted as "teaching" signals, helping participants to adapt their behavior so as to maximize future rewards acquisition (Sutton, 1988; Montague, Hyman, & Cohen, 2004; Staudinger et al., 2009).

Two studies from Staudinger and colleagues (2009; Staudinger, Erk, & Walter, 2011) addressed the question of whether emotion regulation might also impact reward processing. In the first study, Staudinger et al. (2009), the authors used a monetary incentive delay (MID) task (Knutson, Taylor, Kaufman, Peterson, & Glover, 2005) to investigate whether regulation strategies affect the neural processing of reward and its associated motivated behavior. Subjects were scanned with fMRI while playing the MID task, in which subjects observed an abstract cue indicating the amount of money to be won (€0.50, €0.10), followed by a waiting period. A target (square or triangle) was then presented, after which subjects became eligible to win the announced amount of money. Immediately afterwards, a feedback display informed the subjects whether they had actually won (€0.50, €0.10) or not (€0.00). Subjects in one condition were asked to rethink an emotion-eliciting situation in nonemotional terms by distancing themselves from feelings and behaving as neutral observers (distancing; Gross, 2002). In another condition ("Permit") they were allowed to experience their feelings. Distancing significantly lowered subjects' perceived feelings. More importantly, a modulation of expected value and its associated prediction error was observed in the right ventral striatum of subjects while applying the distancing strategy. The comparison of the "Distance" and "Permit" conditions showed a significant activation in the right-hemispheric frontoparietal network (including the rostral cingulate zone, lateral orbitofrontal cortex [OFC] and inferior parietal lobe/temporal parietal junction). Interestingly, reappraisal success was positively correlated with activation in the rostral cingulate zone and the lateral OFC (Staudinger et al., 2009). The authors were therefore able to show that emotion regulation strategies generally impact reward coding, and also modulate expected value and prediction error encoding in the ventral striatum.

In the second study, Staudinger and colleagues (2011) used a similar task but explored whether emotion regulation would also be effective in decreasing the motivation to obtain a reward. The authors found reduced positive evaluative feelings for larger gains (€1) during the "Distance" instruction (from 4.08 to 2.50 points), as well as a slowdown in reaction times, which they interpreted as a lowered motivation to obtain the reward. At a neural level, using psychophysiological interaction analyses, they found that the dorsolateral prefrontal cortex played a fundamental role in exerting direct modulatory control over the striatum by promoting low-reward responses. In the same fashion, another experiment showed that imagery-based strategies successfully modulate reward expectation in a monetary reward–conditioning procedure (Delgado, Nearing, et al., 2008).

Overall, these experiments showed that emotion regulation strategies applied to individual decisions have the power to alter our choices. Emotions such as risk perception, reward, and loss evaluation are modulated by the usage of different kind of strategies (cognitive or relaxation-based imagery), and this alters our decision behavior toward safer behavior (Martin & Delgado, 2011), toward less sensitivity to outcomes (Sokol-Hessner et al., 2013), and toward lower motivation to obtain an economic reward (Staudinger et al., 2011).

Social Decision Making

Despite the extensive literature on emotional "self-regulation" (see Ochsner & Gross, 2008), evidence of emotion regulation in social interactive situations is still relatively scant. Indeed, interest in such "social regulation" has been explored only recently in a study examining the emotion regulation of subjects looking at pictures depicting social versus nonsocial scenes. Studying the mechanisms involved in the regulation of real social situations is particularly important when considering the failures to regulate interpersonal interactions exhibited in psychiatric disorders such as borderline personality disorder or social anxiety disorder (Grecucci, 2012, p. 135); Ochsner, 2008; Ochsner & Gross, 2008). Recent experiments on emotion regulation and social decision making hold the promise to elucidate the mechanisms subserving interpersonal emotion regulation (Grecucci, Giorgetta, Bonini, & Sanfey, 2013). Moreover, emotions elicited in social interactions may well be of a qualitatively different nature than those experienced while making individual decisions about monetary gains, rewards, or risky choices.

Anger

A potentially useful approach to measuring socially driven emotions is to adopt tasks derived from "game theory." Game theory is the study of mathematical models of conflict and cooperation between decision makers (Myerson, 1991), and offers useful models for the psychological investigation of social exchange (Sanfey & Dorris, 2009). One of the more well-known tasks in this field is the Ultimatum Game (UG; Guth, Schmittberger, & Schwarz, 1982), in which two players must agree on the division of a sum of money provided by the experimenter (Van't Wout et al., 2010). One player, the proposer, makes an offer as to how this money should be split. The responder must then make a decision to either accept or reject this offer. If the offer is accepted, then the money is split as proposed, but if the responder rejects the offer, then neither player receives anything. A central focus of the literature has been the well-replicated result that low offers (i.e., less than 30% of the total amount) are rejected approximately 50% of the time (Camerer, 2003). That is, UG choices are not purely driven by financial self-interest, but also incorporate affective information about the social interaction, with evidence that negative emotions play an important role in punishment behavior (Pillutla & Murnighan, 1996; Xiao & Houser, 2005). Put simply, the UG gives us the opportunity to elicit anger reactions in subjects via real social interactions, then to measure their willingness to punish the perpetrator (and, of course, themselves) by examining rejection rates of these unequal offers.

One study from Van't Wout and colleagues (2010) showed that participants who were asked to reappraise their emotions accepted a greater number of unfair offers (i.e., $1 or $2 offered from a pot of $10) than participants who did not use any regulation strategy (Experiment 1). Importantly, in another condition, subjects were asked to use a response-focused strategy, namely, suppression of any negative emotions toward the proposer; however, this strategy was not effective in altering the decision-making of subjects. Moreover, when acting later in the experimental session as proposers themselves (Experiment 2), participants who had used reappraisal strategies made more generous offers to their previously unfair partners than those who had use the strategy of suppression, demonstrating an interesting "follow-on" effect of reappraisal.

Following this line of research, Grecucci, Giorgetta, Van't Wout, Bonini, and Sanfey (2013) applied a similar paradigm to that of Van't Wout et al. (2010) but also employed fMRI to explore the neural bases of regulated

interpersonal decisions. In addition they studied the effects of both up- and down-regulation in participants. This allowed them to explore whether the negative emotions induced by an inequitable proposal can be both reduced and increased depending on the strategy, and, importantly, whether this regulation in turn affects the decision to punish the other player for his or her unfair treatment. When using a down-regulation strategy (*rethink the situation as less negative*), a greater number of unfair offers were accepted (i.e., fewer punishment behaviors). Up-regulation, on the other hand, yielded a greater number of rejections of unfair offers (i.e., more punishment behaviors), with both strategy conditions compared with a baseline "Look" condition. Subjective ratings analyses clarified that subjects' anger about the unfair proposed division was the main emotion regulated by the reappraisal strategies. At the neural level, significant activations during reappraisal were observed in the dorsolateral prefrontal cortex and cingulate gyrus, but only for unfair offers. "Regulated" decisions activated the left inferior frontal gyrus for up-regulation and the middle frontal gyrus for down-regulation strategies, respectively. The insula, one key region that was active when participants were treated unfairly (Sanfey et al., 2003), showed less activation for down-regulation and more activation for up-regulation. In conclusion, these studies provide strong evidence that interpersonal emotions (e.g., anger when treated unfairly) can be successfully modulated by emotion regulation strategies. And, as an important consequence, these studies also clearly show that emotion regulation can alter our social behavior (in this case, punishment of unfair behavior), with these effects observed in within-subject interactions. This has important implications for the regulation of social behavior in interactive contexts.

Selfishness

Another recent study by the same group (Grecucci, Giorgetta, Bonini, et al., 2013) used a similar socioeconomic game, namely, the Dictator Game, to measure emotional responses to both the altruistic and selfish behavior of others. The Dictator Game is similar to the UG, with the important exception that responders cannot reject the division of money made by the proposer; that is, they must accept whatever the proposer decides to give them from the experimenter-provided pot. Here, subjects were trained to reappraise the other player (a form of "mentalizing") in one condition, and to distance from the situation ("distancing") in another. This kind of reappraisal comprised reframing the intention and behavior of the other player in either a more or less negative way. Following both altruistic and selfish divisions of money, emotional ratings were collected separately for arousal and valence. Reappraisal strategies increased the valence ratings (less negative perception) for selfish offers ($1, $2, $3, from a total of $10), and subjective ratings also indicated that the main emotion involved when receiving such offers was "disappointment" (but also to some extent "anger" and "disgust") in relation to the selfish proposers, while distancing did not produce any change compared with the "Look" condition. This study provisionally demonstrates that disappointment regarding selfish behavior can be successfully modulated by mentalizing strategies (reappraisal of the intention of others), whereas distancing from others (an interpersonal strategy that is typical of social and generalized anxiety disorders) is not effective in modulating these reactions.

Overall, these experiments have begun to show that emotions in reaction to complex social motivations such as inequity and selfishness can be successfully modulated by emotion regulation strategies, and that these strategies can lead to changes in observed decision behaviors.

Conclusion

Our aim in this chapter has been to review the extant studies that have used regulatory strategies to moderate affective inputs to the decision-making process. Though relatively few in number to date, there has been encouraging success with these type of manipulations, and overall these studies provide compelling evidence that emotion regulation strategies can have a strong impact on not only perceptions of stimuli, but also

real, consequential, decisions in both social and nonsocial contexts. This turn can lead to interesting hypotheses about how our daily decisions can be modulated by emotion regulation strategies. Though it seems evident that emotional factors play a role in decision-making situations, the mechanisms integrating affective and more deliberative information in decision making are still relatively understudied. Recent experiments have demonstrated changes in decision making following both incidental emotion induction (Harlé & Sanfey 2007; Bonini et al., 2011) and neurophysiological disruption via transcranial magnetic stimulation (Van't Wout, Kahn, Sanfey, & Aleman, 2005; Knoch, Pascual-Leone, Meyer, Treyer, & Fehr, 2006), but emotion regulation studies of decision making have already extended these findings in important ways by demonstrating that emotions directly related to the choice situation itself can be controlled through top-down effort, leading to changes in how the choice sets are represented and the associated decisions themselves. Moreover, these studies are important because they extend the range of emotions that can be affected by emotion regulation strategies.

Neuroanatomical Bases of Emotion Regulation of Decision Making

Overall, the brain regions implicated in the aforementioned studies can be grouped into two main functional clusters: the regions that *implement* the regulation strategies, and the regions *regulated* by the strategies.

For the former, regions acting as "regulators" are primarily the dorsolateral (dlPFC) and ventrolateral (vlPFC) portions of the prefrontal cortex, and in some cases also the ventromedial regions. The dlPFC and vlPFC have been implicated in active cognitive control and inhibition (Miller & Cohen, 2001; Smith & Jonides, 1999), and may underlie the generation and maintenance of reappraisal strategies (Ochsner et al., 2002, 2004). Importantly, the ventral part of the prefrontal cortex is well-connected with brain structures such as the insula, the amygdala, and the striatum. One hypothesis derived from these activations is that the prefrontal cortex actively modulates the activity in more emotional regions under conditions of emotional regulation (Grecucci, Giorgetta, Bonini, et al., 2013a, 2013b). Interestingly, dlPFC and vlPFC have been previously reported as active when reappraising emotional pictures (Ochsner et al., 2004). One interpretation might therefore be that dlPFC is involved in modulating both behavioral and emotional outputs in order to satisfy contextual demands (Mitchell, 2011).

Interestingly, there is an overlap between "decision-making" and "regulatory" brain areas. Indeed, the role of the lateral prefrontal cortex in decision making has been conceptualized along four main lines: (1) negative feedback encoding; (2) motor response inhibition; (3) stimulus–response reconfiguration; and (4) attention to behaviorally significant stimuli (Mitchell, 2011). The same considerations apply to the role of the lateral PFC in emotion regulation. For example, Mitchell hypothesized that the lateral prefrontal cortex is involved in modulating behavioral and emotional output given particular contextual demands. Another key region connected to both emotion regulation and decision making is the cingulate cortex, which may best be interpreted in terms of performance monitoring. One of these monitoring functions is the detection of response conflict between top-down "deliberative" signals and bottom-up "emotional" signals. Other relevant regions for these processes are the temporoparietal junction (TPJ) and the inferior parietal lobe (IPL). Activation of the right TPJ/IPL is in line with previous reappraisal studies (Ochsner et al., 2002; Delgado, Nearing, et al., 2008; Staudinger et al., 2009) and might indicate allocation and dislocation of attention (Corbetta & Shulman, 2002).

The regions modulated by the strategies are generally connected with the emotions elicited in the tasks. For example, tasks involving risk assessment found reduced activity in the ventral striatum (Martin & Delgado, 2011); tasks involving reward found alterations in the activity of OFC, cingulate cortex, and the ventral striatum (Staudinger et al., 2009, 2011); experiments involving the experience of monetary loss found reduced amygdala activity (Sokol-Hessner et al., 2013); and social decision-making tasks such as the UG yielded reduced activity in the insula, known to be associated with anger and feelings of disgust (Grecucci, Giorgetta, Bonini, et al., 2013a). See Table 9.1 for a summary.

TABLE 9.1. Emotion Regulation and Decision Making Studies and Their Main Findings

Emotion or emotion-related behavior	Decision-making task	Emotion regulation strategy	Behavioral effect	Main regulated regions	Regulating regions	Reference
Risky behavior	Balloon Analogue Risk Task; Iowa Gambling Task	Suppression, reappraisal	Reduced risk aversion; increased monetary performance	—	—	Heilman et al. (2010)
Risk perception	Gambling	Imagery-focused regulation	Reduced risky choices	Ventral striatum		Martin and Delgado (2011)
Loss aversion	Gambling	Reappraisal	Reduced loss aversion	Amygdala	dlPFC, vmPFC	Sokol-Hessner et al. (2009, 2013)
Reward	Monetary Incentive Delay Task	Distancing	Attenuation of expected value; modulation of prediction error; attenuation of pleasant anticipation of gains	Ventral striatum, rostral cingulate, orbitofrontal cortex, putamen	dlPFC, CC, IPL/TPJ	Staudinger et al. (2009, 2011)
Anger	Ultimatum Game	Reapprasial, suppression	Reduced rejection rates	Insula	dlPFC, CC	Van't Wout et al. (2010); Grecucci, Giorgetta, Van't Wout, et al. (2013)
Responses to selfish behavior of others	Dictator Game	Reapprasial, distancing	Less negative emotions	Insula, Striatum, posterior cingulate	dlPFC, TPJ	Grecucci, Giorgetta, Bonini, and Sanfey (2013a, 2013b)

Note. dlPFC, dorsolateral prefrontal cortex; vmPFC, ventromedial PFC; CC, cingulate cortex; IPL, inferior parietal lobe; TPJ, temporoparietal junction.

Concluding Remarks and Future Directions

In this chapter, we have reviewed the current experimental evidence concerning emotion regulation as applied to decision making. While relatively few experiments to date have explicitly used regulation strategies in the context of decision and choice, the results thus far are encouraging and point to the usefulness of regulation in potentially modifying decision making. The experimental findings are consistent with a key role of emotional regulatory process in decision making. For example, expressing emotions when bargaining in the UG has effects on the subsequent punishment behavior (Xiao & Houser, 2005). Individual and social decision-making experiments have shown that emotions connected to relevant decision factors such as loss, risk, reward, interpersonal anger, and moral disgust can be successfully regulated by various strategies. Notably, not all the strategies result in equally successful changes in subjects' decision-making behavior. While reinterpretative strategies such as reappraisal appear effective at altering decision making and associated emotions, the same does not necessarily apply to expressive strategies such as suppression (Heilman et al., 2010; Van't Wout et al., 2010) or distancing (Grecucci, Giorgetta, Bonini, et al., 2013; but see Staudinger et al., 2009, for a positive result). Although researchers are now beginning to investigate the role of regulation in decision making, several factors still need to be understood and explored. For example, there are many different types of decision making that have been hitherto unexamined in terms of how regulation may impact choice, such as impulsivity, temporal discounting, charitable donations, assessments of uncertainty and so on. Moreover, some (e.g., Sokol-Hessner et al., 2009) have argued that ability to regulate may be related to more efficient decision-making behavior. One interesting question is therefore whether individual differences in the way we treat and regulate our emotions are predictive of more optimal choices and the better avoidance of detrimental behaviors.

To date, researchers have focused their attention primarily on the effect of negative emotions on decision making. However, positive emotions may also have a large impact on decision making, and future studies could productively examine these affective influences. Another intriguing finding is that, in some studies, augmented activity in the more "decision-related" brain areas has been reported. At least two studies have reported a baseline shift in dlPFC (Grecucci et al., 2013; Sokol-Hessner et al., 2013). The exact interpretation of this shift is a matter for future research studies, but one speculative account is that this may be involved in increased intentional control (Sokol-Hessner et al., 2013) or more flexibility in satisfying contextual demands (Grecucci, 2012; Grecucci, Giorgetta, Van't Wont, et al., 2013). One important point that remains unsolved is how emotion regulation strategies affect decisions. Do regulatory strategies alter decisions themselves, without impacting emotions? Or does emotion regulation influence decision-making processes by altering the emotion, which in turn alters decision making? Even if the latter seems to be the more plausible explanation, the two hypotheses have rarely been tested in the same experiment. Finally, recent studies of experimental psychopathology are providing important insights into how emotional dysregulation (excessive anxiety or depression, etc.) affects our decisions. These studies may provide further proof of the importance of regulatory mechanisms (and their failure) in decision making.

Overall, studying the processes and mechanisms involved in how emotions are regulated in concert with actual decision-making behavior offers an appealing template for future research. Demonstrating how regulation can actually impact both behavior and subsequent choice outcomes extends the current research topic in interesting directions and also has the potential to outline practical strategies that can be usefully employed in real-life situations.

References

Andrade, E. B., & Ariely, D. (2009). The enduring impact of transient emotions on decision-making. *Organizational Behavior and Human Decision Processes, 109*(1), 1–8.

Banks, S. J., Eddy, K. T., Angstadt, M., Nathan, P. J., & Phan, K. L. (2007). Amygdala–frontal

connectivity during emotion regulation. *Social Cognitive and Affective Neuroscience, 2*(4), 303–312.

Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition, 50*, 7–15.

Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science, 275*(5304), 1293–1295.

Bonini, N., Hadjichristidis, C., Mazzocco, K., Demattè, M. L., Zampini, M., Sbarbati, A., et al. (2011). *Pecunia olet*: The role of incidental disgust in the Ultimatum Game. *Emotion, 11*(4), 965–969.

Camerer, C. F. (2003). *Behavioral game theory.* New York: Russell Sage Foundation.

Clark, M. S., & Isen, A. M. (1982). Towards understanding the relationship between feeling states and social behavior. In A. H. Hastorf & A. M. Isen (Eds.), *Cognitive social psychology* (pp. 73–108). New York: Elsevier.

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus driven attention in the brain. *Nature Review Neuroscience, 3*, 201–215.

Dalgleish, T. (2004). The emotional brain. *Nature Review Neuroscience, 5*, 583–589.

Damasio, A. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: Putnam.

Delgado, M. R., Gillis, M., & Phelps, E. A. (2008). Regulating the expectation of reward via cognitive strategies. *Nature Neuroscience, 11*(8), 880–881.

Delgado, M. R., Nearing, K. I., LeDoux, J. E., & Phelps, E. A. (2008). Neural circuitry underlying the regulation of conditioned fear and its relation to extinction. *Neuron, 59*, 829–838.

Duncan, J., Emslie, H., Williams, P., Johnson, R., & Freer, C. (1996). Intelligence and the frontal lobe: The organization of goal-directed behavior. *Cognitive Psychology, 30*, 257–303.

Eippert, F., Veit, R., Weiskopf, N., Erb, M., Birbaumer, N., & Anders, S. (2007). Regulation of emotional responses elicited by threat-related stimuli. *Human Brain Mapping, 28*(5), 409–423.

Evans, J. St. B. T. (2009). How many dual-process theories do we need: One, two or many? In J. St. B. T. Evans & K. Frankish (Eds.), *In two minds: Dual processes and beyond* (pp. 31–54). New York: Oxford University Press.

Forgas, J. P. (1995). Mood and judgment: The affect infusion model (AIM). *Psychological Bulletin, 73*(117), 39–66.

Forgas, J. P. (2003). Affective influences on attitudes and judgments. In R. J. Davidson, K. J. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 596–618). New York: Oxford University Press.

Forgas, J. P., Bower, G. H., & Moylan, S. J. (1990). Praise or blame?: Affective influences on attributions for achievement. *Journal of Personality and Social Psychology, 59*, 809–818.

Grecucci, A. (2012). *Il Conflitto Epistemologico. Psicoanalisi e Neuroscienze dei Processi anticonoscitivi.* Francavilla, Italy: Edizioni Psiconline.

Grecucci, A., Giorgetta, C., Bonini, N., & Sanfey, A. (2013a). Living emotions, avoiding emotions: behavioral investigation of the regulation of socially driven emotions. *Frontiers in Psychology, 3*(616).

Grecucci, A., Giorgetta, C., Bonini, N., & Sanfey, A. (2013b). Reappraising social emotions: The role of inferior frontal gyrus, temporo-parietal junction and insula in interpersonal regulation. *Frontiers in Human Neuroscience*.

Grecucci, A., Giorgetta, C., Van't Wout, M., Bonini, N., & Sanfey, A. G. (2013). Reappraising the ultimatum: An fMRI study of emotion regulation and decision-making. *Cerebral Cortex, 23*(2), 399–410.

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*, 281–291.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*(2), 348–362.

Guth, W., Schmittberger, R., & Schwarz, B. (1982). An experimental analysis of ultimatum bargaining. *Journal of Economic and Behavioral Organization, 3*, 376–388.

Harlé, K., & Sanfey, A. G. (2007). Incidental sadness biases social economic decisions in the ultimatum game. *Emotion, 7*(4), 876–881.

Harlé, K. M., Chang, L. J., Van't Wout, M., & Sanfey, A. G. (2012). The neural mechanisms of affect infusion in social economic decision-

making: A mediating role of the anterior insula. *NeuroImage, 61*(1), 32–40.

Heilman, R. M., Crişan, L. G., Houser, D., Miclea, M., & Miu, A. C. (2010). Emotion regulation and decision-making under risk and uncertainty. *Emotion, 10*(2), 257–265.

Heinz, A. (2002). Dopaminergic dysfunction in alcoholism and schizophrenia—Psychopathological and behavioral correlates. *European Psychiatry, 17*, 9–16.

Hollerman, J. R., & Schultz, W. (1998). Dopamine neurons report an error in the temporal prediction of reward during learning. *Nature Neuroscience, 1*, 304–309.

Isen, A. M., & Daubman, K. A. (1984). The influence of affect on categorization. *Journal of Personality and Social Psychology, 47*, 1206–1217.

Kahneman, D., Knetsch, J. L., & Thaler, R. H. (1990). Experimental tests of the endowment effect and the Coase theorem. *Journal of Political Economy, 98*, 1325–1348.

Kahneman, D., & Tversky, A. (1979). Prospect theory: An analysis of decision under risk. *Econometrica, 47*, 263–291.

Knoch, D., Pascual-Leone, A., Meyer, K., Treyer, V., & Fehr, E. (2006). Diminishing reciprocal fairness by disrupting the right prefrontal cortex. *Science, 314*, 829–832.

Knutson, B., Taylor, J., Kaufman, M., Peterson, R., & Glover, G. (2005). Distributed neural representation of expected value. *Journal of Neuroscience, 25*, 4806–4812.

Koenigs, M., & Tranel, D. (2007). Irrational economic decision-making after ventromedial prefrontal damage: Evidence from the Ultimatum Game. *Journal of Neuroscience, 27*, 951–956.

Lejuez, C. W., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., Stuart, G. L., et al. (2002). Evaluation of a behavioral measure of risk taking: The Balloon Analogue Risk Task (BART). *Journal of Experimental Psychology: Applied, 8*, 75–84.

Lerner, J. S., Small, D. A., & Loewenstein, G. F. (2004). Heart strings and purse strings: Carryover effects of emotions on economic decisions. *Psychological Science, 15*(5), 337–341.

Martin, L. N., & Delgado, M. R. (2011). The influence of emotion regulation on decision-making under risk. *Journal of Cognitive Neuroscience, 23*(9), 2569–2581.

McClure, S. M., Berns, G. S., & Montague, P. R. (2003). Temporal prediction errors in a passive learning task activate human striatum. *Neuron, 38*, 339–346.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*, 167–202.

Mitchell, D. G. V. (2011). The nexus between decision-making and emotion regulation: A review of convergent neurocognitive substrates. *Behavioral Brain Research, 217*, 215–231.

Montague, P. R., Hyman, S. E., & Cohen, J. D. (2004). Computational roles for dopamine in behavioural control. *Nature, 431*, 760–767.

Moretti, L., Dragone, D., & di Pellegrino, G. (2009). Reward and social valuation deficits following ventromedial prefrontal damage. *Journal of Cognitive Neuroscience, 21*(1), 128–140.

Myerson, R. B. (1991). *Game theory: Analysis of conflict*. Cambridge, MA: Harvard University Press.

Ochsner, K. (2008). The social-emotional processing stream: Five core constructs and their translational potential for schizophrenia and beyond. *Biological Psychiatry, 64*(1), 48–61.

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14*, 1215–1229.

Ochsner, K. N., & Gross, J. J. (2008). Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. *Current Directions in Psychological Science, 17*, 153–158.

Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D., et al. (2004). For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *NeuroImage, 23*(2), 483–499.

Pfister, H.-R., & Böhm, G. (2008). The multiplicity of emotions: A framework of emotional functions in decision making. *Judgment and Decision Making, 3*(1), 5–17.

Phillips, M. L., Drevets, W. C., Rauch, S. L., & Lane, R. (2003). Neurobiology of emotion perception: II. Implications for major psychiatric disorders. *Biological Psychiatry, 54*(5), 515–528.

Phillips, M. L., Young, A. W., Senior, C., Brammer, M., Andrew, C., Calder, A. J., et al. (1997). A specific neural substrate for perceiving facial expressions of disgust. *Nature, 389*, 495–498.

Pillutla, M. M., & Murnighan, J. K. (1996). Unfairness, anger, and spite: Emotional rejec-

tions of ultimatum offers. *Organization Behavior and Human Decision Process, 68*, 208–224.

Robinson, T. E., & Berridge, K. C. (2003). Addiction. *Annual Review of Psychology, 54*, 25–53.

Sanfey, A. G. (2007). Social decision-making: Insights from game theory and neuroscience. *Science, 318*(5850), 598–602.

Sanfey, A. G., & Chang, L. J. (2008). Multiple systems in decision making. *Annals of the New York Academy of Sciences, 1128*, 53–62.

Sanfey, A. G., & Dorris, M. (2009). Games in humans and non-human primates: Scanners to single units. In P. W. Glimcher, C. F. Camerer, E. Fehr, & R. A. Poldrack (Eds.), *Neuroeconomics* (pp. 63–80). London: Academic Press.

Sanfey, A. G., Rilling, J. K., Aronson, J. A., Nystrom, L. E., & Cohen, J. D. (2003). The neural basis of economic decision-making in the ultimatum game. *Science, 300*, 1755–1758.

Schultz, W. (2009). Midbrain dopamine neurons: A retina of the reward system? In P. W. Glimcher, C. F. Camerer, E. Fehr, & R. A. Poldrack (Eds.), *Neuroeconomics* (p. 323). London: Academic Press.

Slovic, P., Finucane, M. L., Peters, E., & MacGregor, D. G. (2004). Risk as analysis and risk as feelings: Some thoughts about affect, reason, risk, and rationality. *Risk Analysis, 24*(2), 311–322.

Smith, E. E., & Jonides, J. (1999). Storage and executive processes in the frontal lobes. *Science, 283*(5408), 1657–1661.

Sokol-Hessner, P., Camerer, C. F., & Phelps, E. A. (2013). Emotion regulation reduces loss aversion and decreases amygdala responses to losses. *Social Cognitive and Affective Neuroscience, 8*(3), 341–350.

Sokol-Hessner, P., Hsu, M., Curley, N. G., Delgado, M. R., Camerer, C. F., & Phelps, E. A. (2009). Thinking like a trader selectively reduces individuals' loss aversion. *Proceeding of the National Academy of Sciences, 106*(13), 5035–5040.

Staudinger, M. R., Erk, S., Abler, B., & Walter, H. (2009). Cognitive reappraisal modulates expected value and prediction error encoding in the ventral striatum. *NeuroImage, 47*, 713–721.

Staudinger, M. R., Erk, S., & Walter, H. (2011). Dorsolateral prefrontal cortex modulates striatal reward encoding during reappraisal of reward anticipation. *Cerebral Cortex, 21*(11), 2578–2588.

Stuss, D. T., & Knight, R. T. (2002). *Principles of frontal lobe function*. New York: Oxford University Press.

Sutton, R. (1988). Learning to predict by the methods of temporal differences. *Machine Learning, 3*, 9–44.

Tversky, A., & Kahneman, D. (1992). Advances in prospect theory: Cumulative representation of uncertainty. *Journal of Risk and Uncertainty, 5*, 297–323.

Van't Wout, M., Chang, L. J., & Sanfey, A. G. (2010). The influence of emotion regulation on social interactive decision-making. *Emotion, 10*(6), 815–821.

Van't Wout, M., Kahn, R. S., Sanfey, A. G., & Aleman, A. (2005). Repetitive transcranial magnetic stimulation over the right dorsolateral prefrontal cortex affects strategic decision-making. *NeuroReport, 16*, 1849–1852.

Van't Wout, M., Kahn, R. S., Sanfey, A. G., & Aleman, A. (2006). Affective state and decision-making in the ultimatum game. *Experimental Brain Research, 169*(4), 564–568.

Volkow, N. D., & Wise, R. A. (2005). How can drug addiction help us understand obesity? *Nature Neuroscience, 8*, 555–560.

Winkielman, P., Berridge, K. C., & Wilbarger, J. L. (2005). Unconscious affective reactions to masked happy versus angry faces influence consumption behavior and judgments of value. *Personality and Social Psychology Bulletin, 31*(1), 121–135.

Wrase, J., Schlagenhauf, F., Kienast, T., Wustenberg, T., Bermpohl, F., Kahnt, T., et al. (2007). Dysfunction of reward processing correlates with alcohol craving in detoxified alcoholics. *NeuroImage, 35*, 787–794.

Xiao, E., & Houser, D. (2005). Emotion expression in human punishment behavior. *Proceedings of the National Academy of Sciences, 102*, 7398–7401.

Zajonc, R. B. (2000). Feelings and thinking: Closing the debate over the independence of affect. In J. P. Forgas (Ed.), *Feeling and thinking: The role of affect in social cognition* (pp. 31–58). New York: Cambridge University Press.

PART IV

DEVELOPMENTAL CONSIDERATIONS

CHAPTER 10

Self-Regulation, Effortful Control, and Their Socioemotional Correlates

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In this chapter, we focus on the nature and development of temperamentally based effortful control (EC) processes and the role of effortful self-regulation in children's socioemotional development. First we consider definitional and conceptual issues. Then we briefly summarize the normative development of self-regulation (including EC), followed by review of research illustrating the importance of individual differences in children's self-regulatory skills (including effortful control) for their socioemotional development. We conclude by discussing a few current conceptual and methodological issues and gaps in the research.

Conceptual Issues

Our discussion of EC must be considered in the broader context of conceptions of children's self-regulation. In the work with children, there has been a strong focus on self-regulatory processes involved in the modulation of emotion—an aspect of regulation labeled as *emotion regulation*. Definitions of this construct vary considerably (Gross, this volume). Our definition of *emotion-related self-regulation* includes many of the elements of others' definitions but is broader in some respects and narrower in others. For us, it refers to processes used to manage and change whether, when, and how (e.g., how intensely) one experiences emotions and emotion-related motivational and physiological states, as well as how emotions are expressed behaviorally. Thus, it includes processes used to change one's own emotional state, to prevent or initiate emotional responding (e.g., by selecting or changing situations), to modify the significance of an event for the self, and to modulate the behavioral expression of emotion (e.g., through verbal or nonverbal cues). Our definition is consistent with Gross's (this volume) model, in which emotion regulation includes situation selection, situation modification, attentional deployment, cognitive change, and response modulation.

We have used the term *emotion-related self-regulation* because many of the processes frequently involved in emotional self-regulation (see below) are also used for the self-regulation in the absence of emotion. Although we fully acknowledge that emotional control/regulation often is externally imposed, especially early in life, we believe it is useful to differentiate between inter-

nally generated self-regulatory processes and those processes that the child does not execute (e.g., parental soothing of an infant; Eisenberg et al., 2004; Eisenberg, Spinrad, & Eggum, 2010). Thus, we tend to use the term *self-regulation* only for internally generated regulatory processes (although in the past we used the term *regulation*), similar to Gross's (this volume) distinction between intrinsic and extrinsic regulatory processes.

There are other variations in how the term *regulation* or even *self-regulation* is used. Some people use it to refer to a host of processes that have a controlling effect on cognition, attention, or behavior. As we discuss later, we believe that it is useful to make a distinction between more and less volitional processes involved in regulation/control. Although it is clear that many nonvolitional processes have important modulating (even, in a sense, regulating) effects on attention, cognition, behavior, and physiological responding, it would be useful to differentiate the two types of processes semantically. A term such as *potentially volitional self-regulatory processes* would be more precise than *self-regulation* in referring to volitional processes; however, such terminology is quite awkward. We ask readers to focus less on the use of the word *self-regulation* than on the distinctions we address.

Because it is extremely difficult to differentiate emotion from its regulation, we believe it is useful to focus on the processes used to manage attention, emotion, and associated behavior rather than on the amount of emotion experienced or expressed. In this respect, we find EC to be a useful construct.

Effortful Control

EC, the regulatory component of temperament, is defined as "the efficiency of executive attention, including the ability to inhibit a dominant response and/or to activate a subdominant response, to plan, and to detect errors" (Rothbart & Bates, 2006, p. 129). It involves the ability to deploy attention willfully (often called *attention focusing* and *shifting*, and including cognitive distraction) and to inhibit or activate behavior willfully (inhibitory control and activational control, respectively). Some executive functioning (EF) skills—especially effortful deployment of attention, integration of information to which one attends, and planning—are involved in EC; thus, EF and EC are partially overlapping constructs. In our view, EC and, as a result, aspects of EF, include an array of processes or capabilities that can be used to manage emotion and behavior; however, EC is not completely analogous to emotional self-regulation because EC can be used for other purposes (e.g., to focus on schoolwork). Moreover, self-regulation capacities likely include more than just EC; for example, self-regulation may include the motivation to utilize EC/EF in the service of goals (Eisenberg & Zhou, in press). We view the skills inherent in EC as building blocks for the development of self-regulation, emotional and otherwise, across the lifespan, and thus, as the core of internally based, volitional self-regulation. Therefore, despite some distinctions between EC and self-regulation, the constructs are overlapping and we tend to use the terms interchangeably in this chapter.

The fact that EC is effortful or willful does not mean that the individual is always aware that he or she is modulating emotion or behavior. Some aspects of EC, especially after rehearsal and practice, may often be automatic and executed without much conscious awareness in contexts with relevant trigger cues (Mischel & Ayduk, 2011); however, if needed, the individual is able to shift into a more volitional mode of functioning. Effortful regulatory processes are distinguished from less voluntary reactive processes (see below) by the ease with which they can be volitionally controlled.

Any particular form of self-regulation (or EC) is not necessarily good or bad in terms of its consequences. Nonetheless, we believe that effortful self-regulatory processes are more likely than some less volitional aspects of control (see below) to result in adaptive outcomes in normative contexts, or at least in desired goals, be they actually adaptive or not. This is because effortful capacities can be applied at will and adapted flexibly to the demands of specific contexts. Of course, individuals may also use EC in a manner that is not adaptive. For example, EC can be used to achieve socially inappropriate goals, such as a youth planning and carrying out a series of well-regulated actions to humiliate a peer or to steal items to impress friends. Moreover, an effortful mode of regulation

could be adaptive in the short run but not in the long run, or vice versa. In part, the adaptiveness of EC depends on the goals that an individual is striving to achieve and the social context.

Control and Regulation: Is Control Always Regulation?

Like a number of other investigators (e.g., Block & Block, 1980), we have argued that well-regulated people are neither overly controlled nor undercontrolled; rather, they can respond flexibly to varying demands with a range of responses, depending on the circumstances. Optimally self-regulated children can effortfully initiate or inhibit behaviors when appropriate or when required to achieve goals, but they can also be spontaneous (uncontrolled) when control is not needed. Thus, their regulation is flexible and generally (although not always) adaptive.

Because of the importance of both willfulness and flexibility in EC, it is useful to try to differentiate EC from less voluntary over- or undercontrolled processes. Some control is nearly always automatic and nonvolitional, and often it is emotionally based; such control is likely less flexible and often is less adaptive than volitional self-regulation. An example is children who have been labeled "behaviorally inhibited." They tend to be wary and overly constrained in novel or stressful situations and have difficulty modulating (e.g., relaxing) their inhibition (Kagan & Fox, 2006). Although they may appear to be self-regulated, their inhibition or constraint is relatively involuntary or so automatic that it is difficult for them to modulate effortfully (Eisenberg & Morris, 2002). In addition, the impulse to approach people or inanimate objects in the environment (sometimes called *surgency*; Rothbart & Bates, 2006) often may be relatively involuntary. For example, individuals may be "pulled" toward rewarding or positive situations or stimuli, and this pull may be nonvolitional; such people generally are viewed as "impulsive." We have labeled such overly inhibited and impulsive behavior as two aspects of *reactive control*—reactive overcontrol and undercontrol—based on the distinction by Rothbart and Bates (2006) between effortful and reactive temperamental processes. The constructs of reactive undercontrol and overcontrol are somewhat similar to what the Block and Block (1980) labeled as the extremes of *ego control* (i.e., ego undercontrol and overcontrol), which is defined as the "threshold or operating characteristic of an individual with regard to the expression or containment of impulses, feelings, and desires" (p. 43). In the Blocks' model, ego resiliency provides control and modulation of impulses (i.e., ego control).

We consider EC, but not reactive control, to be part of volitional self-regulation. As noted previously, we certainly acknowledge that reactive control processes influence and even have controlling or "regulatory" effects on individuals' functioning, but we prefer not to equate these effects with volitional self-regulation (see Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001, for a similar distinction when defining coping; also see Carver, 2005, for a review of similar perspectives, including dual-processing models).

Rothbart and colleagues (e.g., Derryberry & Rothbart, 1997) linked what we have labeled reactive overcontrol and undercontrol processes to reactive emotional processes—fear versus desire, hope, and relief, respectively—and their associated motivational systems, defensive and appetitive, respectively. Although we agree that these associations between behavioral inhibition or impulsivity and emotion/motivation exist, we wish to differentiate between emotional reactivity and the aspects of behavior relevant to control that typically are associated with emotion. It is quite possible that children who display behavioral inhibition (reactive overcontrol) do not experience fear or anxiety every time they display overly inhibited behavior. Such behavior, although probably originally based on fear or a reaction to novelty (Kagan & Fox, 2006), seems to become a habitual style of response to novel or potentially stressful contexts. Moreover, highly inhibited children often look controlled (restrained) in their behavior but tend to be prone to fear and anxiety; thus, control of overt behavior is not the same as control in regard to emotional reactivity (although both are elements of temperamental reactivity). Similarly, impulsive behavior that may be linked with both desire/positive affect (e.g., Rothbart, Ahadi, Hershey, & Fisher, 2001) and anger in some contexts

might, in other contexts, not be linked to any clear emotion. Thus, emotional reactivity of a particular sort is not strictly paired with impulsivity, and it is worthwhile to differentiate between impulsivity and emotional reactivity, and among associated motivations in specific interactions. In support of this argument, in factor analyses, dispositional negative emotionality tends to load on one factor, whereas reactive processes that involve avoidance or approach behavior (e.g., shyness, impulsivity) tend to load on a separate surgency factor (Rothbart et al., 2001).

A disadvantage to invoking the distinction between effortful self-regulation and reactive control is that it is difficult to categorize some aspects of regulation/control. For example, it is difficult to know whether the child who is constrained in a new context is self-regulated (i.e., voluntarily constrained) or overly controlled (behaviorally inhibited). In our view, a major challenge for the field is differentiating between these two contributors to constrained behavior.

Although impulsivity and EC are fairly consistently negatively related (Aksan & Kochanska, 2004; Valiente et al., 2003), the two constructs have been found to load on different latent constructs (Eisenberg et al., 2004, 2013; Valiente et al., 2003). The relation between EC and reactive overcontrol is less consistent (e.g., Aksan & Kochanska, 2004; Spinrad et al., 2007), and there is a need for research testing the relation between reactive over- and undercontrol.

A comparison with Ursache, Blair, and Raver's (2012) definition helps to highlight some of the subtle differences in definitions (some other definitions differ from ours in numerous, more obvious ways, including some of those mentioned earlier). They defined self-regulation as

> the primarily volitional management of arousal or activity in attention, emotion, or stress response systems in ways that facilitate the use of EF abilities in the service of goal-directed actions. . . . EF is the top-down or volitional component of self-regulation. . . .This volitional component, however, is reciprocally related to and dependent on bottom-up, less volitional, and more automatic regulation of responses to the environment through attention, emotion, and stress response systems. (p. 123)

We would argue that some responses that are relatively automatic usually can be volitional if necessary, so the critical distinction is whether a capacity can easily become volitional if needed for adaptation, not whether it can be automatic. In addition, Ursache et al. seem to include what we have labeled as reactive control in the construct of regulation ("the bottom-up, less volitional" aspects). Although we prefer to confine the term *self-regulation* to the aspects that can be volitional when needed, as a semantic way to differentiate between volitional and reactive control processes, we agree that both processes occur. We also agree that the relation between more effortful and less volitional processes is bidirectional; moreover, reactive over- and undercontrol systems likely affect one another and are affected by emotions (Derryberry & Rothbart, 1997). Not completely in accord with Ursache et al. (2012), we (Eisenberg & Zhou, in press) suggested that aspects of EF related to inhibition (e.g., in regard to thoughts or behavior and the ability to shift attention) overlap with the construct of self-regulation (as well as the construct of EC), whereas other parts of EF, such as working memory, facilitate self-regulation but are separate capacities from both self-regulation and EC.

In summary, numerous conceptual issues are unresolved regarding the definition of self-regulation and the relations among various processes that have controlling/regulating effects. We believe that more rather than less conceptual differentiation is useful, even if in reality the various processes are interconnected in complex and intricate ways.

The Normative Development of Self-Regulation/EC

Because EC is viewed as providing the building blocks for self-regulation, its development is of importance to many aspects of children's functioning. Children begin to regulate their own emotions and emotion-related behavior in the first few years of life, and there are improvements in regulation skills across childhood and adolescence (see Eisenberg, Spinrad, et al., 2010). Even young children have been found to modulate their distress using a variety of methods. For example, by 6 months of age, infants some-

times reduce their own distress in response to novelty by looking away from the novel object and by using self-soothing strategies (Crockenberg & Leerkes, 2004). Such behaviors appear to be effective in reducing arousal in the first year of life because they are associated with lower negativity in frustrating situations (Stifter & Braungart, 1995). Stifter and Braungart found that self-soothing was the most preferred regulatory strategy at both 5 and 20 months of age. In contrast, Mangelsdorf, Shapiro, and Marzolf (1995) found that (1) 6-month-old infants tended to use gaze aversion as their primary regulatory strategy, (2) 12-month-olds engaged in more self-soothing (e.g., thumb sucking and hair twirling) than did 18-month-olds, and (3) 12- and 18-month-old toddlers used more behavioral avoidance and self-distracting strategies than did 6-month-olds.

There appears to be a decline in the use of self-soothing between 24 and 48 months, coupled with the emergence of new and more complex use of objects and interactions to regulate emotional state (see Diener & Mangelsdorf, 1999, for a review). By 24 months of age, self-distraction may be the most common and successful regulatory strategy in fearful and frustrative situations (Grolnick, Bridges, & Connell, 1996). Advances in cognitive, sociocognitive, motor, and language development that occur between 2 and 5 years of age contribute to the emergence of more sophisticated, diverse, and successful models of self-regulation and EF skills (Garon, Bryson, & Smith, 2008), such that many children are relatively skilled at managing impulses by age 4 or 5 (Mischel & Ayduk, 2011; Posner & Rothbart, 2000; Rothbart, 2011).

Researchers who have focused on the development of EC processes such as attention shifting and attention focusing or the ability to inhibit behavior voluntarily (rather than as specific reactions to distress or frustration) have also noted developmental changes in their use. Eight- to 10-month-olds demonstrate some capacity to focus their attention (Kochanska, Coy, Tjebkes, & Husarek, 1998), and voluntary control of attention increases somewhat between 9 and 18 months of age (Ruff & Rothbart, 1996). Around 12 months of age, infants develop the ability to inhibit predominant responses. For example, Diamond (1991) found that infants were able to inhibit predominant response tendencies when reaching for objects (e.g., move around an object), an ability believed to involve the execution of intentional behavior, planning, and the resistance of more automatic or reactive action tendencies.

According to Posner and Rothbart (2000), a transition in the development of executive attention and the effortful inhibition of behavior can be seen around 30 months of age. Much of the relevant work involved the use of a Stroop-like task that required toddlers to switch attention and inhibit behavior. Children show significant improvement in performance on such a task between 24 and 30 months of age and often perform with high accuracy by 36 to 38 months of age, and this ability has been positively related to adults' ratings of EC (Rothbart & Bates, 2006). In addition, with the maturation of attentional mechanisms, the ability to inhibit motor behavior effortfully greatly improves between 22 and 44 months (Kochanska, Murray, & Harlan, 2000; Rothbart & Bates, 2006) and is fairly good by age 4 (Posner & Rothbart, 2000). Willoughby, Wirth, Blair, and Family Life Project Investigators (2012) found substantial improvements in a latent factor of EF abilities in children between ages 3 and 5 years, with the average child improving more than one standard deviation in EF skills over any 12-month period. Moreover, there appears to be a further increase in the use of internal mental or cognitive regulatory strategies in the school years. Nonetheless, the capacity for EC continues to improve in adolescence and may even continue to develop at a slower pace into adulthood (Murphy, Eisenberg, Fabes, Shepard, & Guthrie, 1999; Williams, Ponesse, Schachar, Logan, & Tannock, 1999; also see Eisenberg, Spinrad, et al., 2010).

The Relation of EC to Socioemotional Development

Children who differ in their self-regulatory capacities are expected to differ in aspects of socioemotional functioning involving self-regulation. Based on the distinction between reactive control and EC, Eisen-

berg and Morris (2002) constructed a heuristic model including descriptions of three styles of control—overcontrolled, undercontrolled, and optimally controlled—to generate predictions regarding the relations of various aspects of EC and reactive control to children's socioemotional adjustment and development. In this heuristic model, the authors described different constellations of self-regulation/reactive control expected to cluster together for some children and the expected socioemotional correlates of each constellation.

In the model, overcontrolled individuals are those high in involuntary reactive control (e.g., high in behavioral inhibition) and low in reactive undercontrol (impulsivity), average in the ability to inhibit behavior effortfully as needed (inhibitory control), low in effortful attentional regulation (e.g., the abilities to shift and focus attention effortfully), and low in the ability to activate behavior effortfully as needed (i.e., effortful activational control). Volitional shifting or focusing attention is expected to be useful in reducing the negative emotions, such as fear, associated with highly inhibited behavior. Individuals with this constellation are expected to be prone to internalizing problems (e.g., depression, anxiety, social withdrawal) and relatively low in social competence, especially if they are also predisposed to experience negative emotions. These children might also lack the ability to be relaxed, socially interactive, and spontaneous in all but very familiar settings, which also is likely to undermine their social attractiveness and peer status.

In contrast, undercontrolled individuals are those high in reactive approach tendencies (i.e., impulsive); low in reactive overcontrol; and low in all types of EC, including attentional, inhibitory, and activation control. People with this style of control are predicted to be relatively low in social competence and prone to externalizing behavior problems such as aggression, defiance, and antisocial behaviors (e.g., delinquency). These children would also be expected to be low in empathy, prosocial behavior, and moral development, because of deficits in social cognition and regulated behavior.

Finally, optimally regulated individuals—those who are most flexible and appropriate in their regulation—are those fairly high in all modes of EC and, in regard to reactive control, are neither overcontrolled (inhibited) nor undercontrolled (impulsive). These individuals are expected to be well adjusted, socially competent (including moral and prosocial), and resilient when faced with stress because they typically regulate their behavior in a goal-directed manner but can also be spontaneous and unconstrained.

Consistent with the heuristic model, EC and self-regulation more generally have been related in predictable ways to a variety of children's developmental outcomes. Next we provide a selective summary of studies on the relation of EC and/or other measures of self-regulation to some positive and negative outcomes expected to be affected by EC and/or reactive control, including social competence, social cognition, (mal)adjustment, school-related outcomes, internalization of parental rules/demands and guilt, empathy/sympathy, and prosocial behavior.

Social Competence

In a number of studies with preschoolers, children, and adolescents, EC has been associated with relatively high levels of social competence (see Eisenberg, Smith, & Spinrad, 2011, for a review). Children's abilities to modulate their attentional resources likely lead to optimal levels of emotional arousal in stressful contexts and, thus, accurate information processing linked to understanding the causes of emotion, effective planning, and action suited to the situation. Moreover, the abilities to inhibit and activate behavior effortfully allow children to implement or inhibit behaviors as is adaptive in a given context, hence facilitating socially appropriate responses. Consistent with these arguments, EC (assessed with EF and/or delay of gratification tasks) in the preschool years has been found to predict parent-reported social competence 6 months later (Lengua, Honorado, & Bush, 2007), as well as socioemotional competencies in adolescence and adulthood (Mischel & Ayduk, 2011). Eisenberg and colleagues (2011) have found support for the positive relation between EC and social competence in multiple longitudinal samples, and this relation has also been observed in other cultures (e.g., Hofer, Eisenberg, & Reiser, 2010; Zhou, Eisenberg, Wang, & Reiser, 2004).

EC (or aspects thereof) frequently has been examined as a mediator of the relation of parenting to social competence. For example, Mintz, Hamre, and Hatfield (2011) found that mothers' reports of 54-month-olds' inhibitory control, but not attentional focusing, partially mediated the relation between early maternal sensitivity (prompt and appropriate responses to the child, nonintrusive, positive regard for child) and observed and teacher-rated social and relational competence, as well as social and relational competence in first grade. Similarly, EC has been found to mediate the relation between maternal support and children's social competence concurrently (at 18 and 30 months), albeit not over time (Spinrad et al., 2007). In addition, other competencies appear to mediate the relation of self-regulation to social competence. For example, the positive relation between EC and peer popularity appears to be mediated by *ego-resiliency* (personality resiliency; the ability to cope with and rebound from stress), both in longitudinal work with American 6- to 8-year-olds (Spinrad et al., 2006) and concurrently with French adolescents (Hofer et al., 2010). Children with high EC are expected to be able to bounce back easily in new or stressful situations, and this resiliency is expected to improve their social competence.

Social Cognition

Children who can avoid emotional overarousal and focus their attention are more likely than less regulated children to focus on relevant information about emotions and the environment in social interactions and, hence, develop a better understanding of emotions. In support of these ideas, preschoolers' self-regulation has predicted their understanding of emotion 2 years later (Schultz, Izard, Ackerman, & Youngstrom, 2001), and emotion understanding mediates the relation of self-regulation to adaptive social behavior (Izard, Schultz, Fine, Youngstrom, & Ackerman, 1999). It is also likely that emotion understanding fosters, as well as stems from, emotion-related regulation. Denham and Burton (2003) suggested that emotion understanding gives children labels for their internal feelings, which can then be made conscious. Such conscious emotional awareness allows children to immediately attach feelings to events, which can then facilitate successful and appropriate regulation. Researchers have found that children who are able to understand and communicate about emotions, and who know how to manage emotions, are better able to regulate themselves (Denham & Burton, 2003).

With the development of EF, children also improve on theory of mind (ToM) tasks, which involve an understanding of others' internal mental representations. Researchers have identified inhibitory control as especially relevant to the development of ToM because it enables children to suppress irrelevant perspectives (Carlson, Moses, & Claxton, 2004). In a number of studies using a variety of observed and reported measures, inhibitory control has been positively related to ToM skills in preschool and kindergarten children (Blair & Razza, 2007; Carlson et al., 2004). Moreover, inhibitory control appears to precede the development of ToM in the preschool years (e.g., Flynn, 2007). In brief, self-regulated children appear to have an advantage in regard to emotional understanding and ToM skills.

Maladjustment

Children with adjustment problems tend to have difficulties managing and controlling their attention, behavior, and emotions in a willful and flexible manner. These difficulties can result in processing information and modulating emotions and behaviors in ways that hinder adaptive functioning (Eisenberg, Spinrad, et al., 2010). In addition, because children with high EC are probably better able to avoid negative interactions and modulate negative emotions such as sadness and anger, it is not surprising that low EC has been related to maladjustment (for reviews, see Eisenberg et al., 2011; Eisenberg, Spinrad, et al., 2010).

Consistent with our heuristic model, many investigators have found negative relations between EC and externalizing problems in preschoolers, elementary schoolchildren, and adolescents (Eisenberg, Spinrad, et al., 2010). For example, Kochanska and Knaack (2003) reported negative associations between EC (assessed with a battery of behavioral measures) and externalizing problems, concurrently and across time,

in young children (Kochanska & Knaack, 2003). In multiple longitudinal samples, Eisenberg and colleagues have found negative relations of teacher-reported, parent-reported, and observed measures of EC with children's externalizing behaviors (Eisenberg et al., 2011; Eisenberg, Spinrad, et al., 2010). For example, Eisenberg et al. (2009) found that change in at-risk elementary schoolchildren's externalizing over the 4 years—whether pure externalizing problems or co-occurring with internalizing problems—was related to change in EC over the same period. Such associations appear to continue throughout adolescence, including in European samples (Hofer et al., 2010). For example, Bakker, Ormel, Verhulst, and Oldehinkel (2011) found that parent-reported EC at age 13½ appeared to buffer Dutch adolescents against externalizing problems at age 16 in families with high adversity.

EC also appears to act as a mediator or moderator when predicting maladjustment (e.g., Hofer et al., 2010; Spinrad et al., 2007). For example, Hardaway, Wilson, Shaw, and Dishion (2012) found that inhibitory control at age 4 mediated the positive and negative relations, respectively, between chaos in the home and positive parental behavior at age 3 to externalizing behaviors at age 5½. Moreover, EC appears to reduce the positive relation of inconsistent discipline and physical punishment to 8- to 12-year-olds' externalizing problems (Lengua, 2008) and the relation of adolescents' proneness to frustration to externalizing problems (Oldehinkel, Hartman, Ferdinand, Verhulst, & Ormel, 2007).

Theoretically, as delineated in the aforementioned model, reactive control and low EC are related to internalizing problems: Children with internalizing problems are often overcontrolled and struggle with the flexible control of their emotions, cognition, attention, and behaviors. Although the literature is not highly consistent (Eisenberg, Spinrad, et al., 2010), a number of studies support a negative relation between EC and internalizing problems. For example, mothers' reports of preschoolers' attention focusing and inhibitory control have been modestly, negatively related with internalizing problems at 5½ years old, even when items that overlapped between the constructs of self-regulation and internalizing problems were removed from the scales (Lemery, Essex, & Smider, 2002). Similarly, negative relations between EC and depressive symptoms have been reported for 11- to 18-year-olds (Verstraeten, Vasey, Raes, & Bijttebier, 2008). Moreover, EC seems to moderate the relation between affectivity and internalizing problems. In one study, adolescents' low positive and high negative affectivity predicted more depressive symptoms for youth with low EC (Verstraeten et al., 2008); in another study, EC buffered the relation of fearfulness to adolescents' internalizing problems (Oldehinkel et al., 2007).

School-Related Outcomes

The role of EC in school-related outcomes has aroused increasing interest (Eisenberg, Valiente, & Eggum, 2010; Ursache et al., 2012), and both direct and indirect relations between EC and school-related outcomes have been predicted. Consistent with our heuristic model, because children high in EC are expected to manage their attention, behavior, and emotion flexibly, they are predicted to have an advantage when learning. Moreover, the effects of EC on academic outcomes are hypothesized to be mediated by children's adjustment, quality relationships with peers and teachers, and school engagement (Eisenberg, Valiente, et al., 2010).

In fact, EC predicts a variety of school-related outcomes, both academic and social, concurrently and longitudinally (Allan & Lonigan, 2011; Eisenberg, Valiente, et al., 2010; Ursache et al., 2012). In recent work, mediators of these relations have been examined. For example, in a 1-year longitudinal study with 3- to five-year-olds, Silva et al. (2011) found that a high-quality teacher–child relationship midyear (i.e., low conflict and high closeness) mediated the positive relation between high EC measured early in the year and positive school attitudes measured at the end of the year with reports of school liking and avoidance. In another study with multiple reporters, high social competence and low externalizing problems at ages 6–10 fully mediated the positive relation between high EC at ages 4–8 and high academic achievement at ages 10–14 (Valiente et al., 2011). In brief, optimally regulated children tend to have better school

outcomes, and these relations appear to be both direct and indirect.

Internalization of Parental Rules/ Demands and Guilt

EC and related measures of emotion-related regulation or control have also been linked to a variety of measures of moral development, including compliance, internalization of rules, and conscience. *Committed compliance*—when children appear to comply willingly and even enthusiastically with adults' expectations/requests—is thought to reflect children's rudimentary internalization of their mothers' commands. In addition, when toddlers comply with their mothers' rules when left alone, they are believed to have internalized the rules (Kochanska, Coy, & Murray, 2001).

Toddlers who exhibit more committed compliance tend to have higher EC than their less compliant peers (e.g., Kochanska et al., 2001). In addition, EC at 30 and 42 months predicts higher committed compliance 1 year later, even after researchers control for earlier levels of committed compliance (although these findings did not hold in models accounting for time-invariant covariates; Spinrad et al., 2012). Infants' attention regulation (components of EC) predicts later committed compliance (Hill & Braungart-Rieker, 2002; Kochanska et al., 1998). In addition, Kochanksa and Knaack (2003) have found a positive relation, often across time, between EC and the development of conscience (i.e., moral self, refrains from cheating on a task, and moral reasoning) during the toddler, preschool, and early school years (Kochanska & Knaack, 2003).

There are few data on the relation between conscience and EC in schoolchildren. However, parent reports of children's EC have been related to reports of their 7-year-olds' guilt (Rothbart, Ahadi, & Hershey, 1994). More research is needed to clarify the relation of EC to conscience and other aspects of moral development in older elementary school-age children and adolescents.

Empathy/Sympathy and Prosocial Behavior

Eisenberg and Fabes (1992) hypothesized that individuals high in effortful emotion-related self-regulation tend to experience sympathy (an other-oriented response to another's emotion or condition) rather than personal distress (i.e., a self-focused, aversive response to another's emotional state or condition). Personal distress is believed to be associated with empathic overarousal (possibly due to underregulation), which in turn can lead to a self-focus and self-concerned behavioral response (rather than concern for others; see Eisenberg, Wentzel, & Harris, 1998). Consistent with this premise, Guthrie et al. (1997) found that children rated high on EC exhibited greater facial sadness (but not distress)—presumed to reflect sympathy/empathy—during an empathy-inducing film than did less regulated peers. Children's postfilm reports of sadness and sympathy during the film were also positively related to parents' ratings of EC. Conversely, children low in parent-rated EC were prone to experience personal distress (e.g., anxiety and tension) during the film.

Children's EC (or aspects thereof) also has been positively related to self- or other-report measures of dispositional empathy/sympathy in children (Eisenberg et al., 1998; Panfile & Laible, 2012; Rothbart et al., 1994; also see Eisenberg, Fabes, & Spinrad, 2006). In an 8-year longitudinal study, relatively high levels of EC and growth in EC were related to high sympathy, although this pattern was somewhat stronger for boys than for girls (Eisenberg et al., 2007). Similar concurrent relations have been found in studies in which adults reported on their own sympathy and regulation, although sometimes the association was not significant until the effects of individual differences in negative emotionality were controlled (see Eisenberg et al., 1998). In addition, EC has been negatively related to children's and adults' reports of personal distress (Bandstra, Chambers, McGrath, & Moore, 2011; see Eisenberg et al., 1998, 2006).

Consistent with the empirical relation between EC and sympathy, adults' ratings of elementary school children's effortful attentional control and/or a behavioral measure of persistence have been correlated with peers' ratings (e.g., Eisenberg et al., 1997) or teachers' ratings (Diener & Kim, 2004) of prosocial behavior. Thus, people who are skilled at regulating their emotion and behavior are not only more likely to feel con-

cern for others but also they are relatively likely to help them.

Conclusions and Future Directions

The study of emotion regulation and related processes has progressed tremendously in the last decade. Yet definitional and methodological issues need to be addressed, and there are gaps in the literature, as well as new directions to exploit.

Terminology

Currently, there is considerable variation in what behavioral scientists mean when they use words such as *regulation, self-regulation*, and *emotion regulation*. The term *regulation* is used so broadly by different investigators that it is often hard to understand what is meant. Even though there may be somewhat of a continuum, we suggest that researchers clearly distinguish volitional self-regulatory processes (e.g., EC) from other, less volitional processes that also "regulate" behavior (e.g., reactive control). It is also important for researchers to distinguish between self-regulation and external regulation.

Distinguishing between EC and EF

Despite the fact that EF and EC typically have been examined in two separate literatures, researchers have begun to recognize that there is considerable overlap between these constructs (Eisenberg & Zhou, in press). The integration of these two bodies of knowledge has great potential to be mutually informative. For example, considerable attention has been given to the factor structure of EF (Garon et al., 2008), whereas similar work on EC is only now beginning (e.g., Allan & Lonigan, 2011; Sulik et al., 2010). The conceptualization of EF as comprising interrelated but independent abilities—working memory, response inhibition, and set shifting—has led to the understanding that task performance may be differentially related to these abilities and consequently to a more fine-grained analysis of what individual tasks are measuring (Best & Miller, 2010). For example, working memory, which is considered an important aspect of EF but not EC, has been examined as a contributor to performance on EF tasks. In contrast, working memory has generally not been considered a contributor to performance on measures of EC, even though it likely plays an important role for very young children and in tasks with more complex working memory demands. Finally, researchers studying EF have made the distinction between hot (i.e., emotional, for example, in delay tasks) and cold (i.e., cognitive, for example, in typical EF measures such as flanker or Stroop tasks) aspects of EF, which may have important implications for emotion-related self-regulation (e.g., Willoughby, Kupersmidt, Voegler-Lee, & Bryant, 2011). However, we would argue that it is not EF that is hot or cold, but that EF may function somewhat differently in hot and cold contexts (Eisenberg & Zhou, in press; see also Kim, Nordling, Yoon, Boldt, & Kochanska, 2013).

Researchers studying EF can also benefit from theoretical distinctions made in the EC literature. For example, temperament researchers have begun to study the development and correlates of *activational control*, which is the ability to activate behavior willfully. This is an aspect of EC that has no corresponding construct in the field of EF but merits further study due to the importance of up-regulating emotional reactions in certain situations—for example, showing gratitude when receiving a disappointing gift—as well as its likely role in persistence on unpleasant tasks and the achievement of long-term goals.

Design of Research Studies

There is growing recognition of the bidirectional nature of the relations between temperament and environmental influences. Researchers have come to understand that although temperament has a biological basis, it is also susceptible to environmental influences. This may be especially true for EC, which develops more slowly than more emotionally reactive aspects of temperament. Conversely, there is also evidence that children's characteristics may affect parenting behavior (e.g., Eisenberg, Vidmar, et al., 2010). Longitudinal designs in which temporal hypotheses can be tested are becoming more common and can help to clarify the degree to which the early environment influences children's temperament and vice versa.

Although such research designs have clear strengths, even well-designed correlational studies cannot definitively test causal relations. There is still a need for experimental studies, such as intervention and prevention trials that promote self-regulatory skills in children, to determine how these skills are causally related to self-regulation processes and other child outcomes. School-based prevention trials have demonstrated effects on self-regulation in low-income preschoolers (Diamond, Barnett, Thomas, & Munro, 2007), but more research is needed to determine whether gains are maintained over long periods of time, the extent to which increases in EC are causally related to a number of different child outcomes, whether such effects can be achieved in other age groups, and whether the intervention effects generalize to other populations (e.g., middle-income children or children in other cultures). Similarly, experimental studies that modify parenting behavior are needed to understand the causal effects of parenting on the development of EC.

Genetically informed research designs could also contribute to an understanding of the influences on, and effects of, children's self-regulation. Not only can twin, adoption, or sibling studies be used to assess the role of genetics and environment in self-regulation (see Rothbart & Bates, 2006), but they also can be used to assess environmental influences, especially when genetic influences are held constant. For example, one could examine factors related to differences between identical twins (who are genetically the same) in EC. A relatively new and promising area of research is the use of measured genes in studies of EC (Kochanska, Kim, Barry, & Philibert, 2011; Voelker, Sheese, Rothbart, & Posner, 2009). More work is needed in this area to determine whether such effects are replicable, but such work may help investigators target interventions and understand the biological mechanisms underlying the development of emotion and self-regulation.

The Measurement of Emotion-Related Self-Regulation

It is now common for studies of self-regulation to include a number of behavioral measures, allowing researchers to study the relations among tasks and to measure more accurately the underlying construct. However, a limitation of the majority of these measures is that performance quickly reaches a ceiling in early school-age children. One challenge for EC researchers is to establish measures that can be used across a relatively wide age range to establish continuity in measurement. In particular, measures appropriate for adolescents and adults are needed, and it would be advantageous if these measures also could be used with children. There is now evidence that it is possible to develop tasks appropriate for a wide age range, including adolescents and adults, making it possible to measure more accurately the development of EC beyond early childhood (Lagattuta, Sayfan, & Monsour, 2011; Tottenham, Hare, & Casey, 2011). Although there has been recent progress in this area, further work is still needed because a single laboratory measure of EC is relatively unreliable, and a battery of tasks is recommended in order to assess the construct more accurately (Kochanska & Knaack, 2003).

Psychophysiological methods have been underutilized in the existing research, even though it is possible to assess many psychophysiological variables inexpensively and noninvasively. Of particular interest for the assessment of EC is respiratory sinus arrhythmia (RSA), a cardiac measure of parasympathetic nervous system function. There is now a consistent body of evidence linking resting RSA to EC (e.g., Marcovitch et al., 2010). RSA reactivity—a change in RSA in response to task demands—has also been found in some studies to correlate with EC and EF, but the exact nature of this relation is still not clear. Although RSA reactivity may also be a promising as a means of assessing emotion-related regulation, further research is needed to determine how task demands relate to the interpretation of this measure (Obradović, Bush, & Boyce, 2011).

Process Models of Emotion-Related Self-Regulation

Finally, there is a need to understand *how* EC supports emotion-related self-regulation. EC likely plays a supporting role in a wide variety of emotion regulation strategies, includ-

ing reappraisal, attentional strategies such as distraction or avoidance, choosing or modifying situations, and inhibiting emotionally motivated behavior. Although there is ample evidence that EC is related to successful emotion regulation, we know relatively little about the extent to which the effectiveness of specific emotion regulation strategies is dependent on individual differences in EC, or whether specific aspects of EC such as activational control, inhibitory control, and attention focusing are differentially related to emotion-related self-regulation.

Conclusion

In summary, although the study of children's self-regulation is flourishing, there is much yet to be explored and understood. A multimethod approach, influenced by conceptual work in developmental, clinical, personality, social, and neurophysiological psychology, including greater integration of research and theory in the adult and child literatures, is likely to be productive in future attempts to expand our knowledge.

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References

Aksan, N., & Kochanska, G. (2004). Links between systems of inhibition from infancy to preschool years. *Child Development, 75*, 1477–1490.

Allan, N. P., & Lonigan, C. J. (2011). Examining the dimensionality of effortful control in preschool children and its relation to academic and socioemotional indicators. *Developmental Psychology, 47*, 905–915.

Bakker, M. P., Ormel, J., Verhulst, F. C., & Oldehinkel, A. J. (2011). Adolescent family adversity and mental health problems: The role of adaptive self-regulation capacities: The TRAILS study. *Journal of Abnormal Child Psychology, 39*, 341–350.

Bandstra, N. F., Chambers, C. T., McGrath, P. J., & Moore, C. (2011). The behavioural expression of empathy to others' pain versus others' sadness in young children. *PAIN, 152*, 1074–1082.

Best, J. R., & Miller, P. H. (2010). A developmental perspective on executive function. *Child Development, 81*, 1641–1660.

Blair, C., & Razza, R. P. (2007). Relating effortful control, executive function, and false belief understanding to emerging math and literacy ability in kindergarten. *Child Development, 78*, 647–663.

Block, J. H., & Block, J. (1980). The role of ego-control and ego-resiliency in the organization of behavior. In W. A. Collins (Ed.), *Minnesota Symposia on Child Psychology* (Vol. 13, pp. 39–101). Hillsdale, NJ: Erlbaum.

Carlson, S. M., Moses, L. J., & Claxton, L. J. (2004). Individual differences in executive functioning and theory of mind: An investigation of inhibitory control and planning ability. *Journal of Experimental Child Psychology, 87*, 299–319.

Carver, C. S. (2005). Impulse and constraint: Perspectives from personality psychology, convergence with theory in other areas, and potential for integration. *Personality and Social Psychology Review, 9*, 312–333.

Compas, B. E., Connor-Smith, J. K., Saltzman, H., Thomsen, A. H., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin, 127*, 87–127.

Crockenberg, S. C., & Leerkes, E. M. (2004). Infant and maternal behaviors regulate infant reactivity to novelty at 6 months. *Developmental Psychology, 40*, 1123–1132.

Denham, S. A., & Burton, R. (2003). *Social and emotional prevention and intervention programming for preschoolers*. New York: Kluwer Academic/Plenum Press.

Derryberry, D., & Rothbart, M. K. (1997). Reactive and effortful processes in the organization of temperament. *Development and Psychopathology, 9*, 633–652.

Diamond, A. (1991). Neuropsychological insights into the meaning of object concept development. In S. Carey & R. Gelman (Eds.), *The epigenesis of mind: Essays on biology and cognition* (pp. 67–110). Hillsdale, NJ: Erlbaum.

Diamond, A., Barnett, W. S., Thomas, J., & Munro, S. (2007). The early years: Preschool program improves cognitive control. *Science, 318*, 1387–1388.

Diener, M. L., & Kim, D. (2004). Maternal and child predictors of preschool children's social competence. *Journal of Applied Developmental Psychology, 25*, 3–24.

Diener, M. L., & Mangelsdorf, S. C. (1999). Behavioral strategies for emotion regulation in toddlers: Associations with maternal involvement and emotional expressions. *Infant Behavior and Development, 22*, 569–583.

Eisenberg, N., Edwards, A., Spinrad, T. L., Sallquist, J., Eggum, N. D., & Reiser, M. (2013, February 11). Are effortful and reactive control unique constructs in young children? *Developmental Psychology.*

Eisenberg, N., & Fabes, R. A. (1992). Emotion, regulation, and the development of social competence. In M. S. Clark (Ed.), *Review of personality and social psychology. Vol 14. Emotion and Social Behavior* (pp. 119–150). Newbury Park, CA: Sage Publications.

Eisenberg, N., Fabes, R. A., & Spinrad, T. L. (2006). Prosocial development. In W. Damon & R. M. Lerner (Series Eds.), & N. Eisenberg (Vol. Ed.), *Handbook of child psychology, Vol 3. Social, emotional, and personality development* (pp. 646–718). New York: Wiley.

Eisenberg, N., Guthrie, I. K., Fabes, R. A., Reiser, M., Murphy, B. C., Holgreen, R., et al. (1997). The relations of regulation and emotionality to resilience and competent social functioning in elementary school children. *Child Development, 68*, 295–311.

Eisenberg, N., Michalik, N., Spinrad, T. L., Hofer, C., Kupfer, A., Valiente, C., et al. (2007). Relations of effortful control and impulsivity to children's sympathy: A longitudinal study. *Cognitive Development, 22*, 544–567.

Eisenberg, N., & Morris, A. S. (2002). Children's emotion-related regulation. In R. Kail (Ed.), *Advances in child development and behavior* (pp. 190–229). Amsterdam: Academic Press.

Eisenberg, N., Smith, C. L., & Spinrad, T. L. (2011). Effortful control: Relations with emotion regulation, adjustment, and socialization in childhood. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 263–283). New York: Guilford Press.

Eisenberg, N., Spinrad, T. L., & Eggum, N. D. (2010). Emotion-related self-regulation and its relation to children's maladjustment. *Annual Review of Clinical Psychology, 6*, 495–525.

Eisenberg, N., Spinrad, T. L., Fabes, R. A., Reiser, M., Cumberland, A., Shepard, S. A., et al. (2004). The relations of effortful control and impulsivity to children's resiliency and adjustment. *Child Development, 75*, 25–46.

Eisenberg, N., Valiente, C., & Eggum, N. D. (2010). Self-regulation and school readiness. *Early Education and Development, 21*, 681–698.

Eisenberg, N., Valiente, C., Spinrad, T. L., Cumberland, A., Liew, J., Reiser, M., et al. (2009). Longitudinal relations of children's effortful control, impulsivity, and negative emotionality to their externalizing, internalizing, and co-occurring behavior problems. *Developmental Psychology, 45*, 988–1008.

Eisenberg, N., Vidmar, M., Spinrad, T. L., Eggum, N. D., Edwards, A., Gaertner, B., et al. (2010). Mothers' teaching strategies and children's effortful control: A longitudinal study. *Developmental Psychology, 46*, 1294–1308.

Eisenberg, N., Wentzel, M., & Harris, J. D. (1998). The role of emotionality and regulation in empathy-related responding. *School Psychology Review, 27*, 506–521.

Eisenberg, N., & Zhou, Q. (in press). Conceptions of executive functioning and regulation: When and to what degree do they overlap? In J. A. Griffin, L. S. Freund, & P. McCardle (Eds.), *Executive function in preschool age children: Integrating measurement, neurodevelopment and translational research.* Washington, DC: American Psychological Association.

Flynn, E. (2007). The role of inhibitory control in false belief understanding. *Infant and Child Development, 16*, 53–69.

Garon, N., Bryson, S. E., & Smith, I. M. (2008). Executive function in preschoolers: A review using an integrative framework. *Psychological Bulletin, 134*, 31–60.

Grolnick, W. S., Bridges, L. J., & Connell, J. P. (1996). Emotion regulation in two-year-olds: Strategies and emotional expression in four contexts. *Child Development, 67*, 928.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Guthrie, I. K., Eisenberg, N., Fabes, R. A., Murphy, B. C., Holmgren, R., Mazsk, P., et al. (1997). The relations of regulation and emotionality to children's situational empathy-related responding. *Motivation and Emotion, 21*, 87–108.

Hardaway, C. R., Wilson, M. N., Shaw, D. S., &

Dishion, T. J. (2012). Family functioning and externalizing behaviour among low-income children: Self-regulation as a mediator. *Infant and Child Development, 21*, 67–84.

Hill, A. L., & Braungart-Rieker, J. M. (2002). Four-month attentional regulation and its prediction of three-year compliance. *Infancy, 3*, 261–273.

Hofer, C., Eisenberg, N., & Reiser, M. (2010). The role of socialization, effortful control, and ego resiliency in French adolescents' social functioning. *Journal of Research on Adolescence, 20*, 555–582.

Izard, C. E., Schultz, D., Fine, S. E., Youngstrom, E., & Ackerman, B. P. (1999). Temperament, cognitive ability, emotion knowledge, and adaptive social behavior. *Imagination, Cognition and Personality, 19*(4), 305–330.

Kagan, J., & Fox, N. A. (2006). Biology, culture, and temperamental biases. In W. Damon & R. M. Lerner (Series Eds.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 167–225). Hoboken, NJ: Wiley.

Kim, S., Nordling, J. K., Yoon, J. E., Boldt, L. J., & Kochanska, G. (2013). Effortful control in "hot" and "cool" tasks differentially predicts children's behavior problems and academic performance. *Journal of Abnormal Child Psychology, 41*(1), 43–56.

Kochanska, G., Coy, K. C., & Murray, K. T. (2001). The development of self-regulation in the first four years of life. *Child Development, 72*, 1091–1111.

Kochanska, G., Coy, K. C., Tjebkes, T. L., & Husarek, S. J. (1998). Individual differences in emotionality in infancy. *Child Development, 69*, 375–390.

Kochanska, G., Kim, S., Barry, R. A., & Philibert, R. A. (2011). Children's genotypes interact with maternal responsive care in predicting children's competence: Diathesis–stress or differential susceptibility? *Development and Psychopathology, 23*, 605–616.

Kochanska, G., & Knaack, A. (2003). Effortful control as a personality characteristic of young children: Antecedents, correlates, and consequences. *Journal of Personality, 71*, 1087–1112.

Kochanska, G., Murray, K. T., & Harlan, E. T. (2000). Effortful control in early childhood: Continuity and change, antecedents, and implications for social development. *Developmental Psychology, 36*, 220–232.

Lagattuta, K. H., Sayfan, L., & Monsour, M. (2011). A new measure for assessing executive function across a wide age range: Children and adults find happy–sad more difficult than day–night. *Developmental Science, 14*, 481–489.

Lemery, K. S., Essex, M. J., & Smider, N. A. (2002). Revealing the relation between temperament and behavior problem symptoms by eliminating measurement confounding: Expert ratings and factor analyses. *Child Development, 73*, 867–882.

Lengua, L. J. (2008). Anxiousness, frustration, and effortful control as moderators of the relation between parenting and adjustment in middle-childhood. *Social Development, 17*, 554–577.

Lengua, L. J., Honorado, E., & Bush, N. R. (2007). Contextual risk and parenting as predictors of effortful control and social competence in preschool children. *Journal of Applied Developmental Psychology, 28*, 40–55.

Liew, J., Eisenberg, N., Spinrad, T. L., Eggum, N. D., Haugen, R. G., Kupfer, A., et al. (2011). Physiological regulation and fearfulness as predictors of young children's empathy-related reactions. *Social Development, 20*, 111–134.

Mangelsdorf, S. C., Shapiro, J. R., & Marzolf, D. (1995). Developmental and temperamental differences in emotion regulation in infancy. *Child Development, 66*, 1817–1828.

Marcovitch, S., Leigh, J., Calkins, S. D., Leerks, E. M., O'Brien, M., & Blankson, A. N. (2010). Moderate vagal withdrawal in 3.5-year-old children is associated with optimal performance on executive function tasks. *Developmental Psychobiology, 52*, 603–608.

Mintz, T. M., Hamre, B. K., & Hatfield, B. E. (2011). The role of effortful control in mediating the association between maternal sensitivity and children's social and relational competence and problems in first grade. *Early Education and Development, 22*, 360–387.

Mischel, W., & Ayduk, O. (2011). Willpower in a cognitive-affective processing system: The dynamics of delay of gratification. In K. D. Vohs & R. F. Baumeister (Eds.), *Handbook of self-regulation: Research, theory, and applications* (2nd ed., pp. 83–105). New York: Guilford Press.

Murphy, B. C., Eisenberg, N., Fabes, R. A., Shepard, S., & Guthrie, I. K. (1999). Consistency and change in children's emotionality and regulation: A longitudinal study. *Merill–Palmer Quarterly, 46*, 413–444.

Obradović, J., Bush, N. R., & Boyce, W. T. (2011). The interactive effect of marital conflict and stress reactivity on externalizing and internalizing symptoms: The role of laboratory stressors. *Development and Psychopathology, 23*, 101–114.

Oldehinkel, A. J., Hartman, C. A., Ferdinand, R. F., Verhulst, F. C., & Ormel, J. (2007). Effortful control as modifier of the association between negative emotionality and adolescents' mental health problems. *Development and Psychopathology, 19*, 523–539.

Panfile, T. M., & Laible, D. J. (2012). Attachment security and child's empathy: The mediating role of emotion regulation. *Merrill–Palmer Quarterly, 58*, 1–21.

Posner, M. I., & Rothbart, M. K. (2000). Developing mechanisms of self-regulation. *Development and Psychopathology, 12*, 427–441.

Rothbart, M. K. (2011). *Becoming who we are: Temperament and personality in development*. New York: Guilford Press.

Rothbart, M. K., Ahadi, S. A., & Hershey, K. L. (1994). Temperament and social behavior in childhood. *Merill–Palmer Quarterly, 40*, 21–39.

Rothbart, M. K., Ahadi, S. A., Hershey, K., & Fisher, P. (2001). Investigations of temperament at three to seven years: The children's behavior questionnaire. *Child Development, 72*, 1394–1408.

Rothbart, M. K., & Bates, J. E. (2006). Temperament. In W. Damon & R. M. Lerner (Series Eds.) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 99–166). Hoboken, NJ: Wiley.

Ruff, H. A., & Rothbart, M. K. (1996). *Attention in early development: Themes and variations*. London: Oxford University Press.

Schultz, D., Izard, C. E., Ackerman, B. P., & Youngstrom, E. A. (2001). Emotion knowledge in economically disadvantaged children: Self-regulatory antecedents and relations to social difficulties and withdrawal. *Development and Psychopathology, 13*, 53–67.

Silva, K. M., Spinrad, T. L., Eisenberg, N., Sulik, M. J., Valiente, C., Huerta, S., et al. (2011). Relations of children's effortful control and teacher–child relationship quality to school attitudes in a low-income sample. *Early Education and Development, 22*, 434–460.

Spinrad, T. L., Eisenberg, N., Cumberland, A., Fabes, R. A., Valiente, C., Shepard, S., et al. (2006). Relation of emotion-related regulation to children's social competence: A longitudinal study. *Emotion, 6*, 498–510.

Spinrad, T. L., Eisenberg, N., Gaertner, B., Popp, T., Smith, C. L., Kupfer, A., et al. (2007). Relations of maternal socialization and toddlers' effortful control to children's adjustment and social competence. *Developmental Psychology, 43*, 1170–1186.

Spinrad, T. L., Eisenberg, N., Silva, K. M., Eggum, N. D., Reiser, M., Edwards, A., et al. (2012). Longitudinal relations among maternal behaviors, effortful control and young children's committed compliance. *Developmental Psychology, 48*, 552–566.

Stifter, C. A., & Braungart, J. M. (1995). The regulation of negative reactivity in infancy: Function and development. *Developmental Psychology, 31*, 448–455.

Sulik, M. J., Huerta, S., Zerr, A. A., Eisenberg, N., Spinrad, T. L., Valiente, C., et al. (2010). The factor structure of effortful control and measurement invariance across ethnicity and sex in a high-risk sample. *Journal of Psychopathology and Behavioral Assessment, 32*, 8–22.

Tottenham, N., Hare, T. A., & Casey, B. J. (2011). Behavioral assessment of emotion discrimination, emotion regulation, and cognitive control in childhood, adolescence, and adulthood. *Frontiers in Developmental Psychology, 2*, 1–9.

Ursache, A., Blair, C., & Raver, C. C. (2012). The promotion of self-regulation as a means of enhancing school readiness and early achievement in children at risk for school failure. *Child Development Perspectives, 6*, 122–128.

Valiente, C., Eisenberg, N., Haugen, R., Spinrad, T. L., Hofer, C., Liew, J., et al. (2011). Children's effortful control and academic achievement: Mediation through social functioning. *Early Education and Development, 22*, 411–433.

Valiente, C., Eisenberg, N., Smith, C. L., Reiser, M., Fabes, R. A., Losoya, S., et al. (2003). The relations of effortful control and reactive control to children's externalizing problems: A longitudinal assessment. *Journal of Personality, 71*, 1172–1196.

Verstraeten, K., Vasey, M. W., Raes, F., & Bijttebier, P. (2008). Temperament and risk for depressive symptoms in adolescence: Mediation by rumination and moderation by effortful control. *Journal of Abnormal Child Psychology, 37*, 349–361.

Voelker, P., Sheese, B. E., Rothbart, M. K., & Posner, M. I. (2009). Variations in catechol-

O-methyltransferase gene interact with parenting to influence attention in early development. *Neuroscience, 164*, 121–130.

Williams, B. R., Ponesse, J. S., Schachar, R. J., Logan, G. D., & Tannock, R. (1999). Development of inhibitory control across the life span. *Developmental Psychology, 35*, 205–213.

Willoughby, M. T., Kupersmidt, J., Voegler-Lee, M., & Bryant, D. (2011). Contributions of hot and cool self-regulation to preschool disruptive behavior and academic achievement. *Developmental Neuropsychology, 36*, 162–180.

Willoughby, M. T., Wirth, R. J., Blair, C. B., & Family Life Project Investigators. (2012). Executive function in early childhood: Longitudinal measurement invariance and developmental change. *Psychological Assessment, 24*, 418–431.

Zhou, Q., Eisenberg, N., Wang, Y., & Reiser, M. (2004). Chinese children's effortful control and dispositional anger/frustration: Relations to parenting styles and children's social functioning. *Developmental Psychology, 40*, 352–366.

CHAPTER 11

Socialization of Emotion and Emotion Regulation in the Family

Ross A. Thompson

The development of emotion regulation is one of the central goals of early socialization because of its importance to social competence, academic achievement, and psychological well-being (Saarni, 1999). It is also important to parent–child relationships (Cassidy, 1994). Parents typically overestimate how much young children can manage their feelings, especially when their judgments are compared with what research has shown about the development of self-regulatory ability (Newton & Thompson, 2010). In a recent national survey, for example, many parents reported that children under the age of 3 could control their emotions when frustrated, and most thought that children of this age could competently share and take turns with other children (Newton & Thompson, 2010). This may help to explain why parental efforts to foster emotional self-control in offspring are so concerted and multifaceted. Their efforts include proactive strategies to manage the everyday emotional demands that children face, direct interventions to regulate children's emotions, parents' evaluations of children's emotional displays, sanctions on unregulated emotional displays, and conversation in which emotional control strategies are discussed and the benefits of self-management are described. The socialization of emotion regulation in the family is particularly interesting, therefore, as a process of considerable importance to parents but also as one in which parents' efforts are leading, sometimes significantly, children's developing behavioral and neurobiological capacities.

As the study of the development of emotion regulation has expanded, research on the socialization of emotion regulation has changed. Emotion regulation was conventionally viewed in terms of the top-down imposition of cognitive or neurobiological control on basic emotional processes, and socialization was perceived primarily as helping children acquire the understanding, self-control strategies, motivation, and competence needed to enact these control processes. In recent years, however, developmental researchers have recognized that emotion regulation also involves bottom-up processes, such as how basic emotion appraisals originating in limbic structures influence higher control processes (Thompson, 2011). From this view, therefore, the socialization of emotion regulation involves family processes that influence the growth of emotional reactivity and the demands this imposes on emotional self-control. This chapter addresses both top-down and bottom-up facets of the socialization of emotion regulation, because each is important to how family experiences affect children's emotional self-control.

The chapter begins with a definition of emotion regulation that highlights its relevance to understanding these socialization processes. Next, the discussion turns to family processes that influence bottom-up aspects of emotion regulation, with particular attention to experiences of stress that can shape the neurobiology of emotion. In the third section, the socialization of emotion regulation as a top-down process is considered with respect to the diverse parental influences that affect how children interpret and appraise their feelings, learn about strategies of emotion management, achieve competence in controlling their feelings, and acquire cultural and gender expectations for emotion regulation (for a more extended discussion of these influences, see Morris, Silk, Steinberg, Myers, & Robinson, 2007; Thompson & Meyer, 2007). In the fourth section I integrate these perspectives with respect to the broader security of parent–child attachment, then conclude the chapter with final reflections.

Defining Emotion Regulation

Definitions of emotion regulation are built on broader conceptualizations of emotion (see Gross, this volume). A developmental approach to emotion regulation highlights the changes that occur over time in multiple components of emotion and their interaction, and how these emotion components unfold through biological maturation, social influences, the growth of self-referential processes, and many other developmental influences (Thompson, 1990, 1994, 2011). The following definition of *emotion regulation* reflects this developmental approach: Emotion regulation consists of the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one's goals (Thompson, 1994, pp. 27–28). Incorporated within this definition are several assumptions about emotion and emotion regulation.

First, emotion regulation processes target positive as well as negative emotions and can entail diminishing, heightening, or simply maintaining one's current level of emotional arousal in particular circumstances. Emotion regulation usually alters the *dynamics* of emotion rather than changing its quality. In other words, individuals alter the intensity, escalation (i.e., latency to onset and rise time), or duration of an emotional response, speed its recovery, or reduce or enhance the lability or range of emotional responding in particular situations depending on the individual's goals for that situation (Thompson, 1990). We usually think of emotionally well-regulated people as those who are capable of altering how long, how intensely, or how quickly they feel as they do, rather than transforming the valence of emotion (e.g., changing anger into happiness).

Second, consistent with broader understanding of emotional development, emotion regulation can occur through the influence of other people or through the individual's own efforts. Both extrinsic and intrinsic processes of emotion regulation are important. From birth, children's emotional lives are managed by others, and this remains so throughout life. Moreover, extrinsic influences on emotion regulation can be either facilitative (i.e., those that assist the child in emotional self-control) or hindering (i.e., those that impair the child's emotional self-control), with the latter occurring often in contexts of family disruption. In addition, extrinsic influences on emotion regulation can be either deliberate (i.e., intentionally managing the child's emotions) or unintended (i.e., not meant to have the effects on children's emotions that they do).

Third, consistent with a functionalist approach to emotion, strategies of emotion regulation are rarely inherently optimal or maladaptive. Rather, emotion regulation strategies must be evaluated in relation to the individual's goals for the situation. This functionalist orientation is especially important for developmental analysis. A toddler's petulant crying or an adolescent's sullenness may be intuitively interpreted by others as revealing deficient skills in emotion regulation until one realizes that the toddler's crying causes parents to accede and the adolescent's sullenness causes peers to provide support, each of which may be the child's goal (even if these emotional reactions have other, negative consequences). It is important, however, to recognize that a person can have multiple goals in a situation requiring emotion regulation (e.g., managing fear

and defending oneself when in the presence of a bully). These alternative goals may conflict, which further complicates evaluating whether specific emotion regulation strategies are adaptive.

This functionalist orientation is also important for understanding emotion regulatory processes relevant to developmental psychopathology. Children with anxiety disorders are typically viewed as deficient in emotion regulation, for example, but their hypervigilance to threatening events, fear-oriented cognitions, and sensitivity to internal visceral cues of anxiety are part of a constellation of self-regulatory strategies for anticipating and avoiding encounters with fear-provoking events. In light of their genetic vulnerability and family processes that heighten risk for anxious pathology, these emotion regulatory strategies may be the most adaptive options available to the child (Thompson, 2000). Characterizing such children simply as emotionally dysregulated seems oversimplified. To be sure, the same emotion regulatory strategies that provide immediate relief can exact long-term costs that make anxious children vulnerable to continued pathology. This double-edged sword is typical of emotional regulatory processes for many forms of developmental psychopathology (Thompson & Goodman, 2010). Understanding emotion regulation for children at risk requires comprehending the emotional goals the child is seeking to achieve and interpreting his or her self-regulatory efforts in light of these goals.

Fourth, consistent with a developmental analysis, emotion regulation includes understanding how people monitor and evaluate, as well as modify, their emotions. Children's developing capacities for emotional self-awareness and for appraising their feelings in light of personal and cultural expectations are core features of developing emotion regulation (Saarni, 1999). Emotion monitoring and evaluation function nonconsciously as well as consciously to affect self-regulation. A preschooler arriving at child care after witnessing her parents' morning argument may not be consciously monitoring how upset she is, for example, but the argument might affect her emotional self-control when she is later provoked by a peer. In this situation, antecedent emotional arousal creates a lowered emotional threshold for subsequent negative arousal that impairs the child's emotion management.

Emotion regulation can be studied at multiple levels of analysis, from genetic and neurobiological processes to developing cognition and personality, to family processes and overarching cultural values. A developmental analysis highlights that although psychologists tend to regard "emotion regulation" as if it were a single, coherent personality construct or developmental process, the growth of emotion regulation is actually based on a multidimensional network of loosely allied processes arising from within and outside the child. Developmental study of emotion regulation requires that theorists keep the broader view in mind even as they are studying specific components of the growth of emotion regulation, such as its socialization in the family.

The developmental study of emotion regulation requires distinct methodological strategies. Studies of emotion regulation in adults or adolescents typically rely on respondent self-report, commonly through questionnaires, to index individual differences in emotion self-regulation. Infants and young children are not very informative reporters. Thus, developmental researchers must use other procedures. These include detailed observations of emotional reactions in carefully structured experimental situations, often with convergent behavioral and psychophysiological measures, sometimes accompanied by the reports of mothers and other secondary sources concerning the child's emotional qualities. These methods are informative, but behavioral measures (whether of infants or adults) are also complicated by the following interpretive difficulties: (1) behavior that reflects the influence of emotion regulatory processes is multidetermined; (2) emotional reactions and emotional regulatory influences are not easily distinguished behaviorally; and (3) the situational context can have a profound effect on children's emotional reactions (Cole, Martin, & Dennis, 2004). Studying emotion regulation *in vivo* in this manner is thus conceptually and methodologically more difficult than enlisting self-report.

Adding further complexity to the study of emotion regulation is the functionalist requirement of understanding the goals motivating self-regulatory efforts. Although

these goals are usually assumed in studies of adults (e.g., diminishing negative affect and enhancing positive emotion), behavioral studies of emotion regulation with children require carefully designed assessments in which the goals for managing emotion are either explicit or incorporated into the design (e.g., observing children as they cope with a disappointing gift). In short, because of the special methodological challenges it presents, developmental research into emotion regulation is not for the fainthearted.

Family Influences on Emotional Reactivity and Self-Regulation

Two conclusions from recent research have changed how developmental researchers understand the growth of emotion regulation (Thompson, 2011; Thompson, Lewis, & Calkins, 2008). First, as noted earlier, neurobiological systems that are higher and lower on the neuroaxis exert mutual regulatory influences related to emotion. "Top-down" regulatory control (e.g., projections from the prefrontal cortex to the amygdala) is important, but so is the "bottom-up" regulatory influence (e.g., from the limbic system to higher cortical regions). Second, these higher and lower neurobiological systems are shaped by the quality of early experience, particularly family interactions.

The view that emotion regulation involves bidirectional influences between higher and lower systems is consistent with current work in the neuroscience of emotion. Neuroimaging studies show that responses to emotion tasks are widely distributed throughout the brain, including areas commonly regarded as relevant to affective activation (including the amygdala, hypothalamus, brainstem, and striatum) and areas often viewed as important to emotion regulation (including medial and lateral prefrontal cortex and anterior cingulate). Neuroimaging studies show that cortical and limbic areas are coactive in responses to emotion probes (Kober et al., 2008; Ochsner et al., 2009), and that limbic systems exert influence over cortical systems in affective arousal, as well as the reverse. Amygdala activation is associated, for example, with enhanced perceptual sensitivity to cues of danger, consistent with its role in affective learning and appraisals that influence higher-order emotion processing (Barrett & Bar, 2009; Ochsner et al., 2009; Woltering & Lewis, 2009).

Consistent with the view that early experiences shape brain architecture, research has shown that experience is important to the functioning of higher and lower brain regions relevant to emotion. The best evidence concerns the development of stress responding, which involves neural systems shared with emotion. Developmental studies with humans and animals indicate that children growing up in adverse conditions develop altered autonomic nervous system functioning that causes them to become more sensitive to environmental demands, more likely to become biologically and emotionally reactive (or underresponsive) to challenge, and less capable of adaptive self-regulation (Loman & Gunnar, 2010; Lupien, McEwen, Gunnar, & Heim, 2009). These consequences have been documented in the limbic–hypothalamic–pituitary–adrenocortical (LHPA) axis (which is relevant to affect and stress responding), the parasympathetic system, and multiple areas of the prefrontal cortex, indicating that early stress can have diverse and pervasive effects on emotion and emotion regulation (Blair, 2010; El-Sheikh & Erath, 2011). Importantly, early family experiences have been the model for early adversity in this research literature, related both to the stresses directly imposed by parental behavior (e.g., experiences of abuse or chronic neglect) and the failure of caregivers to buffer the effects of other stresses on children. Children with a history of abuse, for example, exhibit multiple indications of difficulty in emotion processing relevant to their maltreatment: They are hypersensitive to adult expressions of anger, have a lower threshold for detecting anger in maternal vocal expressions, and have more difficulty attentionally disengaging from perceived angry cues when these responses are measured both behaviorally and neurobiologically (see Pollak, 2008, for a review). Hypersensitivity to anger and threat is probably an adaptive emotional regulatory strategy in contexts in which children cannot avert an adult attack but may be capable of anticipating it and flee, avoid, or otherwise prepare for it. But it comes with a cost: Children who are hyperreactive to cues of threat are less likely to be capable

of managing their arousal, as this research indicates. Moreover, outside the home, hyperreactivity to potential threat is likely to be a liability in dealing with other adults and peers, whose social cues are more likely to be misinterpreted and imbued with hostile intent (Thompson & Goodman, 2010). It is important to note that these deficiencies in emotion regulation are not pervasive, and abused children are not hyperreactive to cues of adult sadness or happiness (Pollak, 2008). Rather, children's self-regulatory problems are related to the circumstances of their family adversity, consistent with a functionalist view of emotion regulation.

The research does not permit an evaluation of the nature, severity, or chronicity of early stresses in the family that prompt these neurobiological and behavioral adaptations. There is some evidence that the association between family stress and its effects on behavioral functioning increment in a dose–response fashion (Repetti, Robles, & Reynolds, 2011). Nor is there clear evidence concerning the extent to which early experiences have privileged influence on the growth of neurobiological and behavioral systems related to emotion and its regulation, or whether later experiences of adversity have comparable effects. There is reason to believe, however, that early experiences may be especially important because of the plasticity of immature brain systems related to emotion and stress (Goldsmith, Pollak, & Davidson, 2008). Taken together, however, this research indicates that one way emotion regulation is socialized in the family is through children's exposure to the emotional demands and stresses of family life, especially in the context of the (lack of) support that exists for children's competent management of the emotional demands on them. These experiences shape the functioning of lower and higher neurobiological systems associated with emotional arousal and its regulation, and equip children with response propensities that are likely to influence their emotional responding outside of the home.

Such a conclusion is consistent with considerable behavioral research conducted in the context of less extreme family contexts. In the following sections, I discuss developmental studies of emotion regulation that are consistent with the foregoing account of neurobiological growth in two areas: parents' direct interventions to manage children's emotions, and effects of the broader emotional climate of the family.

Parents' Interventions to Manage Emotion

From birth, parents exert considerable effort to manage the emotions of their infants and young children by soothing distress, provoking positive emotion, and allaying fear. They proactively seek to make the everyday emotional demands on children manageable and predictable by organizing the child's daily routine, scheduling meals and naps, choosing appropriate child care arrangements, and in other ways. Parents also seek to alter children's direct appraisals of emotionally evocative situations, such as distracting children away from potentially frightening or distressing events, assisting in solving problems that children find frustrating, striving to alter the children's interpretations of negatively arousing experiences (e.g., "It's just a game"), and through social referencing. By directly intervening in these ways, parents act as external regulators of young children's emotions.

Beyond their immediate consequences, these parental interventions have broader consequences for early emotion and emotion regulation. With respect to parental soothing of infant distress, for example, Lamb (1981) has argued that such distress relief sequences are easily learned by infants and contribute to the emergence of rudimentary expectations that the parent's arrival will relieve distress. By 6 months of age, for example, distressed infants begin quieting in anticipation of the arrival of their mother when they hear her approaching footsteps; infants also protest loudly if mother approaches but does not pick them up to soothe them (Gekoski, Rovee-Collier, & Carulli-Rabinowitz, 1983; Lamb & Malkin, 1986). The parent's sensitive responding thus contributes to anticipatory soothing (an early self-regulatory response) before the parent's arrival. Similarly, the mother's sensitivity during episodes of animated, face-to-face play with the baby contributes to the growth of early capacities for self-regulation as the infant learns how to maintain manageable arousal in the context of social play

with the caregiver (Feldman, Greenbaum, & Yirmiya, 1999; Gianino & Tronick, 1988).

Parents who provide support to very young children facing emotional challenges have children who are more emotionally competent. Calkins and Johnson (1998) found that 18-month-olds who became more distressed during frustration tasks had mothers who were independently observed to be more interfering when interacting with their offspring, while children who could use problem solving and distraction during frustration had mothers who had earlier offered greater support, suggestions, and encouragement. In another study, mothers who insisted that their toddlers approach and confront potentially fearful objects in the laboratory had children who exhibited greater stress, as indexed by postsession cortisol levels (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). These findings suggest that parental assistance is important not only for managing immediate emotional behavior but also for the development of children's emotional self-control through the child's expectation that distress is manageable and that adults can assist in managing emotionally challenging situations.

These studies focus primarily on parental interventions that buffer children's emotional arousal and support self-regulatory capacity. As the research on stress indicates, however, parental behavior may not always be so supportive, and when this occurs there is greater risk for problems with emotional reactivity and self-regulation. Another research literature supporting this view concerns parental *expressed emotion*, which is an index of parental attitudes of criticism or emotional overinvolvement in the child's problems that can undermine competent emotional functioning (e.g., Hooley & Richters, 1995). Although expressed emotion has been examined most extensively in clinical studies of schizophrenia, depression, and bipolar disorder, because of its relevance to the maintenance or relapse of clinical symptomatology, developmental studies have found that expressed emotion is associated with the onset of psychological problems in children (Caspi et al., 2004). For example, one study found expressed emotion to be particularly prevalent in homes with a depressed parent (Rogosch, Cicchetti, & Toth, 2004). In the context of expressed emotion, therefore, parental responses that are critical of a child's emotional behavior can contribute to the child's risk of developing psychopathology involving emotion dysregulation. Risk is enhanced because the parent's critical demeanor is stressful, undermines opportunities for the child to learn more adaptive forms of emotional coping, and creates a more difficult family emotional climate for troubled children.

Direct parental interventions to manage children's emotions decline in frequency in early childhood as young children acquire their own self-regulatory strategies. However, direct interventions remain an important source of extrinsic influence on emotion regulation throughout life as they are supplemented by other socialization influences. Adults commonly rely on the support of family and friends when facing emotional stresses. Social support is thus a significant buffer of psychosocial stress, and social isolation is a risk factor for stress-related problems (Thompson, Flood, & Goodvin, 2006).

Emotional Climate of Family Life

The emotional climate of family life can make emotion management easier or more difficult because of the emotional demands that children encounter in the home. As suggested by the research on expressed emotion, when children must cope with frequent, intensive negative emotion from other family members, particularly when it is directed at them, it can contribute to their hypersensitivity to threat and undermine emotion regulation capacities. The family emotional climate is also relevant to emotion regulation because of the models of emotion regulation to which children are exposed and how the family environment shapes children's developing schemas for emotion and its management (e.g., Are emotions threatening? Empowering? Uncontrollable?).

Another illustration of the importance of the family emotional climate for developing child emotional reactivity and self-regulation is research on marital conflict (Davies & Woitach, 2008). According to the Cummings and Davies (2010) emotional security hypothesis, children seek to reestablish the emotional security they have lost in families with marital conflict, even when parental conflict is not directed at

them. They do so by intervening in parental arguments to end them, monitoring parental moods to anticipate the outbreak of arguments, and otherwise striving to manage their emotions in a conflicted home environment. As a consequence, they show heightened sensitivity to parental distress and anger, tend to become overinvolved in their parents' emotional conflicts, have difficulty managing the strong emotions that conflict arouses in them (in a manner resembling the "emotional flooding" described by emotion theorists), and exhibit signs of early development of internalizing problems. Research derived from this view has found that grade school children who witness the most intense marital conflict not only exhibit the greatest enmeshment in family conflict but also go to greater efforts to avoid conflict, while also showing more signs of internalizing symptomatology (Davies & Forman, 2002; see also Davies, Harold, Goeke-Morey, & Cummings, 2002). An important facet of the family emotional life is, of course, parents' emotional expressiveness (Halberstadt, Crisp, & Eaton, 1999; Halberstadt & Eaton, 2003). A series of studies by Eisenberg and her colleagues showed that children's social competence is affected by how mothers convey positive or negative feelings in the home—and this association is mediated by differences in children's self-regulatory behavior (Eisenberg et al., 2001, 2003; Valiente, Fabes, Eisenberg, & Spinrad, 2004). These findings suggest that a family climate characterized by moderate to high amounts of positive emotion among family members contributes to the growth of emotion regulation, perhaps because emotional demands are manageable within the home and children are exposed to models of skillful emotion self-regulation.

With respect to the influence of negative emotional expressiveness in the family, several studies report that negative maternal expressions of emotion are associated with children's lack of self-regulation and coping, but others have found a positive association (Eisenberg, Cumberland, & Spinrad, 1998; Eisenberg et al., 2001, 2003; Valiente et al., 2004). Viewed in the context of the research on expressed emotion and marital conflict, the effects of negative parental emotional expressiveness may depend on whether emotions are directed toward the child or not, the frequency and intensity of these emotions, and whether negative emotions are *negative dominant* (e.g., anger and hostility) or *negative submissive* (e.g., sadness and distress), with the former more likely to elicit the child's fear or defensiveness. One can see how emotion regulatory skills might be enhanced (rather than undermined) by a child's exposure to nonhostile negative emotions of moderate intensity in contexts in which negative feelings can be safely expressed and managed, in contrast with exposure to a more hostile, threatening family emotional environment.

An important influence on the emotional climate of the family—which also affects how parents evaluate and respond to the emotions of offspring—is parental beliefs about emotion and its expression. These beliefs can be considered part of a parent's "meta-emotion philosophy," which includes the adult's awareness of her or his own emotions, an understanding and acceptance of the child's emotions, and the parent's own approach to managing the child's feelings (Gottman, Katz, & Hooven, 1997; Hooven, Gottman, & Katz, 1995). Based on parental interviews, Gottman and his colleagues distinguish between "emotion coaching" and "emotion dismissing" parenting styles. Emotion-coaching parents are attentive to their own emotions and the child's feelings, and do not believe that feelings should be stifled. They consider the child's emotional expressions an occasion to validate the child's feelings, and an opportunity to teach the child about emotions, expression, and coping. Thus, emotion-coaching parents foster the growth of emotion regulation in offspring by offering warm support and specific guidance for managing feelings. Dismissive parents tend to ignore their own emotions or belittle the importance of emotions, and they may not constructively attend to their children's feelings. They view emotions (especially negative ones) as potentially harmful and believe that parents are responsible for promptly subduing negative outbursts in offspring and teaching their children that negative emotions are fleeting and unimportant.

Several studies testing this provocative formulation have yielded supportive but mixed results (Katz, Maliken, & Stettler, 2012). One study indicated that emotion

coaching and emotion dismissing interact complexly as influences on children's emotion regulation (Lunkenheimer, Shields, & Cortina, 2007; see also Gottman, Katz, & Hooven, 1996; Ramsden & Hubbard, 2002). Research in my laboratory confirmed that the association between maternal representations of emotion and children's emotion regulation was mediated by mothers' constructive emotion-related socialization practices (Thompson, Virmani, Waters, Meyer, & Raikes, 2013). Mothers' emotionally supportive representations (i.e., attention to her feelings; her focus on constructive emotion self-regulation) were associated with children's constructive emotion regulation (e.g., problem-focused and emotion-focused strategies) primarily through their association with positive, supportive emotion regulation socialization behaviors (i.e., maternal problem-solving encouragement, emotion-focused comfort, validation of children's feelings, and positive family expressivity).

Taken together, the emotional climate of family life influences the development of emotion regulation through both the emotional demands and supports it provides that affect children's developing emotional reactivity. In studies of the development of stress reactivity, the effects of marital conflict, and the consequences of parental positive and negative emotional expressivity, it is clear that skills in emotion management develop, in part, in reaction to the emotional demands with which children must daily cope. The research on parental meta-emotion philosophy highlights one source of variability in the family emotional climate, and it also identifies the basis for parental coaching of emotion regulatory strategies in offspring. In the next section, I consider these and other kinds of top-down regulatory influences.

Family Influences on Emotion Self-Regulatory Strategies

As prefrontal regions of the brain progressively mature, these higher brain systems enable children to become more capable of deliberately managing their feelings, impulses, and behavior. Growth in children's capacities to understand and enact cognitive and attentional strategies to manage emotional reactions accompanies the development of these neurobiological control processes. These top-down influences on emotion regulation highlight another avenue for socialization influences in the family. In direct and indirect ways, parents guide how children learn to appraise their feelings, confront the demands of emotion regulation at home or in other social settings, acquire specific skills for managing their feelings, understand cultural expectations for emotional self-control, and represent emotion processes relevant to self-regulation. In contrast to the family influences (e.g., marital conflict) that shape developing emotional reactivity and usually have unintended effects on children's emotion regulation, these socialization influences are usually deliberate. In coaching, conversation, and other modes of family interaction, parents encourage the development of well-regulated emotion and children's understanding, motivation, and competence to accomplish this.

Because these socialization processes extend throughout life and mediate cultural and gender differences in emotion management, individuals reach adulthood with emotion self-regulation skills that have been, to a large extent, socially constructed. In the following sections, two general socialization influences on developing emotion regulation are considered: parental evaluations of children's emotions, and parent–child conversation about emotion and its regulation.

Parental Evaluations of Children's Emotions

Emotion regulation can be facilitated or impaired by how others evaluate one's feelings. Sympathetic, constructive responses affirm that one's feelings are justified and provide social support in understanding and advice that help people cope with difficult situations. However, denigrating, critical, or dismissive responses contain implicit messages that demean the appropriateness of the feelings or their expression, or the competence of the person feeling this way. Indeed, others' dismissiveness can exacerbate the negative emotions that one is trying to manage (in part by arousing further emotion), as well as diminish opportunities for acquiring more adaptive modes of emotion regulation.

For children, these parental evaluations also become internalized guides for how children judge their own emotional reactions.

Developmental studies indicate that children cope more adaptively with their emotions in immediate circumstances and acquire more constructive emotion regulatory capacities when parents respond acceptingly and supportively to their negative emotional displays. By contrast, outcomes are more negative when parents are denigrating, punitive, or dismissive, or when the child's negative emotions elicit parents' personal distress (for reviews, see Denham, 1998; Denham, Bassett, & Wyatt, 2007; Eisenberg et al., 1998). In a socioeconomically disadvantaged sample, for example, mothers who reported exerting more positive control (using warmth and approval) over their sons at age 1½ had children who were observed to manage their negative emotions more constructively (e.g., by using self-distraction) at age 3½ (Gilliom, Shaw, Beck, Schonberg, & Lukon, 2002). Eisenberg, Fabes, and Murphy (1996) found that mothers' self-reported problem-solving responses to their grade school children's negative emotions were associated with independent reports of their children's constructive coping with problems (e.g., seeking support, problem solving, and positive thinking), while mothers' punitive and minimizing reactions to children's emotions were negatively associated with children's constructive coping and positively associated with avoidant coping. Research in my laboratory indicates that parents who accepted and validated young children's feelings during conversations about recent difficult events had children who were more willing to talk about these experiences and as a result more likely to learn about their emotions and how to manage them (Waters, Virmani, Thompson, Meyer, Raikes, & Jochem, 2010). These studies indicate that how parents respond to children's emotions, and the behaviors that result, predict children's emotion-related coping in later assessments.

Parent–Child Conversation and Developing Emotion Representations

From an early age, parents actively coach children on strategies of emotion regulation when they encourage offspring to "use words to say how you feel," take a deep breath, or think of something nice when emotionally aroused. Research in my laboratory indicates that in conversation with their preschoolers about recent emotional events, mothers directly suggest strategies of emotional self-control and explicitly endorse certain strategies over others (Thompson, Waters, Meyer, Raikes, Jochem, & Virmani, 2009). They do so in light of their expectations for age-appropriate emotional behavior in social settings (Cassano & Zeman, 2010).

Parent–child conversations have, however, broader significance for the growth of emotion regulation. Consistent with the work of Gottman and his colleagues (1997) on parental coaching, conversation shapes children's broader representations of emotion. Understanding the causes and consequences of their feelings, comprehending gender and cultural expectations for emotional expression, learning the social functions of emotional behavior, and other aspects of emotion understanding are advanced by parent–child conversation (Thompson, 2006a, 2006b). These broader aspects of emotion understanding are important to developing an informed comprehension of emotion regulation. As their conceptual skills mature, children begin to appreciate, especially through conversations with parents, that they can enlist internal constituents of emotion to manage external emotion (e.g., redirecting attention; cognitive reappraisal; mental distraction), that one need not always show what one feels, and that mental processes (e.g., expectations, memories, thoughts) affect how one feels.

Parent–child conversation has further significance for emotion regulation, moreover, as a means of guiding children's immediate appraisals of events. By managing information the child receives about potentially stressful events (e.g., describing an anticipated dental visit as "teeth tickling") or introducing alternative interpretations of an event (e.g., eliciting sympathy for a physically challenged person the child fears or finds amusing), parents influence emotion-related appraisals that facilitate emotion regulation. In an experimental probe, Morris et al. (2011) found that mothers' use of attention refocusing and cognitive reframing was associated with less sadness and anger

in children who received a disappointing gift in her presence (see Kliewer, Fearnow, & Miller, 1996, for similar results with mothers and fathers). Comparable to how parent–child conversations influence memory through attentional redirection, therefore, these conversations can influence emotion through reorienting appraisal processes (Thompson, 2006b).

It is important to note that conversations with peers and siblings are also important catalysts to the growth of emotion regulation in childhood. Young children talk about their feelings more frequently with friends and siblings than they do with their mothers, and these conversations also contribute to developing emotional understanding (Brown, Donelan-McCall, & Dunn, 1996). Peer conversations are important; there is evidence that adaptive strategies of emotion regulation with peers are not the same as those with family members and adult interactions (Thompson & Waters, 2010). Venting distress does not typically elicit as much support from peers as from parents, for example. Thus, as children travel between different social settings, they must develop competence in applying different norms for emotional behavior and accepted strategies for emotion regulation.

An Integrative Perspective: Parent–Child Attachment Security

The preceding discussion focused on immediate and longer-term socialization processes by which children become competent at managing their emotions. But *what* happens and *who* does it are both important in emotion socialization. Most of the socialization influences on emotion regulation discussed in this chapter occur in a relational context, and the quality of the parent–child relationship colors the influence of parents' direct interventions, appraisals of children's emotions, coaching, and conversations about emotion and its regulation. In this section, I discuss the significance of the security of the parent–child relationship, and in doing so reintegrate the bottom-up and top-down processes discussed earlier.

According to attachment researchers, differences in the security of child–parent attachment may be especially significant for the growth of emotion regulation (Cassidy, 1994; Thompson, 1994; see also Shaver & Mikulincer, this volume). Young children in secure relationships have more sensitive mothers who are accepting of children's positive and negative feelings, and open to talking about intense, disturbing, or confusing feelings with them. Consequently, like the offspring of emotion-coaching parents, securely attached children are likely to become more emotionally self-aware, acquire deeper emotion understanding, and develop a more flexible capacity to manage their emotions appropriate to circumstances. Moreover, the security of the parent–child relationship provides a continuing resource of support on which the child can rely. By contrast, young children in insecure relationships have mothers who are less sensitive and more inconsistently responsive to their feelings, and less likely to be comfortable talking with them about difficult emotional experiences. These children are likely to have a more limited understanding of emotion and to become more easily emotionally dysregulated, especially in stressful circumstances, because of the lack of support in the parent–child relationship.

There is research evidence in support of this view. In a longitudinal study of children's first 3 years, Kochanska (2001) reported that over time, insecurely attached children, compared with secure children, exhibited progressively greater fear and/or anger, and diminished joy, in standardized assessments. Even by age 1, the mothers of secure infants commented on both positive and negative emotions when interacting with their children, while the mothers of insecurely attached infants rarely discussed their feelings or commented primarily about negative emotions (Goldberg, MacKay-Soroka, & Rochester, 1994). By early childhood, securely attached preschoolers talked more about emotions in everyday conversations with their mothers, and their mothers were more richly elaborative than mothers of insecurely attached preschoolers in their discussions of emotion with them. This may help to explain why secure children are more advanced in emotion understanding (Laible & Thompson, 1998; Raikes & Thompson, 2006). There is also evidence that children in secure relationships are better at managing negative emotions. Gilliom and his col-

leagues (2002) reported that boys who were securely attached at age 1½ were observed to use more constructive anger management strategies at age 3½. In a study of the responses of 18-month-olds to moderate stressors, Nachmias and colleagues (1996) reported that postsession cortisol elevations, indicating stressful responding, were found only in temperamentally inhibited toddlers in insecure relationships with their mothers. For inhibited toddlers in secure relationships, the mother's presence helped to buffer the physiological effects of challenging events. In another study, Contreras, Kerns, Weimer, Gentzler, and Tomich (2000) reported that by middle childhood, attachment security was significantly associated with children's constructive coping with stress, and the extent of coping mediated the association between attachment and children's peer competence. Research in our laboratory has also shown that mothers in secure relationships with their children are more accurate in judging what their children are feeling and potentially are thus more capable of providing helpful guidance about managing those feelings (Waters et al., 2010).

These findings indicate that the relational context in which emotion regulation develops is important not only for the specific ways that parents respond to children's feelings but also for the relational support that shapes the growth of emotion self-regulation.

Conclusions

In the broadest sense, the research surveyed in this chapter confirms how significantly social influences shape the growth of emotional experience and emotion regulation. This literature underscores the multifaceted ways that socialization processes in the family affect children's emotional reactivity and self-regulation.

Viewed in terms of bottom-up influences, family processes shape children's emotional reactivity through the everyday emotional demands to which children must adapt, anticipate, and cope. These influences are most apparent in studies of at-risk children, for whom the challenges of family dysfunction, marital conflict, and even maltreatment underscore how emotional development and self-regulation conform to the functional requirements of managing difficult everyday emotional demands at home. Viewed in terms of top-down influences, family processes also shape children's understanding of emotion, and their motivation and competence to manage their feelings. These processes contribute to significant individual differences in children's emotional appraisals, goals for emotion regulation, and strategies for self-control.

Although study of the socialization of emotion regulation has long been a topic of vigorous research interest, a considerable research agenda remains. Continuing work in this field is justified not only for theoretical reasons, but also for its importance to understanding the challenges faced by children at risk for problems of emotion-related psychopathology.

References

Barrett, L. F., & Bar, M. (2009). See it with feeling: Affective predictions during object perception. *Philosophical Transactions of the Royal Society B: Biological Sciences, 364*, 1325–1334.

Blair, C. (2010). Stress and the development of self-regulation in context. *Child Development Perspectives, 4*, 181–188.

Brown, J. R., Donelan-McCall, N., & Dunn, J. (1996). Why talk about mental states?: The significance of children's conversations with friends, siblings, and mothers. *Child Development, 67*, 836–849.

Calkins, S. D., & Johnson, M. C. (1998). Toddler regulation of distress to frustrating events: Temperamental and maternal correlates. *Infant Behavior and Development, 21*, 379–395.

Caspi, A., Moffitt, T. E., Morgan, J., Rutter, M., Taylor, A., Arseneault, L., et al. (2004). Maternal expressed emotion predicts children's antisocial behavior problems: Using monozygotic-twin differences to identify environmental effects on behavioral development. *Developmental Psychology, 40*, 149–161.

Cassano, M. C., & Zeman, J. L. (2010). Parental socialization of sadness regulation in middle childhood: The role of expectations and gender. *Developmental Psychology, 46*, 1214–1226.

Cassidy, J. (1994). Emotion regulation: Influ-

ences of attachment relationships [Special issue]. *Monographs of the Society for Research in Child Development, 59*(2–3, Serial No. 240), 228–249.

Cole, P., Martin, S., & Dennis, T. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development, 75*, 317–333.

Contreras, J. M., Kerns, K. A., Weimer, B. L., Gentzler, A. L., & Tomich, P. L. (2000). Emotion regulation as a mediator of associations between mother–child attachment and peer relationships in middle childhood. *Journal of Family Psychology, 14*, 111–124.

Cummings, E. M., & Davis, P. T. (2010). *Children and marital conflict: An emotional security perspective*. New York: Guilford Press.

Davies, P. T., & Forman, E. M. (2002). Children's patterns of preserving emotional security in the interparental subsystem. *Child Development, 73*, 1880–1903.

Davies, P. T., Harold, G. T., Goeke-Morey, M. C., & Cummings, E. M. (2002). Child emotional security and interparental conflict. *Monographs of the Society for Research in Child Development, 67*(Serial No. 270), 1–127.

Davies, P. T., & Woitach, M. J. (2008). Children's emotional security in the interparental relationship. *Current Directions in Psychological Science, 17*, 269–274.

Denham, S. (1998). *Emotional development in young children*. New York: Guilford Press.

Denham, S., Bassett, H. H., & Wyatt, T. (2007). The socialization of emotional competence. In J. Grusec & P. Hastings (Eds.), *Handbook of socialization* (pp. 614–637). New York: Guilford Press.

Eisenberg, N., Cumberland, A., & Spinrad, T. L. (1998). Parental socialization of emotion. *Psychological Inquiry, 9*, 241–273.

Eisenberg, N., Fabes, R. A., & Murphy, B. C. (1996). Parents' reactions to children's negative emotions: Relations to children's social competence and comforting behavior. *Child Development, 67*, 2227–2247.

Eisenberg, N., Gershoff, E. T., Fabes, R. A., Shepard, S. A., Cumberland, A. J., Losoya, S. H., et al. (2001). Mothers' emotional expressivity and children's behavior problems and social competence: Mediation through children's regulation. *Developmental Psychology, 37*, 475–490.

Eisenberg, N., Valiente, C., Morris, A. S., Fabes, R. A., Cumberland, A., Reiser, M., et al. (2003). Longitudinal relations among parental emotional expressivity, children's regulation, and quality of socioemotional functioning. *Developmental Psychology, 39*, 3–19.

El-Sheikh, M., & Erath, S. A. (2011). Family conflict, autonomic nervous system functioning, and child adaptation: State of the science and future directions. *Development and Psychopathology, 23*, 703–721.

Feldman, R., Greenbaum, C. W., & Yirmiya, N. (1999). Mother–infant affect synchrony as an antecedent of the emergence of self-control. *Developmental Psychology, 35*, 223–231.

Gekoski, M., Rovee-Collier, C., & Carulli-Rabinowitz, V. (1983). A longitudinal analysis of inhibition of infant distress: The origins of social expectations? *Infant Behavior and Development, 6*, 339–351.

Gianino, A., & Tronick, E. (1988). The mutual regulation model: The infant's self and interactive regulation and coping and defensive capacities. In T. Field, P. McCabe, & N. Schneiderman (Eds.), *Stress and coping* (Vol. 2, pp. 47–68). Hillsdale, NJ: Erlbaum.

Gilliom, M., Shaw, D. S., Beck, J. E., Schonberg, M. A., & Lukon, J. L. (2002). Anger regulation in disadvantaged preschool boys: Strategies, antecedents, and the development of self-control. *Developmental Psychology, 38*, 222–235.

Goldberg, S., MacKay-Soroka, S., & Rochester, M. (1994). Affect, attachment, and maternal responsiveness. *Infant Behavior and Development, 17*, 335–339.

Goldsmith, H. H., Pollak, S. D., & Davidson, R. J. (2008). Developmental neuroscience perspectives on emotion regulation. *Child Development Perspectives, 2*, 132–140.

Gottman, J. M., Katz, L. F., & Hooven, C. (1996). Parental meta-emotion philosophy and the emotional life of families: Theoretical models and preliminary data. *Journal of Family Psychology, 10*, 243–268.

Gottman, J. M., Katz, L. F., & Hooven, C. (1997). *Meta-emotion: How families communicate emotionally*. Mahwah, NJ: Erlbaum.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Halberstadt, A. G., Crisp, V. W., & Eaton, K. L.

(1999). Family expressiveness: A retrospective and new directions for research. In P. Philippot & R. S. Feldman (Eds.), *The social context of nonverbal behavior* (pp. 109–155). New York: Cambridge University Press.

Halberstadt, A. G., & Eaton, K. L. (2003). A meta-analysis of family expressiveness and children's emotion expressiveness and understanding. *Marriage and Family Review, 34*, 35–62.

Hooley, J. M., & Richters, J. E. (1995). Expressed emotion: A developmental perspective. In D. Cicchetti & S. Toth (Eds.), *Emotion, cognition, and representation* (pp. 133–166). Rochester, NY: University of Rochester Press.

Hooven, C., Gottman, J. M., & Katz, L. F. (1995). Parental meta-emotion structure predicts family and child outcomes. *Cognition and Emotion, 9*, 229–264.

Katz, L. F., Maliken, A. C., & Stettler, N. M. (2012). Parental meta-emotion philosophy: A review of research and theoretical framework. *Child Development Perspectives, 6*, 417–422.

Kliewer, W., Fearnow, M. D., & Miller, P. A. (1996). Coping socialization in middle childhood: Tests of maternal and paternal influences. *Child Development, 67*, 2339–2357.

Kober, H., Barrett, L. F., Joseph, J., Bliss-Moreau, E., Lindquist, K., & Wager, T. D. (2008). Functional grouping and cortical–subcortical interactions in emotion: A meta-analysis of neuroimaging studies. *NeuroImage, 42*, 998–1031.

Kochanska, G. (2001). Emotional development in children with different attachment histories: The first three years. *Child Development, 72*, 474–490.

Laible, D. J., & Thompson, R. A. (1998). Attachment and emotional understanding in preschool children. *Developmental Psychology, 34*(5), 1038–1045.

Lamb, M. (1981). Developing trust and perceived effectance in infancy. In L. Lipsitt (Ed.), *Advances in infancy research* (Vol. 1, pp. 101–127). New York: Ablex.

Lamb, M., & Malkin, C. (1986). The development of social expectations in distress–relief sequences: A longitudinal study. *International Journal of Behavioral Development, 9*, 235–249.

Loman, M. M., & Gunnar, M. R. (2010). Early experience and the development of stress reactivity and regulation in children. *Neuroscience and Biobehavioral Reviews, 34*, 867–876.

Lunkenheimer, E. S., Shields, A. M., & Cortina, K. S. (2007). Parental emotion coaching and dismissing in family interaction. *Social Development, 16*, 232–248.

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10*, 434–445.

Morris, A. S., Silk, J. S., Morris, M. D. S., Steinberg, L., Aucoin, K. J., & Keyes, A. W. (2011). The influence of mother–child emotion regulation strategies on children's expression of anger and sadness. *Developmental Psychology, 47*, 213–225.

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development, 16*, 361–388.

Nachmias, M., Gunnar, M., Mangelsdorf, S., Parritz, R. H., & Buss, K. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. *Child Development, 67*, 508–522.

Newton, E. K., & Thompson, R. A. (2010). Parents' views of early social and emotional development: More and less than meets the eye. *Zero to Three, 31*, 10–15.

Ochsner, K. N., Ray, R. R., Hughes, B., McRae, K., Cooper, J. C., Weber, J., et al. (2009). Bottom-up and top-down processes in emotion generation: Common and distinct neural mechanisms. *Psychological Science, 20*, 1322–1331.

Pollak, S. D. (2008). Mechanisms linking early experience and the emergence of emotions: Illustrations from the study of maltreated children. *Current Directions in Psychological Science, 17*, 370–375.

Raikes, H. A., & Thompson, R. A. (2006). Family emotional climate, attachment security, and young children's emotion understanding in a high-risk sample. *British Journal of Developmental Psychology, 24*, 989–1004.

Ramsden, S. R., & Hubbard, J. A. (2002). Family expressiveness and parental emotion coaching: Their role in children's emotion regulation and aggression. *Journal of Abnormal Child Psychology, 30*, 657–667.

Repetti, R. L., Robles, T. F., & Reynolds, B. (2011). Allostatic processes in the family. *Development and Psychopathology, 23*, 921–938.

Rogosch, F., Cicchetti, D., & Toth, S. (2004).

Expressed emotion in multiple subsystems of the families of toddlers with depressed mothers. *Development and Psychopathology, 16*, 689–709.

Saarni, C. (1999). *The development of emotional competence*. New York: Guilford Press.

Shaver, P. R., & Mikulincer, M. (this volume, 2014). Adult attachment and emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 237–250). New York: Guilford Press.

Thompson, R. A. (1990). Emotion and self-regulation. In R. A. Thompson (Ed.), *Socio-emotional development: Nebraska Symposium on Motivation* (Vol. 36, pp. 383–483). Lincoln: University of Nebraska Press.

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development, 59*(2–3, Serial No. 240), 25–52.

Thompson, R. A. (2000). Childhood anxiety disorders from the perspective of emotion regulation and attachment. In M. W. Vasey & M. R. Dadds (Eds.), *The developmental psychopathology of anxiety* (pp. 160–182). Oxford, UK: Oxford University Press.

Thompson, R. A. (2006a). The development of the person: Social understanding, relationships, self, conscience. In W. Damon & R. M. Lerner (Eds.), N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 24–98). New York: Wiley.

Thompson, R. A. (2006b). Conversation and developing understanding: Introduction to the special issue. *Merrill–Palmer Quarterly, 52*(1), 1–16.

Thompson, R. A. (2011). Emotion and emotion regulation: Two sides of the developing coin. *Emotion Review, 3*(1), 53–61.

Thompson, R. A., Flood, M. F., & Goodvin, R. (2006). Social support and developmental psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology: Vol. III. Risk, disorder, and adaptation* (2nd ed., pp. 1–37). New York: Wiley.

Thompson, R. A., & Goodman, M. (2010). Development of emotion regulation: More than meets the eye. In A. Kring & D. Sloan (Eds.), *Emotion regulation and psychopathology* (pp. 38–58). New York: Guilford Press.

Thompson, R. A., Lewis, M., & Calkins, S. D. (2008). Reassessing emotion regulation. *Child Development Perspectives, 2*(3), 124–131.

Thompson, R. A., & Meyer, S. (2007). The socialization of emotion regulation in the family. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 249–268). New York: Guilford Press.

Thompson, R. A., Virmani, E., Waters, S. F., Meyer, S., & Raikes, A. (2013). The development of emotion self-regulation: The whole and the sum of the parts. In K. Barrett, N. A. Fox, G. A. Morgan, D. J. Fidler, & L. A. Daunhauer (Eds.), *Handbook of self-regulatory processes in development* (pp. 5–26). New York: Taylor & Francis.

Thompson, R. A., & Waters, S. F. (2010). The development of emotion regulation: Parent and peer influences. In R. Sanchez-Aragon (Ed.), *Emotion regulation: A crossing from culture to the development of personal relationships* (pp. 125–157). Mexico City: National Autonomous University of Mexico.

Thompson, R. A., Waters, S. F., Meyer, S. C., Raikes, A., Jochem, R. A., & Virmani, E. A. (2009, April). Conversational catalysts to developing understanding of emotion regulation. In L. J. Levine (Chair), *How kids keep their cool: Pathways to effective emotion regulation*. Symposium presented at the biennial meeting of the Society for Research in Child Development, Denver, CO.

Valiente, C., Fabes, R. A., Eisenberg, N., & Spinrad, T. L. (2004). The relations of parental expressivity and support to children's coping with daily stress. *Journal of Family Psychology, 18*, 97–106.

Waters, S., Virmani, E., Thompson, R. A., Meyer, S., Raikes, A., & Jochem, R. (2010). Emotion regulation and attachment: Unpacking two constructs and their association. *Journal of Psychopathology and Behavioral Assessment, 32*(1), 37–47.

Woltering, S., & Lewis, M. D. (2009). Developmental pathways of emotion regulation in childhood: A neuropsychological perspective. *Mind, Brain, and Education, 3*, 160–169.

CHAPTER 12

Emotion Regulation in Adolescence

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Adolescents' emotional lives are distinct from those of children or adults: They react more strongly to emotion-eliciting situations (e.g., Miller & Shields, 1980; Stroud et al., 2009), experience negative and mixed emotions more frequently (e.g., Larson & Asmussen, 1991; Larson & Lampman-Petraitis, 1989; Riediger, Schmiedek, Wagner, & Lindenberger, 2009; Riediger, Wrzus, & Wagner, 2012), and fluctuate more rapidly in their emotional states (e.g., Larson, Moneta, Richards, & Wilson, 2002). Emotion regulation is assumed to play a key role in these characteristics of adolescents' emotional experiences (e.g., Opitz, Gross, & Urry, 2012). It has also been proposed to be a central component of adolescents' more general socioemotional adaptation, that is, their ability to adjust to the socioemotional challenges they face (e.g., Silk, Steinberg, & Morris, 2003; Yap, Allen, & Sheeber, 2007). Studying emotion regulation is therefore essential for understanding the developmental phase of adolescence.

In line with the widely accepted definition by Gross (e.g., 1999), we conceive of *emotion regulation* as comprising those deliberate and automatic processes that allow individuals to influence which emotional experiences they have, when they have them, and how they experience and express them. This chapter provides an overview of the current status of research on adolescent emotion regulation in Western societies. To set the stage, we briefly characterize the developmental period of adolescence and the relevance of emotion regulation for adolescents' socioemotional adjustment. The main part of the chapter integrates evidence on three questions that are important for the understanding of emotion regulation in adolescence: Which factors contribute to the development of emotion regulation skills in that life phase? Does adolescents' motivations to regulate their feelings differ from those of other age groups? Which strategies do adolescents use to regulate their emotions, and how adaptive and effective are these strategies? We conclude with an outlook on important future research directions.

Setting the Stage

The life phase "adolescence" refers to the transition from being a child to being an independent adult. It can roughly be defined as beginning with the physical changes of puberty and ending with the assumption of adult social roles. Thus, the particular age range of adolescence varies widely between individuals, depending on the onset of puberty and life circumstances. Empirical

investigations often approximate adolescence as a period within the time window from about 10 to about 25 years of age. In Western industrialized societies, the life phase of adolescence has undergone a major expansion in recent history. Throughout the past century, the average onset of pubertal processes has occurred progressively earlier, particularly in girls. Nowadays, hormonal changes of female puberty begin between 9 and 12 years of age, and most of the physical changes of puberty are typically completed by the midteens. The acceleration of puberty onset has coincided with a progressive delay of assuming adult roles in Western societies. This is often postponed until around the early 20s (for an overview, see Dahl, 2004).

Adolescence is characterized by vast changes in multiple domains of functioning (for an overview, see, e.g., Eccles, Templeton, Barber, & Stone, 2003). Puberty-related hormonal changes, for example, lead to sexual maturation, the development of secondary gender characteristics, and dramatic changes in body size and composition. Adolescence is also a phase of profound psychological change, such as marked growth in cognitive functioning or moral reasoning. Furthermore, many aspects of social experience change as adolescents move away from their family and toward their peers. Importantly, there is large variability in the timing of these various changes, both between adolescents (e.g., regarding the onset of puberty) and within adolescents (i.e., development in one domain of functioning does not necessarily imply parallel development in another; e.g., Dahl, 2004).

During adolescence, emotionally challenging situations typically become more frequent and intense. In Western industrialized societies, for example, this derives from an increased potential for conflict with parents, from adolescents' greater sensitivity to peer-related social interactions, or from early romantic and sexual experiences. In addition, the physical changes of puberty make social demands regarding adolescents' behavior more challenging. Social environments expect adolescents who look like adults to behave like adults (Dahl, 2004). Furthermore, cognitive growth during adolescence provides the basis for greater engagement with emotion-relevant aspects of one's own and others' existence and future (e.g., Thorne, 2004). These challenges can stipulate the development of skills that are necessary for identity formation, autonomy increase, and other developmental tasks of adolescence (Erikson, 1968). This, however, is only true as long as the challenges do not overtax the individual (Yap et al., 2007). Temporary increases in affective reactivity (which we address in greater detail when discussing neurophysiological aspects of emotion regulation in adolescence) may amplify adolescents' vulnerability to being overwhelmed by the emotional challenges they face (e.g., Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Miller & Shields, 1980; Stroud et al., 2009; Sumter, Bokhorst, Miers, Van Pelt, & Westenberg, 2010). In fact, increases during adolescence in internalizing (e.g., depressiveness) and externalizing (e.g., aggressiveness) problems, as well as in various forms of psychopathology (e.g., Allen & Sheeber, 2009; Shortt, Stoolmiller, Smith-Shine, Mark Eddy, & Sheeber, 2010; Silk et al., 2003) demonstrate that developmental demands of adolescence may overstretch the adaptational capacity of vulnerable individuals.

Emotion regulation has been proposed to play a core role in adolescents' ability to weather the developmental challenges they face (e.g., McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011; Silk et al., 2007; Yap et al., 2007). Both over- and underregulation of emotion are assumed to imply risks for adolescents' socioemotional adaptation, that is, for their ability to adjust to the socioemotional challenges they encounter. Available evidence indeed links emotion regulation skills to higher social competence, more prosocial behavior, better academic achievements, and fewer internalizing and externalizing problems (e.g., Bell & Calkins, 2000; Buckley & Saarni, 2009; Silk et al., 2003; Zeman, Cassano, Perry-Parrish, & Stegall, 2006). Emotion regulation has also been related to more peer acceptance, higher peer status, and a lower likelihood of experiencing chronic victimization and bullying by peers (for an overview, see Buckley & Saarni, 2009). In terms of adolescent psychopathology, emotion regulation has been implicated in the development of diverse problems, including anxiety, depressive or conduct disorders, as well as eating disorders in females (e.g.,

McLaughlin et al., 2011; Silk et al., 2007; Sim & Zeman, 2006; Yap et al., 2007).

To date, the vast majority of studies are cross-sectional (but see, e.g., McLaughlin et al., 2011). Thus, whether emotion dysregulation is a risk factor for, or a consequence of adaptational problems in adolescence cannot yet be conclusively anwered. There also is substantial variability across studies in the particular aspects of emotion regulation investigated. Taken together, however, ample evidence links emotion regulation skills to socioemotional adjustment in adolescence. Insight into the development of emotion regulation skills during adolescence can thus have important practical implications. This chapter reviews three fundamental research topics in this respect that refer to influences on emotion regulation in adolescence, emotion regulation motivations, and the ways and effectiveness of adolescents' attempts to regulate emotions.

Influences on Emotion Regulation

The development of emotion regulation skills in adolescence is shaped by the interaction of multiple influences. We focus on two examples in this chapter, namely, individuals' neurophysiological development and their familial context. A discussion of other factors—such as genetic disposition, gender, personality, temperament, attachment style, or influences from developments during infancy and childhood—is beyond the scope of this chapter (but see, e.g., Shaver & Mikulincer, and Mesquita, De Leersnyder, & Albert, this volume; Bariola, Gullone, & Hughes, 2011).

Neurophysiology and Emotion Regulation

Advances in neuroimaging technologies have led to a recent upsurge of interest in the role of neurophysiological development in emotion regulation during adolescence. Research has shown that similar brain regions are activated when participants regulate emotions and when they control cognitive operations (Mauss, Bunge, & Gross, 2007; Mohanty et al., 2007; Ochsner & Gross, 2005). Thus, neurophysiologically oriented research typically ascribes the exertion of cognitive control an essential role for effective emotion regulation (Gray & Braver, 2007; Ochsner & Gross, 2005, this volume). Cognitive control encompasses the various processes that are necessary for successful goal pursuit, which include maintaining a current goal in working memory or shielding it from distraction (Best & Miller, 2010). Intentional emotion regulation can be conceived of as an instance of such goal-directed behavior (e.g., to not express one's anger). Indeed, several studies with participants from various age groups indicate associations between measures of cognitive control and the effectiveness of emotion regulation (e.g., Compton et al., 2008; Hoeksma, Oosterlaan, & Schipper, 2004; Robinson, 2007). For example, Schmeichel and Demaree (2010; Schmeichel, Volokhov, & Demaree, 2008) demonstrated that undergraduates with higher working memory capacity (a facet of cognitive control) were better able to suppress negative emotional expressions and to reappraise stimuli in an unemotional manner.

Cognitive control capacities become increasingly complex and efficient throughout childhood and adolescence and into young adulthood (for review, see Yurgelun-Todd, 2007). Maturation of the prefrontal cortex has been associated with the development of these skills (e.g., Casey, Getz, & Galvan, 2008; Casey et al., 2010; Dahl, 2001, 2004; Steinberg, 2008). Structural maturation includes a decrease in gray matter. This is reflective of synaptic pruning, that is, the elimination of unused neural connections, which possibly enables more focal activation of these brain regions. These structural developments are also associated with functional changes. Studies examining performance on tasks requiring cognitive control (e.g., Stroop or flanker tasks), for example, suggest an age-related increase in the activation of the dorsolateral prefrontal cortex during childhood and adolescence, and a further age-related increase from adolescence to young adulthood in the focalization and efficiency of selective recruitment of cognitive control areas (for reviews, see Casey et al., 2008; Steinberg, 2008). Behavioral studies show that these structural and functional changes are associated with performance improvements in a variety of cognitive tasks from childhood to late adolescence (e.g., Best & Miller, 2010; Forman,

Mäntylä, & Carelli, 2011; Ordaz, Davis, & Luna, 2010).

The pattern of improving cognitive control associated with maturation of the prefrontal cortex suggests that self-regulation competence in general, and emotion regulation competence in particular, should improve from childhood to adulthood (Casey et al., 2010). However, the well-documented peak in impulsivity and risky/reckless behavior in adolescents and emerging adults demonstrates limitations in the ability or willingness to resist temptations and peer influence (see also Luerssen & Ayduk, Grecucci & Salfey, and Cole, this volume) and suggests a nonlinear development of self-regulation competence (Steinberg, 2008). The temporary increase in negative emotionality (e.g., Ciarrochi, Heaven, & Supavadeeprasit, 2008) and affective instability (e.g., Larson et al., 2002) during adolescence gives rise to the assumption that a similar nonlinear development is characteristic of emotion regulation skills as well.

Several contemporary positions on adolescent brain development conclude that an exclusive focus on prefrontal cognitive control areas is insufficient for understanding the neurophysiological correlates of adolescent emotion regulation. These models propose that it is also necessary to take into consideration subcortical regions involved in the processing of emotional information, as well as the coordination between these and prefrontal regions (e.g., Casey et al., 2010; Dahl, 2001; Steinberg, 2008). Central to these models is the assumption that structural and functional maturation of these various brain systems do not occur in parallel. For example, subcortical regions critical to affective processing are assumed to mature earlier in adolescence than cortical regions subserving cognitive control. The differential timing of maturation in different brain regions is expected to result in a temporarily increased disjunction between younger adolescents' affective experiences and their emotion regulation abilities. This disjunction is smaller during childhood, when both systems are still developing, and becomes smaller again during adulthood, when both systems have fully matured. The assumption of differential developmental timing in prefrontal and subcortical regions is consistent with nonhuman primate and human postmortem studies but needs further empirical confirmation (e.g., Steinberg, 2008). Furthermore, Pfeifer and Allen (2012) recently criticized this assumption as being based on an overly simplifying conception of structure–function mappings in the brain. Development of the prefrontal cortex, for example, may also be associated with more emotional understanding and complexity, which in turn could be related to emotional experiences. In addition, while neurophysiological maturation undeniably plays a central role in emotion regulation in adolescence, external influences, to which we turn next, are also important for those learning processes.

Familial Contexts and Emotion Regulation

Emotion regulation abilities are fundamentally shaped by the continual interactions between individuals and their social environments. On the one hand, demands on emotion regulation skills are often particularly high in social situations (Bell & Calkins, 2000). Individuals are required to comply with sociocultural norms and expectations regarding appropriate emotional experiences and expressions in social situations. Furthermore, attainment of situational goals would often not be possible if individuals were not able to regulate their emotions effectively (e.g., to control their anxiety or anger appropriately; Thompson, 1994).

On the other hand, social contexts also present important influences that facilitate or hinder emotion regulation skills (Bariola et al., 2011). Most of the available research focuses on the respective role of familial contexts. Morris, Silk, Steinberg, Myers, and Robinson (2007), for example, propose that family contexts influence the development of emotion regulation during childhood and adolescence in three important ways: through observation learning (e.g., when parents' own emotion regulation serves as a social role model for their offspring), through parenting practices and explicit instruction (e.g., when parents coach emotion regulation strategies), and through the emotional climate in the family (e.g., when parental behaviors and attitudes toward the offspring's emotional expressions and

experiences reinforce or discourage emotion regulation).

Empirical research on the role of parental influences on the emotion regulation of offspring has typically focused on families with infants or children (see Thompson, this volume). Fewer investigations are available on the role of familial contexts for emotion regulation in adolescence. This work suggests that parents' emotion–socialization behaviors continue to be important when their children become adolescents. This is the case even though adolescents' orientation toward more autonomy increases the relative importance of extrafamilial influences, such as peers, media, neighborhood, or culture, as socialization agents of emotion regulation (Bariola et al., 2011). Shortt and colleagues (2010), for example, found that maternal emotion coaching (e.g., approaching the adolescent when he or she is upset to talk about the situation and experienced emotions) was associated with fewer difficulties of their 10- to 13-year-old children in regulating anger. Better anger control, in turn, was associated with fewer externalizing problems of the adolescent. Maternal emotion coaching in this study also predicted fewer externalizing problems of the adolescent 3 years later (i.e., at ages 13–16). Sheeber, Allen, Davis, and Sorensen (2000) found that maternal approval or affirmation of adolescent depressive behaviors was predictive of longer episodes of negative affect on the part of the adolescent (ages 12–19 years). The authors assumed that this reflects a poorer ability to regulate negative affect. Yap, Allen, and Ladouceur (2008) found a relationship between self-reported maternal tendencies to be restrictive and unaccepting of positive affective displays and adolescents' use of maladaptive emotion regulation strategies in conflict situations. More restrictive maternal tendencies were associated with a higher tendency among early adolescents to use maladaptive strategies, such as venting or dysregulated expression of negative affect, which in turn were associated with more depressive symptoms.

Evidence also suggests that emotion–socialization styles in the family need to be adjusted to the age and developmental status of the offspring. Direct intervention in the form of soothing or directive instructions is effective in children at younger ages. For adolescents, however, indirect influences, such as talking about possible emotion regulation strategies, fit better with their cognitive and self-regulation competencies, and their increased need for autonomy (Bell & Calkins, 2000; Morris et al., 2007). Thus, parental balancing of the adolescent's opposing needs for autonomy on the one hand, and for guidance and structure on the other is particularly important for emotion–socialization in adolescence. Imbalance in either direction can have potentially disadvantageous effects on adolescents' socioemotional adaptation. Adolescents who remain overly dependent on their parents appear to be at a higher risk of internalizing problems (e.g., depression; for review, see Morris et al., 2007). Lack of emotion regulation skills has been proposed to play a mediating role in this regard (for review, see Bell & Calkins, 2000). In contrast, adolescents who lack or refuse emotional guidance from their parents seem to be at a higher risk for externalizing problems (e.g., rule breaking and delinquency, substance use, aggression). These behaviors, again, have been associated with a lack of skills to regulate emotions, particularly anger (for review, see Morris et al., 2007).

Interaction between Internal and External Factors

Evidence suggests that a complex set of internal and external factors contributes to the development of emotion regulation skills during adolescence. We illustrated this using the examples of individuals' neurophysiological development, which represents an essential internal factor, and their familial context, which can provide significant external influences on emotion regulation. Importantly, the effects of these internal and external factors are not unidirectional. They reciprocally shape each other. Familial influences on emotion regulation competence, for example, are modulated by the propensity of adolescents to experience and express intense affect. The latter, as discussed earlier, can in turn be related to a temporary dissociation of neurophysiological changes yielding increased emotional reactivity at a time when control functions have not yet fully developed. In a parent–adolescent interaction study, Schulz, Waldinger,

Hauser, and Allen (2005) indeed showed that parents were less positively engaged and showed more hostility when their adolescent children fully expressed their emotions than when they controlled their emotion expressions. Other studies also indicate that parents may have difficulties in coping with adolescents' uninhibited displays of intense emotions, and that these parental difficulties can shape emotion coaching and the emotional climate in the family, and thus feed back to the adolescent (e.g., Dishion, Nelson, & Bullock, 2004).

Taken together, the research reviewed so far suggests that interactions among a complex set of internal and external factors are associated with the development of emotion regulation skills during adolescence. Structural and functional changes of the prefrontal cortex subserving cognitive control abilities might facilitate cognitive emotion regulation skills throughout adolescence. Simultaneous increases in affective reactivity associated with the development of subcortical regions, however, might overtax these evolving regulatory abilities. Such neurobiological factors could interact with aspects of the individual's social context in promoting or hindering the development of emotion regulation skills.

Emotion Regulation Motivation

To date, only little attention has been paid to the fact that emotion regulatory behaviors are preceded and fundamentally shaped by motivational processes (see John & Eng, this volume). One reason for this is that most investigators seem to assume that emotion regulation is inevitably "prohedonic," that is, always directed at optimizing one's well-being. Only recently has awareness arisen that occasional exceptions are possible, that is, that emotion regulation can sometimes be "contrahedonic." People may occasionally be inclined to dwell on or intensify negative emotional experiences, such as anger or sadness, or to lessen positive ones, such as pride or amusement (e.g., Erber & Wang Erber, 2000; Riediger et al., 2009; Tamir, 2009).

Various experience-sampling studies reported by Riediger and colleagues demonstrated adolescent peaks in the prevalence of contra-hedonic motivation in samples ranging in age from 12 years to late adulthood (Riediger et al., 2009; Riediger, Wrzus, & Wagner, 2013). Participants reported their momentary emotional experiences, on average 54 times over 3 weeks, and whether they momentarily wanted to influence their feelings. Contrahedonic motivation (wanting to maintain or enhance negative affect, or to dampen positive affect) was considerably less prevalent than prohedonic motivation (wanting to maintain or enhance positive affect, or to dampen negative affect) in all investigated age groups. There were, however, pronounced age differences. Adolescents reported contrahedonic motivation most frequently, namely, in about 25% of the measurement occasions. There were steep decreases in the prevalence of contrahedonic motivation between the adolescent and the young adult subsamples, and a further decline throughout the adult subsamples into old age. Prohedonic motivation, in contrast, showed an opposite prevalence pattern. It was least prevalent among adolescent and young adult participants, and most prevalent in later adulthood (Riediger et al., 2009, 2013).

Furthermore, analyses of associations with within-person fluctuations in working memory capacity indicated that contrahedonic motivation was more cognitively demanding than prohedonic motivation. The more contrahedonic motivation participants reported, the lower their momentary working memory capacity. Prohedonic motivation, in contrast, was only weakly associated with fluctuations in working memory. This was the case irrespective of participants' ages. Therefore, despite the pronounced age-related differences in the prevalence of different affect regulation motivations, their cognitive requirements appeared to be independent of the individual's age (Riediger, Wrzus, Schmiedek, Wagner, & Lindenberger, 2011).

Two complementary theoretical perspectives on possible reasons for contrahedonic motivation are currently being discussed in the literature. From an instrumental perspective, it has been argued that there may be situations in which negative affect is useful, or when positive affect is disadvantageous for the individual (e.g., Tamir, 2009). Anger, for example, can help to assert one's interests in an argument, whereas expressions of joy can be inappropriate when attending a funeral. Contrahedonic orientation may therefore

derive from people being (consciously or unconsciously) strategic in seeking affective states that are instrumental in a given context (Ford & Tamir, 2012). Supporting this position, Riediger and colleagues (2009) found contrahedonic motivation to be less strongly related to people's current emotional experiences than prohedonic orientations. That is, while participants tended to report that they wanted to maintain positive affect when it was high, to enhance positive affect when it was low, and to dampen negative affect when it was high, contrahedonic orientations were less strongly associated with participants' momentary affect. This is consistent with the idea that contrahedonic motivation may serve instrumental functions that are not necessarily related to the individual's current affect. The authors speculated that the relatively higher prevalence of contrahedonic motivation in adolescence might reflect an instrumental value in tackling the developmental tasks of that life phase. Repudiating prevailing hedonic conventions, for example, might put adolescents in situations where they have to deal with negative emotional experiences. This, in turn, might help them to establish emotional autonomy from their parents, affirm a sense of maturity, develop their sense of identity, or refine their self-regulatory competencies. To date, however, these assumptions have not yet been empirically verified.

A complementary account of possible reasons for contrahedonic motivation is the mixed-affect perspective. It proposes that contrahedonic motivation may also arise when apparently negative emotional states are accompanied or followed by positive experiences, that is, when the emotional episode is mixed (Andrade & Cohen, 2007; Riediger et al., 2009). Such mixed affective experiences might motivate individuals, for example, to seek or maintain a given negative affective state because of the positive aspects they associate with it. In fact, the experience sampling studies by Riediger and colleagues demonstrated that mixed affective experiences in everyday life were related to participants' being more likely to report contrahedonic orientations. In addition, these studies also showed pronounced age-related differences in the prevalence of mixed-affective experiences that followed the same pattern as those of contrahedonic motivation: Both mixed affective experiences and contrahedonic motivation were most prevalent among adolescent participants and least prevalent among older adults (Riediger et al., 2009, 2013).[1] Importantly, this pattern of age-related differences was not restricted to participants' self-report; it was also reflected in implicit representations of affect valence, as assessed with implicit association tests. Compared to adults from various age groups, adolescents associated positive affect least distinctively with pleasantness (vs. unpleasantness) and unhappiness least distinctively with unpleasantness (vs. pleasantness). The older the participants, however, the more differentiated their representations of the valence of affective states. Furthermore, the less differentiated people's mental representations of affect valence, the more likely they were to report mixed affect and contrahedonic motivation in their everyday lives (Riediger et al., 2013). Although causal conclusions are not possible given the correlational nature of these studies, these findings are in line with the mixed affective perspective. Thus, the comparatively higher prevalence of mixed affective experiences in adolescence could be among the factors that contribute to a comparatively higher prevalence of self-reported contrahedonic motivation in that age group. This seems to be associated with relatively more undifferentiated mental representations of the valence of affective states in adolescence.

Emotion Regulation Strategies

The research reviewed so far demonstrates that internal and external influences on emotion regulation abilities undergo profound changes during childhood, adolescence, and into young adulthood, and that adolescents' emotion regulation motivation differs from those of adults. But what are the consequences? Are there age-related differences in the strategies individuals use to regulate their emotions, or in their emotion regulation effectiveness? An integrated and interpretable body of research addressing this question has yet to emerge. Only few studies have investigated cross-sectional age differences within or across developmental periods, and longitudinal evidence on change within persons over time is scarce. These studies differ widely in their conceptualization of regulatory strategies. This is

partly due to the fact that these studies were conducted either in the research tradition of emotion regulation or (more frequently) in that of coping, with the latter including emotion regulation as one of many ways of dealing with stress (Compas, 2009; Gross, 1999). Despite considerable overlap in research interests, there has been little cross-talk between these two traditions.

Research within the emotion regulation tradition has most typically proceeded from the process model of emotion regulation (e.g., Gross, 1999), which distinguishes antecedent-focused strategies (directed at changing the emotional input prior to the actual emotional experience) from response-focused strategies (directed at modifying emotional responses after they have been elicited). Most research has centered around two prototypical examples, namely, "cognitive reappraisal" (i.e., changing one's thinking about an emotion-eliciting situation or about the capacity to manage it) as an instance of antecedent-focused emotion regulation, and "expressive suppression" (i.e., inhibiting the outward expression of an emotional experience) as an instance of response-focused emotion regulation.

More research on regulatory strategies in adolescence has been conducted within the coping research tradition, which views emotion regulation as one of various ways of dealing with stress. These studies, however, often referred to different classification systems of regulatory strategies, making an integration of findings difficult. Two widely used classification systems are the primary–secondary control model (e.g., Rothbaum, Weisz, & Snyder, 1982) and the ways-of-coping model (e.g., Lazarus & Folkman, 1984). The primary–secondary control model distinguishes regulatory strategies according to their underlying goals. The aim of primary coping is to influence characteristics of the stress-eliciting conditions, whereas the aim in secondary coping is to adapt oneself to the conditions as they are. The ways-of-coping model, in contrast, distinguishes regulatory strategies according to their respective target. Problem-focused coping refers to modifying the source of stress, whereas emotion-focused coping refers to modifying the elicited emotional response. Primary and secondary coping, as well as problem-focused coping, partly overlap with Gross's (1999) conceptualization of antecedent-focused emotion regulation. Central to these strategies is that they are directed at factors influencing the process of emotion generation. Emotion-focused coping, in contrast, overlaps with response-focused emotion regulation in Gross's model, as both conceptualizations refer to strategies directed at modifying an emotional response after it has been activated.

Use of Emotion Regulation Strategies

Zimmer-Gebeck and Skinner (2011) integrated findings from 58 studies comparing coping strategies within the age range from childhood to adolescence. Overall, the authors concluded that age differences in regulatory behaviors are characterized by two trends.

First, there appears to be an age-graded increase in regulatory capacities. This is reflected in not only an increased understanding of emotional situations (Labouvie-Vief, DeVoe, & Bulka, 1989) but also a broader and more sophisticated repertoire of regulatory strategies. For example, from childhood to adolescence, instrumental action in response to stressors appears to become gradually supplemented by planful problem solving. Furthermore, distraction tactics seem to become more diverse and increasingly include cognitive distraction, in addition to behavioral distraction. Also, compared to children, adolescents appear to be better able to attend to and reflect on their own emotional states. They also seem to use progressively more sophisticated cognitive strategies to deal with emotions, such as positive self-talk and reappraisal.

The second developmental trend from childhood to adolescence, according to the analysis by Zimmer-Gebeck and Skinner (2011), reflects age-related improvement in tailoring regulatory attempts to the situation. That is, adolescents seem to become increasingly able to engage in those regulatory strategies that are most effective in dealing with particular kinds of stressors. For example, evidence suggests increasing use of both problem solving to deal with modifiable difficulties (e.g., in school or sports) and distraction to deal with uncontrollable stressors (e.g., parental illness) from childhood to adolescence. Another example derives from a longitudinal study on anger regulation among friends. Salisch

and Vogelgesang (2005) observed a decline from older childhood to adolescence in individuals' self-reported use of confrontation and harming, redirection of attention, and ignoring and self-blame when dealing with situations involving anger toward a friend. The authors also found an increase in participants' use of explanation and reconciliation in such situations.

Zimmer-Gembeck and Skinner (2011) also emphasized that the apparent improvement in emotion regulatory competence does not necessarily need to be linear. Some of the reviewed evidence points to temporarily increased struggles with regulation in response to stressors during the transition from late childhood to early adolescence. Compared to older adolescents, for example, younger adolescents have occasionally shown lower levels of help-seeking behaviors and effort expenditure in domains in which this would be helpful (e.g., regarding school-related stressors). The authors also reviewed evidence of a temporary rise in the use of potentially more maladaptive regulation strategies during early adolescence, such as cognitive escape, rumination, verbal aggression, or venting.

Most of the extant research investigated the age range from childhood to adolescence. Only a few available studies compare the use of regulatory strategies in adolescence and adulthood. Overall, this research suggests continued change into young adulthood (e.g., Blanchard-Fields & Coats, 2008; Garnefski, Legerstee, Kraaij, van den Kommer, & Teerds, 2002). Garnefski and colleagues, for example, found that adolescents around 13 years of age reported using various cognitive change strategies (e.g., positive reappraisal or refocus on planning) significantly less frequently than did adults of various age groups.

Effects of Emotion Regulation Strategies: Adaptiveness and Effectiveness

Most studies on the effects of emotion regulation strategies proceed from the idea that it is meaningful to conceive of an adolescent's regulatory style as a trait. These studies characterize adolescents according to their overarching tendency to use particular types of regulatory strategies more than others. The focus is on the adaptiveness of different regulatory styles, as characterized by their association with self- or other-reports of various indicators of psychological adjustment, such as emotional or behavioral problems, psychosomatic health, subjective well-being, and social or academic competence.

Within the research tradition on emotion regulation, investigations of the adaptiveness of specific regulatory strategies have most typically focused on reappraisal and expressive suppression. Investigations with young adults suggest that habitual use of reappraisal is associated with a healthier profile of socioemotional adjustment than is habitual use of expressive suppression, and that the latter is also associated with greater cognitive and physiological costs (for an overview, see Gross, 2002). Investigations in adolescent samples are rare, but they have yielded similar patterns of findings (Betts, Gullone, & Allen, 2009; Hughes, Gullone, Dudley, & Tonge, 2010).

More research on the adaptiveness of regulatory styles in adolescence has been conducted in the research tradition on coping. Compas and colleagues (2001) reviewed 63 of these studies and concluded that the majority of the studies demonstrate that problem-focused and engagement coping (which overlap with antecedent-focused emotion regulation) were associated with better psychological adjustment during childhood and adolescence. The specific subtypes most consistently associated with better adjustment included the generation of positive and hopeful thoughts (i.e., cognitive reappraisal), careful analysis of the stressful situation, and selective attention to positive aspects of the situation. In contrast, disengagement and emotion-focused coping (which overlap largely with response-focused emotion regulation) were associated with poorer psychological adjustment in most studies. However, it was not focusing on one's emotions in general that was related to lower psychological adjustment. Rather, these effects were due to disengagement from the stressor or from one's emotions (e.g., expressive suppression, avoidance, or withdrawal), negative cognitions about the self and the situation, and unregulated release or ventilation of emotions.

Compas and colleagues (2001) also emphasized that notable differences in results across studies may be associated with the nature of the investigated stressor, par-

ticularly its controllability. This is consistent with the view that an adaptive regulatory style may not be characterized primarily by more frequent use of particular regulation strategies relative to others, but by a high level of flexibility (e.g., Gross, 1999). This flexibility should be evident in positive associations between socioemotional adaptation and adolescents' ability to tailor specific regulation attempts to the nature (e.g., the controllability) of a given situation. While this claim seems intuitively appealing, future research needs to strengthen the empirical foundation supporting it.

The research on the adaptiveness of affect regulation styles takes a person-centered (i.e., trait-oriented) approach. Another approach is process-centered (i.e., state-oriented) and focuses on how effective individuals actually are in influencing their emotional experiences in intended ways. Hardly any evidence is available on the effectiveness of emotion regulation in adolescence. This is probably due to the methodological challenges involved in operationalizing emotion regulation effectiveness. In fact, there is an ongoing debate whether and to what degree it is possible to disentangle emotion regulation from the respective emotional experience. While some researchers regard emotion regulation as being experientially and structurally indistinguishable from the emotional experience, others claim that it is possible and meaningful to differentiate between emotion and emotion regulation (for an overview of positions, see, e.g., Matarazzo, 2008).

In line with the latter position, emotion regulation effectiveness in adolescence has recently been addressed from two different perspectives. One focuses on the effectiveness of particular emotion regulation strategies in adolescence. Here, adolescents' use of regulatory strategies is not conceived of as a trait but as a process that can fluctuate within persons over time. The covariation of this process with fluctuations in affective experiences is of particular interest. Reijntjes, Stegge, Terwogt, Kamphuis, and Telch (2006), for example, experimentally manipulated experiences of social rejection during an ostensible computer game in 10- to 13-year-olds. Contrary to what one would expect from research on the adaptiveness of regulatory styles, problem-focused engagement in this study was unrelated to mood improvement after social rejection. In contrast, better mood improvement was associated with more behavioral distraction and less passive behaviors. Silk and colleagues (2003) used experience sampling in 12- to 17-year-old participants to investigate the role of regulatory strategies for mood dynamics after participants had encountered negative events in their daily lives. When adolescents used disengagement-related strategies in response to subjectively severe negative events, or when they reacted involuntarily (e.g., by ruminating or acting impulsively), they maintained higher levels of negative affect over time, suggesting limited effectiveness of these strategies. Engagement in primary and secondary control attempts, however, was unrelated to the dynamics of negative affect. The latter, again, is not consistent with what one would expect based on the majority of studies on associations between regulatory styles and more general outcomes of socioemotional adaptation. Differences in findings across studies on the effectiveness versus the adaptiveness of regulatory strategies might be attributable to differences in assessment approaches (process- vs. person-centered) and/or in the investigated emotion-eliciting situations. Future research is necessary to better integrate the findings from these two lines of research.

A second perspective on emotion regulation effectiveness in adolescence focuses on respective age-related differences. In two recent investigations (McRae et al., 2012; Silvers et al., 2012), participants between 10 and 23 years of age were presented neutral and aversive picture stimuli from the International Affective Picture System (IAPS). Viewing instructions varied across studies but generally included a reappraisal condition (e.g., thinking about the picture in a way that makes one feel less negative) and a comparison condition (e.g., reacting naturally to the picture, but not reappraising). After viewing each picture, participants rated their current negative affect. Regulation effectiveness was determined as the relative decrease in negative affect after viewing aversive stimuli in the reappraisal versus the comparison condition. Silvers and colleagues observed an age-related increase in regulation effectiveness from late childhood

to late adolescence that tapered off in young adulthood. McRae and colleagues (2012), in contrast, found relative age invariance in regulation effectiveness from ages 10 to 17 years, but an increase in effectiveness from late adolescence to young adulthood. These inconsistencies in findings might be due to methodological differences between the studies. Procedures in both studies varied, for example, in the selection of stimuli for the analyses, and in whether or not participants were trained in the reappraisal task. More empirical investigations are necessary to clarify and expand the empirical picture on emotion regulation effectiveness throughout adolescence.

Wrapping Up and Looking Forward

Adolescence is characterized by an upsurge in the frequency and intensity of emotional challenges, in a time of life when individuals have to manage such challenging situations more and more independently. This provides developmental stimulation, but it can also increase vulnerability to adaptational difficulties. Emotion regulation has been proposed to play an important role in how adolescents adapt to these challenges. Empirical investigations of this claim vary considerably regarding their operationalizations of what "better" emotion regulation entails. Systematic comparisons of implications of different facets of emotion regulation for adolescents' socioemotional adaptation are still lacking. In addition, causal relations between emotion regulation and developmental adaptation need more systematic investigation in the future. Another open task for the future is a more rigorous investigation of possible interindividual differences in adaptational outcomes of emotion regulation. There are, for example, indications that outcomes may differ between male and female adolescents (Bowie, 2010; Perry-Parrish & Zeman, 2011).

In addition to assuming implications of emotion regulation for adolescents' socioemotional adjustment in general, it has also been proposed that emotion regulation is involved in the unique emotional lives of adolescents in particular. Compared to children and adults, adolescents report more negative, more mixed, and more rapidly varying affective states. Empirical evidence that directly links emotion regulation to the characteristic emotional experiences of adolescents is rare. Providing such evidence represents an important undertaking for future studies. Knowledge about emotion regulation in adolescence can thus have important practical implications. We believe that an integration of findings from three research areas—influences on the development of emotion regulation, emotion regulation motivation, and emotion regulation strategies—is crucial for a better understanding of adolescents' emotion regulation. This chapter has reviewed contemporary positions within each of these areas of inquiry.

A complex set of internal and external factors contribute to the development of emotion regulation skills during adolescence. A temporal dissociation in the maturation of prefrontal and subcortical brain regions, for example, is assumed to result in a temporary decrease in emotion regulation capacity when cognitive control capacities have not yet sufficiently improved to provide adequate control of the progressively more active emotion system. It seems plausible that this may be one of the factors contributing to the unique quality of emotional experiences observed during adolescence. However, predictions about relations between neurophysiological changes during adolescence and adolescents' development of emotion regulation abilities are still in need of systematic empirical verification. So far, little is known about the possibility that different emotion regulation strategies might vary in terms of how much cognitive control they require, and that people can recruit external resources when internal resources are insufficient for effective emotion regulation (e.g., by asking somebody else to distract oneself from emotion-eliciting thoughts when attempts of self-initiated distraction are not successful, Opitz et al., 2012). Furthermore, emotion regulation skills in adolescence are also influenced (i.e., facilitated or impaired) by many other factors, such as varying degrees of parental structure and supervision. Future research needs to provide more empirical evidence on the interplay of these various internal and external factors to arrive at a more integrated understanding of influences on emotion regulation in adolescence.

In comparison to adults from various age groups, adolescents have been found to report more contrahedonic and less prohedonic motivation. This suggests that part of the negative emotionality that is characteristic of adolescence may be intentionally sought and maintained by the individual. Many questions, however, still remain open. For example, future research will need to employ well-controlled experiments to disentangle the causal mechanisms involved in these associations. This will also contribute to a better understanding of the reasons for adolescent patterns of pro- and contrahedonic orientations. Another open question pertains to potential functions of contrahedonic motivation, for example, related to the development of autonomy, self-regulatory skills, or other aspects of socioemotional development of adolescents. Finally, relations between adolescents' emotion regulation motivation and their actual engagement in emotion regulatory behaviors, as well as the effectiveness of these emotion regulation attempts, need to be explored in future investigations.

Available research on age-related differences in the use, adaptiveness, and effectiveness of adolescents' strategies for regulating their emotions differs widely in the conceptual frameworks employed. This leads to difficulty in deriving a cohesive picture about possible developmental changes during adolescence and adjacent developmental periods. Tentatively, however, this research suggests an age-related increase in regulation competence from childhood to adolescence, as reflected in an increasingly sophisticated understanding of emotional situations and a broadening repertoire of regulatory strategies. This appears to coincide with an age-related increase in the ability to tailor regulatory strategies to the specific requirements of the emotion-eliciting situation. Future research should further clarify the complex empirical picture on adolescent development in the use and adaptiveness of regulatory strategies. Meta-analyses of the available empirical evidence would be helpful in this respect. Furthermore, more empirical evidence is needed on differences in the actual effectiveness of regulatory attempts between children, adolescents, and adults.

The three themes on emotion regulation development in adolescence reviewed in this chapter currently represent relatively independent bodies of research. A more explicit integration of these various research perspectives will help us to arrive at a more integrated and interpretable picture of the development of emotion regulation during adolescence. In addition, future research is necessary to address various limitations of the currently available research. For example, more longitudinal evidence is necessary to portray developmental changes within persons as they move from childhood to adolescence and young adulthood. Such portrayals should also take into account more explicitly the possibility of nonlinear trends in development, such as temporary perturbations or deviations from growth trajectories. Furthermore, in addition to adolescents' chronological ages, the role of their pubertal status (which can vary substantially by age) should be taken into consideration more comprehensively, because affective processes during adolescence are strongly associated with puberty-related developments (Steiner, Dunn, & Born, 2003). Finally, multimethodological enrichment of research approaches would be desirable to overcome the methodological limitations associated with the currently prevailing dominance of self-report approaches in most research on emotion regulation in adolescence.

Note

1. Note that research on age differences in the frequency of mixed affect during adulthood has yielded inconclusive findings. Results from these studies range from age-related decrease, to age invariance, to age-related increase in the propensity to experience mixed affect. Heterogeneity between studies regarding the operationalization of mixed affect may be among the reasons for the inconclusiveness of findings with this measure (for a review, see Riediger & Rauers, in press).

References

Allen, N., & Sheeber, L. (Eds.). (2009). *Adolescent emotional development and the emergence of depressive disorders*. New York: Cambridge University Press.

Andrade, E. B., & Cohen, J. B. (2007). On the

consumption of negative feelings. *Journal of Consumer Research, 34*, 283–300.

Bariola, E., Gullone, E., & Hughes, E. K. (2011). Child and adolescent emotion regulation: The role of parental emotion regulation and expression. *Clinical Child and Family Psychology Review, 14*, 198–212.

Bell, K. L., & Calkins, S. D. (2000). Relationships as inputs and outputs of emotion regulation. *Psychological Inquiry, 11*, 160–163.

Best, J. R., & Miller, P. H. (2010). A developmental perspective on executive function. *Child Development, 81*, 1641–1660.

Betts, J., Gullone, E., & Allen, J. S. (2009). An examination of emotion regulation, temperament, and parenting style as potential predictors of adolescent depression risk status: A correlational study. *British Journal of Developmental Psychology, 27*, 473–485.

Blanchard-Fields, F., & Coats, A. H. (2008). The experience of anger and sadness in everyday problems impacts age differences in emotion regulation. *Developmental Psychology, 44*, 1547–1556.

Bowie, B. H. (2010). Understanding the gender differences in pathways to social deviancy: Relational aggression and emotion regulation. *Archives of Psychiatric Nursing, 24*, 27–37.

Buckley, M., & Saarni, C. (2009). Emotion regulation: Implications for positive youth development. In R. Gilman, E. S. Huebner, & M. J. Furlong (Eds.), *Handbook of positive psychology in schools* (pp. 107–118). New York: Routledge.

Casey, B., Getz, S., & Galvan, A. (2008). The adolescent brain. *Developmental Review, 28*, 62–77.

Casey, B., Jones, R. M., Levita, L., Libby, V., Pattwell, S. S., Ruberry, E. J., et al. (2010). The storm and stress of adolescence: Insights from human imaging and mouse genetics. *Developmental Psychobiology, 52*, 225–235.

Ciarrochi, J., Heaven, P. C., & Supavadeeprasit, S. (2008). The link between emotion identification skills and socio-emotional functioning in early adolescence: A 1-year longitudinal study. *Journal of Adolescence, 31*, 565–582.

Cole, S. W. (this volume, 2014). Emotion regulation and gene expression. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 571–585). New York: Guilford Press.

Compas, B. E. (2009). Coping, regulation, and development during childhood and adolescence. *New Directions for Child and Adolescent Development, 2009*, 87–99.

Compas, B. E., Connor-Smith, J. K., Saltzman, H., Thomsen, A. H., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin, 127*, 87–127.

Compton, R. J., Robinson, M. D., Ode, S., Quandt, L. C., Fineman, S. L., & Carp, J. (2008). Error-monitoring ability predicts daily stress regulation: Research article. *Psychological Science, 19*, 702–708.

Dahl, R. E. (2001). Affect regulation, brain development, and behavioral/emotional health in adolescence. *CNS Spectrums, 6*, 60–72.

Dahl, R. E. (2004). Adolescent brain development: A period of vulnerabilities and opportunities. *Annals of the New York Academy of Sciences, 1021*, 1–22.

Dishion, T. J., Nelson, S. N., & Bullock, B. M. (2004). Premature adolescent autonomy: Parent disengagement and deviant peer process in the amplification of problem behavior. *Journal of Adolescence, 27*, 515–530.

Eccles, J. S., Templeton, J., Barber, B., & Stone, M. (2003). Adolescence and emerging adulthood: The critical passage ways to adulthood. In M. H. Bornstein, L. Davidson, C. L. M. Keyess, & K. A. Moore (Eds.), *Well-being: Positive development across the life course* (pp. 383–406). Mahwah, NJ: Erlbaum.

Erber, R., & Wang Erber, M. (2000). The self-regulation of moods: Second thoughts on the importance of happiness in everyday life. *Psychological Inquiry, 11*, 142–148.

Erikson, E. E. (1968). *Identity: Youth and crisis*. New York: Norton.

Ford, B. Q., & Tamir, M. (2012). When getting angry is smart: Emotional preferences and emotional intelligence. *Emotion, 12*, 685–689.

Forman, H., Mäntylä, T., & Carelli, M. G. (2011). Time keeping and working memory development in early adolescence: A 4-year follow-up. *Journal of Experimental Child Psychology, 108*, 170–179.

Garnefski, N., Legerstee, J., Kraaij, V., van den Kommer, T., & Teerds, J. (2002). Cognitive coping strategies and symptoms of depression and anxiety: A comparison between adolescents and adults. *Journal of Adolescence, 25*, 603–611.

Gray, J. R., & Braver, T. S. (2007). Integration of emotion and cognitive control: A neurocomputational hypothesis of dynamic goal regulation. In S. C. Moore & M. Oaksford (Eds.),

Emotional cognition: From brain to behavior (pp. 289–316). Amsterdam: Benjamins.

Grecucci, A., & Sanfey, A. G. (this volume, 2014). Emotion regulation and decision making. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed.). New York: Guilford Press.

Gross, J. J. (1999). Emotion regulation: Past, present, and future. *Cognition and Emotion, 13*, 551–573.

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*, 281–291.

Gunnar, M. R., Wewerka, S., Frenn, K., Long, J. D., & Griggs, C. (2009). Developmental changes in hypothalamus–pituitary–adrenal activity over the transition to adolescence: Normative changes and associations with puberty. *Development and Psychopathology, 21*, 69–85.

Hoeksma, J. B., Oosterlaan, J., & Schipper, E. M. (2004). Emotion regulation and the dynamics of feelings: A conceptual and methodological framework. *Child Development, 75*, 354–360.

Hughes, E. K., Gullone, E., Dudley, A., & Tonge, B. (2010). A case control study of emotion regulation and school refusal in children and adolescents. *Journal of Early Adolescence, 30*, 691–706.

Labouvie-Vief, G., DeVoe, M., & Bulka, D. (1989). Speaking about feelings: Conceptions of emotion across the life span. *Psychology and Aging, 4*, 425–437.

Larson, R., & Asmussen, L. (1991). Anger, worry, and hurt in early adolescence: An enlarging world of negative emotions. In M. E. Colton & S. Gore (Eds.), *Adolescent stress: Causes and consequences* (pp. 21–41). New York: de Gruyter.

Larson, R., & Lampman-Petraitis, C. (1989). Daily emotional states as reported by children and adolescents. *Child Development and Psychopathology, 60*, 1250–1260.

Larson, R., Moneta, G., Richards, M. H., & Wilson, S. (2002). Continuity, stability, and change in daily emotional experience across adolescence. *Child Development, 73*, 1151–1165.

Lazarus, R. S., & Folkman, S. (1984). Coping and adaption. In W. D. Gentry (Ed.), *Handbook of behavioral medicine* (pp. 282–325). New York: Guilford Press.

Luerssen, A., & Ayduk, O. (this volume, 2014). The role of emotion and emotion regulation in the ability to delay gratification. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 111–125). New York: Guilford Press.

Matarazzo, O. (2008). Use and effectiveness of three modalities of emotion regulation after negative life events: Rumination, distraction and social sharing. In A. B. Turley & G. C. Hofmann (Eds.), *Life style and health research progress* (pp. 87–138). Hauppauge, New York: Nova Biomedical Books.

Mauss, I. B., Bunge, S. A., & Gross, J. J. (2007). Automatic emotion regulation. *Social and Personality Psychology Compass, 1*, 146–167.

McLaughlin, K. A., Hatzenbuehler, M. L., Mennin, D. S., & Nolen-Hoeksema, S. (2011). Emotion dysregulation and adolescent psychopathology: A prospective study. *Behaviour Research and Therapy, 49*, 544–554.

McRae, K., Gross, J. J., Weber, J., Robertson, E. R., Sokol-Hessner, P., Ray, R. D., et al. (2012). The development of emotion regulation: An fMRI study of cognitive reappraisal in children, adolescents and young adults. *Social Cognitive and Affective Neuroscience, 7*, 11–22.

Mesquita, B., De Leersnyder, J., & Albert, D. (this volume, 2014). The cultural regulation of emotions. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 284–302). New York: Guilford Press.

Miller, E. M., & Shields, S. A. (1980). Skin conductance response as a measure of adolescents' emotional reactivity. *Psychological Reports, 46*, 587–590.

Mohanty, A., Engels, A. S., Herrington, J. D., Heller, W., Ho, M. H., Banich, M. T., et al. (2007). Differential engagement of anterior cingulate cortex subdivisions for cognitive and emotional function. *Psychophysiology, 44*, 343–351.

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development, 16*, 361–388.

Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9*, 242–249.

Ochsner, K. N., & Gross, J. J. (this volume, 2014). The neural bases of emotion and emotion regulation: A valuation perspective. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 23–42). New York: Guilford Press.

Opitz, P. C., Gross, J. J., & Urry, H. L. (2012). Selection, optimization, and compensation in the domain of emotion regulation: Applications to adolescence, older age, and major depressive disorder. *Social and Personality Psychology Compass, 6*, 142–155.

Ordaz, S., Davis, S., & Luna, B. (2010). Effects of response preparation on developmental improvements in inhibitory control. *Acta Psychologica, 134*, 253–263.

Perry-Parrish, C., & Zeman, J. (2011). Relations among sadness regulation, peer acceptance, and social functioning in early adolescence: The role of gender. *Social Development, 20*, 135–153.

Pfeifer, J. H., & Allen, N. B. (2012). Arrested development? Reconsidering dual-systems models of brain function in adolescence and disorders. *Trends in Cognitive Sciences, 16*, 322–329.

Reijntjes, A., Stegge, H., Terwogt, M. M., Kamphuis, J. H., & Telch, M. J. (2006). Emotion regulation and its effects on mood improvement in response to an *in vivo* peer rejection challenge. *Emotion, 6*, 543–552.

Riediger, M., & Rauers, A. (in press). Do everyday affective experiences differ throughout adulthood? A review of ambulatory-assessment evidence. In P. Verhaeghen & C. Hertzog (Eds.), *The Oxford handbook of emotion, social cognition, and everyday problem solving during adulthood*. New York: Oxford University Press.

Riediger, M., Schmiedek, F., Wagner, G., & Lindenberger, U. (2009). Seeking pleasure and seeking pain: Differences in pro- and contrahedonic motivation from adolescence to old age. *Psychological Science, 20*, 1529–1535.

Riediger, M., Wrzus, C., Schmiedek, F., Wagner, G. G., & Lindenberger, U. (2011). Is seeking bad mood cognitively demanding? Contrahedonic orientation and working-memory capacity in everyday life. *Emotion, 11*, 656–665.

Riediger, M., Wrzus, C., & Wagner, G. G. (2013). *Happiness is pleasant, or is it? Representations of affect valence are associated with contra-hedonic motivation and mixed affect in daily life*. Manuscript submitted for publication.

Robinson, M. D. (2007). Gassing, braking, and self-regulating: Error self-regulation, well-being, and goal-related processes. *Journal of Experimental Social Psychology, 43*, 1–16.

Rothbaum, F., Weisz, J. R., & Snyder, S. S. (1982). Changing the world and changing the self: A two-process model of perceived control. *Journal of Personality and Social Psychology, 42*, 5–37.

Salisch, M. V., & Vogelgesang, J. (2005). Anger regulation among friends: Assessment and development from childhood to adolescence. *Journal of Social and Personal Relationships, 22*, 837–855.

Schmeichel, B. J., & Demaree, H. A. (2010). Working memory capacity and spontaneous emotion regulation: High capacity predicts self-enhancement in response to negative feedback. *Emotion, 10*, 739–744.

Schmeichel, B. J., Volokhov, R. N., & Demaree, H. A. (2008). Working memory capacity and the self-regulation of emotional expression and experience. *Journal of Personality and Social Psychology, 95*, 1526–1540.

Schulz, M. S., Waldinger, R. J., Hauser, S. T., & Allen, J. P. (2005). Adolescents' behavior in the presence of interparental hostility: Developmental and emotion regulatory influences. *Development and Psychopathology, 17*, 489–507.

Shaver, P. R., & Mikulincer, M. (this volume, 2014). Adult attachment and emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 237–250). New York: Guilford Press.

Sheeber, L., Allen, N., Davis, B., & Sorensen, E. (2000). Regulation of negative affect during mother–child problem-solving interactions: Adolescent depressive status and family processes. *Journal of Abnormal Child Psychology, 28*, 467–479.

Shortt, J. W., Stoolmiller, M., Smith-Shine, J. N., Mark Eddy, J., & Sheeber, L. (2010). Maternal emotion coaching, adolescent anger regulation, and siblings externalizing symptoms. *Journal of Child Psychology and Psychiatry, 51*, 799–808.

Silk, J. S., Steinberg, L., & Morris, A. S. (2003). Adolescents' emotion regulation in daily life: Links to depressive symptoms and problem behavior. *Child Development, 74*, 1869–1880.

Silk, J. S., Vanderbilt-Adriance, E., Shaw, D. S., Forbes, E. E., Whalen, D. J., Ryan, N. D., et al. (2007). Resilience among children and adolescents at risk for depression: Mediation and moderation across social and neurobiological contexts. *Development and Psychopathology, 19*, 841–865.

Silvers, J. A., McRae, K., Gabrieli, J. D., Gross, J. J., Remy, K. A., & Ochsner, K. N. (2012). Age-related differences in emotional reactivity, regulation, and rejection sensitivity in adolescence. *Emotion, 12*(6), 1235–1247.

Sim, L., & Zeman, J. (2006). The contribution of emotion regulation to body dissatisfaction and disordered eating in early adolescent girls. *Journal of Youth and Adolescence, 35*, 219–228.

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review, 28*, 78–106.

Steiner, M., Dunn, E., & Born, L. (2003). Hormones and mood: From menarche to menopause and beyond. *Journal of Affective Disorders, 74*, 67–83.

Stroud, L. R., Foster, E., Papandonatos, G. D., Handwerger, K., Granger, D. A., Kivlighan, K. T., et al. (2009). Stress response and the adolescent transition: Performance versus peer rejection stressors. *Development and Psychopathology, 21*, 47–68.

Sumter, S., Bokhorst, C., Miers, A., Van Pelt, J., & Westenberg, P. (2010). Age and puberty differences in stress responses during a public speaking task: Do adolescents grow more sensitive to social evaluation? *Psychoneuroendocrinology, 35*, 1510–1516.

Tamir, M. (2009). What do people want to feel and why? Pleasure and utility in emotion regulation. *Current Directions in Psychological Science, 18*, 101–105.

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development, 59*, 25–52.

Thompson, R. A. (this volume, 2014). Socialization of emotion and emotion regulation in the family. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 173–186). New York: Guilford Press.

Thorne, A. (2004). Putting the person into social identity. *Human Development, 47*, 361–365.

Yap, M. B., Allen, N. B., & Ladouceur, C. D. (2008). Maternal socialization of positive affect: The impact of invalidation on adolescent emotion regulation and depressive symptomatology. *Child Development, 79*, 1415–1431.

Yap, M. B., Allen, N. B., & Sheeber, L. (2007). Using an emotion regulation framework to understand the role of temperament and family processes in risk for adolescent depressive disorders. *Clinical Child and Family Psychology Review, 10*, 180–196.

Yurgelun-Todd, D. (2007). Emotional and cognitive changes during adolescence. *Current Opinion in Neurobiology, 17*, 251–257.

Zeman, J., Cassano, M., Perry-Parrish, C., & Stegall, S. (2006). Emotion regulation in children and adolescents. *Journal of Developmental and Behavioral Pediatrics, 27*, 155–168.

Zimmer-Gembeck, M. J., & Skinner, E. A. (2011). Review: The development of coping across childhood and adolescence: An integrative review and critique of research. *International Journal of Behavioral Development, 35*, 1–17.

CHAPTER 13

Emotion Regulation and Aging

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Developmental psychologists have documented age-related changes in the regulation of emotions across childhood that parallel biological maturation. As children and adolescents acquire and develop abilities to control impulses and gain awareness of themselves and others, they begin to achieve mastery over their environments and become increasingly effective at describing and regulating their emotions. Brain maturation and patterns of neurological functioning related to emotional processes continue to develop throughout childhood and adolescence. Greater inhibitory control, for example, is reflected in increased activity in the prefrontal cortex from childhood into the early 20s (e.g., McRae et al., 2012). In the latter half of the lifespan, however, the role of biological development in emotional experience and regulation is relatively understudied. Physiological functioning is gradually degraded with age, and it has long been assumed that such changes affect emotional functioning. A growing literature, however, suggests that aging is associated with relatively positive emotional experience in everyday life. As the understanding of emotional aging improves, it is becoming increasingly clear that older people typically engage in strategies that maintain positive emotional experiences and limit negative ones. It is also becoming clear that circumstances that restrict strategy usage may have especially dire outcomes for older adults.

For our purposes herein, we define *emotion regulation* as efforts to experience relatively low levels of negative affect and elicit relatively high levels of positive affect (see Gross, this volume, for a more complete description of emotion regulation). The study of age differences in emotion regulation has grown, although direct comparisons across the lifespan remain limited. Similarly, studies that focus on thoughts and behaviors that enable people to avoid or mitigate exposure to distressing events rarely link such strategies to affective outcomes. Only a few studies, which we describe below, examine age differences in regulation when people are intentionally increasing (i.e., up-regulating or amplifying) or decreasing (i.e., down-regulating) their positive and negative emotional states.

We focus our review on normal aging as opposed to disease-related aging, and on the general experience of positive and negative affect as opposed to discrete emotions. We begin with a brief overview of age differences in physiological processes involved in emotional experience. We then describe the scope of the existing literature examining age and affective well-being. Next, we pres-

ent socioemotional selectivity theory (SST) and strength and vulnerability integration (SAVI), both of which guide our understanding of emotional development in adulthood. SST has received considerable empirical support since it was proposed in the early 1990s (see Charles & Carstensen, 2010). Its core tenets concern the ways that time horizons influence goals. SST initially addressed emotion somewhat indirectly, based on the premise that as people become increasingly focused (selectivity) on emotionally meaningful goals, well-being likely benefits. As evidence for social selection accrued along with evidence that emotional well-being is relatively high in later life, we began to consider selection as a highly effective form of antecedent emotion regulation (Carstensen, Fung, & Charles, 2003). SAVI, in contrast, was proposed relatively recently. It aims to integrate the relatively positive profile of emotional well-being with concurrent evidence of repercussions for older adults who experience sustained and inescapable exposure to toxic situations. We review empirical findings that are consistent with the premise that motivational shifts lead to differences in attention, memory, social partner preferences, and problem-solving strategies that serve to maintain or optimize emotional experience. Next, we discuss situations posited by SAVI, in which benefits of age may be less pronounced and may even reverse in direction. We then review areas that we previously highlighted as especially important and how they have been addressed since the previous edition of this volume. We conclude that the emerging literature suggests that age differences in emotional experience, cognitive processes, and physiological functioning operate in concert to yield relatively well-preserved if not enhanced emotional well-being in later life.

Emotion and the Aging Physiological System

Emotional experience, like all psychological phenomena, is inextricably linked to physiological functioning. Aging in three biological systems—the central nervous system (specifically the brain), the cardiovascular system, and the neuroendocrine system—is relevant for emotional aging.

Age Changes in Brain Structure and Functioning

Researchers have documented normative age-related changes in both brain structure and functioning. Brain volume decreases with age, with reductions in the synaptic density of white matter and a consequent slowing of neural processing that accompanies demyelination (e.g., Westlye et al., 2010). These age-related decreases are observed after the fourth decade of life, and exhibit an accelerating decline beginning in the early to mid-60s (Westyle et al., 2010).

Brain activation in response to emotional stimuli shifts throughout childhood and into early adulthood toward less activation in the amygdala and greater activation of the prefrontal cortex, findings that researchers use to explain developmental increases in emotion regulation abilities (McRae et al., 2012). The same difference in relative activity of these brain regions is found in comparisons of younger and older adults, a phenomenon referred to as the *posterior–anterior shift in aging* (PASA; Davis, Dennis, Dasellar, Fleck, & Cabeza, 2008). In memory and perceptual studies, with and without emotional stimuli, older adults show greater activity in the prefrontal regions and reduced activity in medial and temporal regions compared to younger adults (Davis et al., 2008).

Age Changes in Cardiovascular and Neuroendocrine Functioning

Cardiovascular and neuroendocrine responses also change with age. Although the overall pattern of cardiovascular reactivity to stressors is the same, aging is related to relative differences in the degree of this reactivity. Specifically, older age is related to a smaller increase in heart rate in response to stressors but greater increases in blood pressure reactivity (see review by Uchino, Birmingham, & Berg, 2010). The reduced heart rate reactivity most likely represents age-related reductions in heart rate variability (HRV) that are normative and begin in adolescence, and that are a risk factor for mortality (Zulfigar, Jurivich, Gao, & Singer, 2010).

The neuroendocrine response also varies with age (Björntorp, 2002). The perception of a real or imagined threat mobilizes the

body for action through a series of reactions along the hypothalamic–pituitary–adrenal (HPA) axis that results in the release of cortisol by the adrenal glands. In both human and animal models, cortisol reactivity in response to psychological and physical stressors is prolonged with age, such that more time is necessary to return to baseline levels (see review by Björntorp, 2002).

Age Differences in Emotional Experience

Early theories positing that emotional well-being and regulation would parallel age-related biological decline were so pervasive that researchers did not bother to test them. In the last few decades, however, researchers have dispelled many myths about inevitable depression and despair in late life. Older age is not associated with high levels of emotional distress in examinations of people ranging from ages 18 to 84 years (Kobau, Safran, Zack, Moriarty, & Chapman, 2004). Rates of anxiety and major depressive disorder are lower among adults age 65 years and older than in younger cohorts (see review by Piazza & Charles, 2006; Villamil, Huppert, & Melzer, 2006). Reports of subdromal depression—feeling sad, blue, or depressed within the past 30 days—decrease linearly across increasingly older age groups (Kobau et al., 2004).

This age-related decrease in self-reported negative affect is observed longitudinally in normal populations as well (e.g., Carstensen, Pasupathi, Mayr, & Nesselroade, 2000; Carstensen et al., 2011), although patterns vary across studies when examining people over age 60. Findings from a number of cross-sectional studies suggest that the experience of negative emotion continues to decline after age 60 (e.g., Grühn, Kotter-Grühn, & Röcke, 2010; Stone, Schwartz, Broderick, & Deaton, 2010). This decrease has also been found over time among cohorts of younger, middle-aged, and older adults followed across 20 years (Charles, Reynolds, & Gatz, 2001). In one experience sampling study, the decrease in negative emotional experience peaked and then stabilized somewhere between ages 60 and 70 (Carstensen et al., 2011). Other studies have found slight age-related increases in depressive symptoms in advanced old age (e.g., after age 80 in one study; Teachman, 2006), although levels were still lower than those observed in young adults. In another study, adjusting for functional limitations and chronic illness eliminated the upturn in negative affect among the octogenarians (e.g., Kunzmann, Little, & Smith, 2000).

Findings from studies that examine positive affect are mixed but overall point to considerable stability. Although age-related linear decreases (about half a point on a 5-point Positive Affect Scale) were observed among people from a large, multinational sample of people ranging in age from their 20s to 80s (Diener & Suh, 1998), this pattern has not been found in other studies. Using the same scale, a large longitudinal study found age-related stability across 20 years among younger and middle-aged adults, and a small decline only among people age 65 and older who began the study (Charles et al., 2001). Another large U.S. national survey observed slightly higher levels of positive affect with age in a sample of people ranging in age from 25 to 74 years (Mroczek & Kolarz, 1997). Two other large surveys found that reports of daily happiness and enjoyment (in a U.S. sample) and reports of joviality and serenity (in a German sample) exhibited an age-related U-shaped pattern, decreasing among successively older adults from ages 18 to the mid-50s, but increasing thereafter, such that people in their 70s and 80s had levels of these emotions similar to those of people in their late teens and early 20s (Grühn et al., 2010; Stone et al., 2010); people in their 50s reported the lowest levels of these positive emotions in both the U.S. and German samples.

Taking the findings on negative and positive affect together, overall well-being does not follow the physiological trajectories of linear decline across adulthood. One potential explanation for these findings is that age-related reductions in physiological ability may paradoxically lead to flattened, or diminished, emotional experiences that are easier to regulate. Studies provide little evidence, however, for this hypothesis. Older adults report less negative emotion in daily life, but the reported *intensity* of both positive and negative emotions is similar across age groups (Carstensen et al., 2000, 2011). In other words, when older

people do experience emotions, the intensity is similar to that of younger people. Older and middle-aged adults report similar levels of emotional experience when asked to relive emotionally charged events or to engage in a confrontation with their spouses (Levenson, Carstensen, & Gottman, 1994; Magai, Consedine, Krivoshekova, Kudadjie-Gyamfi, & McPherson, 2006; Story et al., 2007). Some studies in which people react to film clips have revealed higher subjective ratings of intensity in older adults than in younger adults in response to scenes eliciting sadness, especially when scenes were particularly salient for older people, for example, in a depiction of a person realizing that she has Alzheimer's disease (Kunzmann & Grühn, 2005; Kunzmann & Richter, 2009; Kliegel, Jäger, & Phillips, 2007; Seider, Shiota, Whalen, & Levenson, 2011). Older adults also reported greater reports of intensity to film clips depicting injustice (Charles, 2005; Phillips, Henry, Hosie, & Milne, 2008). Other film-based studies have not observed age differences (Tsai, Levenson, & Carstensen, 2000).

Socioemotional Selectivity Theory

SST, a lifespan theory of motivation that addresses how and why emotional well-being changes across the lifespan, posits that at conscious and subconscious levels, human monitor time, continuously adjusting their time horizons as they move through life (see Carstensen, 1992, 2006; Carstensen, Isaacowitz, & Charles, 1999). This accounting of time influences the relative importance of two constellations of goals that dominate human thought and behavior. One set of goals includes knowledge and information-based goals, and is prioritized when time is perceived as expansive. The other goal constellation includes a focus on emotional life, encompassing motivations to derive meaning in actions and to invest in activities for their emotional significance.

Because time perspective is strongly related to place in the life cycle, growing older comes with an increasing awareness of the ephemeral nature of existence: As a result, emotional goals increase in relative importance to knowledge-related goals as people age. People become increasingly selective in their goals as they age, so that experiences are more emotionally meaningful and satisfying. This selection process falls under the rubric of antecedent-focused emotion regulation, whereby people engage in actions to avoid the experience of distress and to maintain or enhance the experience of positive emotions. Accordingly, people allocate more effort to maintaining emotional balance, attempting to improve important relationships, and setting aside relatively trivial matters. Of course, experience grows with age as well, so older people are well-positioned to manage social relationships and have the skills to solve important conflicts. According to SST, motivational shifts that also operate at a subconscious level influence cognitive processing in which people favor positive material and disattend to negative information. Goals always direct attention and memory, and with age, cognition comes to operate in the service of emotion regulation.

Strength and Vulnerability Integration

SAVI endorses SST and its tenet that awareness of time left to live directs priorities, such that a focus on emotional experience, and specifically positive emotional experiences above negative ones, gradually comes to influence thought and action (Charles, 2010; Charles & Piazza, 2009). People act on these motivations based on their accrued knowledge about themselves and their social environment, and their experience from time lived. As a result, aging is associated with motivational shifts—both conscious and subconscious—that direct attention, influence memorial processing, and change preferences in ways that allow people to circumvent negative experiences. If they cannot avoid these experiences altogether, cognitive-behavioral emotion regulation strategies allow them to decrease their exposure and mitigate relatively minor stressors in their day-to-day lives.

According to SAVI, older adults more often engage in emotion regulation strategies (i.e., thoughts and behaviors whose aim is to reduce negative affect and maintain or even enhance positive affect) than do younger adults who fail to shy away from negative

situations, presumably because there is informational value in negative experiences that prepares people for the future. When confronted with minor events, older adults appraise them as less stressful (e.g., Charles & Carstensen, 2008). When older adults cannot employ strengths of aging to avoid or mitigate their exposure to a negative event, however, they experience distress. During these demanding and highly arousing situations, SAVI posits that the age-related benefits disappear or even reverse in direction. Because age is related to biological changes that present greater threats to a less robust physiological system, SAVI posits that high levels of physiological reactivity and consequent recovery in response to sustained negative arousal are more costly to older adults.

Age, Cognitive Functioning, and Antecedent-Focused Emotion Regulation

Emotional Salience and Emotion Regulation

Brains do not operate like computers, treating all information equally. Rather, cognitive resources are directed to goal-relevant information. SST posits that older adults place more importance and prominence on emotional than on nonemotional aspects of stimuli they encounter in the laboratory or in their daily lives, and they do so without explicit awareness of their actions. Laboratory studies using incidental memory paradigms reveal that the proportion of emotional versus nonemotional information increases linearly as a function of age (Carstensen & Turk-Charles, 1994), and emotional details are recalled better than perceptual details of stories (e.g., Mather, Johnson, & De Leonardis, 1999). In addition, the salience of emotion is greater with age when people recall autobiographical information (Alea, Bluck, & Semegon, 2004). Overall, this pattern of findings suggests that older adults pay attention to emotionally relevant information that in and of itself may aid emotion regulation.

Rather than "positivity bias" we coined the term *positivity effect* to describe the age-related pattern of change (Carstensen & Mikels, 2005; Mather & Carstensen, 2005). Age differences are as frequently driven by a preference for negative material in the young as a preference for positive material in the old. The positivity effect refers to the age-related pattern of change.

Brain Activity and the Positivity Effect

Studies of brain activity during the processing of emotional information support the positivity effect. Mather et al. (2004) speculate that subconscious changes in motivation influence activation of the amygdala, consistent with their finding that older adults show greater amygdalar activity when viewing positive rather than negative images. Other brain regions also show age differences in activation depending on the emotional valence of the information (Samanez-Larkin et al., 2007). In a study examining brain activity in response to decision making, older adults exhibited less activation in insula and caudate regions than did younger adults when they anticipated loss, but similar activation when anticipating gains (Samanez-Larkin et al., 2007).

The general PASA in processing information discussed previously (St. Jacques, Bessette-Symons, & Cabeza, 2009) may serve emotion regulation goals. For example, one study revealed that older adults display less amygdalar activity and greater activation of areas associated with emotion regulation (prefrontal cortex) when viewing negative images compared to positive images relative to younger adults (Leclerc & Kensinger, 2011). Another study revealed age differences in connectivity of the amygdala to other brain regions, consistent with age-related increases in emotion regulation processes (St. Jacques, Dolcos, & Cabeza, 2009, 2010). Specifically, older adults showed more activity in a brain region associated with emotion regulation (ventral anterior cingulate cortex), and less activity in areas associated with perceptual processing (visual cortex) than did the younger adults (St. Jacques et al., 2010). Age-related reductions in activities related to perceptual processing are consistent with the well-established age-related reductions in facial emotion recognition in the literature (e.g., Lambrecht, Kreifelts, & Wildgruber, 2012). Similar patterns emerge with brain activation and consequent memory for these pictures (St. Jacques et al., 2009). Older adults exhibit greater activity

in frontal regions associated with emotional control (bilateral dorsolateral prefrontal cortex) and less activity in regions associated with later memory encoding (hippocampus and bilateral ventrolateral prefrontal cortices) for negative stimuli relative to neutral stimuli than do younger adults. Behavioral data again reflected these findings: Older adults were less likely than younger adults to remember the negative stimuli relative to the neutral stimuli (St. Jacques et al., 2009).

Age-Related Changes in Attention and Working Memory

The positivity effect explains how chronic activation of emotion-related goals directs cognitive resources systematically toward positive and away from negative stimuli—often without conscious awareness. Older adults focus their attention on more positive and less negative stimuli than do younger adults in studies of simple attention, as assessed by reaction times (Mather & Carstensen, 2003) and eye gaze (e.g., Isaacowitz, Toner, Goren, & Wilson, 2008; Nikitin & Freund, 2011).

SST predicts that top-down motivational changes with age lead to changes in attention and memory that serve emotion regulation goals, such that performance may be better for emotional information than for nonemotional information. One such example lies in working memory, which is vital for emotional functioning and planning, because working memory allows an individual to focus on the potential for rewards and enhances goal-seeking behavior (Davidson, Jackson, & Kalin, 2000). In a test of SST, the working memory performance of older and younger adults was examined on tasks in which participants compared images based on either their brightness intensity or emotional intensity (Mikels, Larkin, Reuter-Lorenz, & Carstensen, 2005). Findings reveal the usual age-related decrement in working memory for visual brightness but no age difference in working memory for affective arousal. Moreover, the lack of age differences was driven by older adults' better performance for positive stimuli than for negative stimuli compared to younger adults (Mikels et al., 2005). Other studies suggest that emotion regulation is less cognitively taxing for older adults than for younger adults (e.g., Scheibe & Blanchard-Fields, 2009). In one study, Scheibe and Blanchard-Fields asked younger and older adults to perform a working memory task after having been induced into a negative mood and asked to down-regulate or maintain current mood, or were provided no specific instructions. Although the performance of younger adults deteriorated when they were asked to down-regulate their emotions, the performance of older adults did not vary from that in the other conditions. Similarly, another study found that emotional suppression was related to worse memory for younger adults but not for older adults (Emery & Hess, 2011).

Evidence That Memory Supports Emotion Regulation

Antecedent-focused emotion regulation strategies prevent or reduce exposure to events and thoughts that threaten well-being. To the extent that memories influence current mood states, thoughts, and behavior (see review by Levine & Pizarro, 2004), memory can be a powerful regulation strategy. This shift in valence toward positive information with age has been documented in studies examining memory for both life events and laboratory stimuli. Memory about past events is more positive for older compared to younger adults (Kennedy, Mather, & Carstensen, 2004). Even when asked about prior negative events, older adults report them as having been more positive than do younger adults (Schryer & Ross, 2012).

Laboratory studies underscore the shift toward older people remembering events more positively. In a study in which younger, middle-aged, and older adults viewed positive, negative, and neutral images, and were later asked to recall and to recognize these previously viewed images from a larger set of images, younger adults recalled similar levels of positive and negative stimuli, and they recognized a greater proportion of negative images than positive images (Charles, Mather, & Carstensen, 2003). Older adults displayed no such negative bias in their recognition, and they recalled significantly fewer negative than positive images (Charles et al., 2003). This age-associated shift toward positive emotion and away from negative

information has been replicated in a number of studies from many laboratories (e.g., Petrican, Moscovitch, & Schimmack, 2008; Piguet, Connally, Krendl, Huot, & Corkin, 2008; see reviews by Reed & Carstensen, 2012; Scheibe & Carstensen, 2010).

Cognitive Appraisal of Negative Events

Older adults not only focus their attention on less negative aspects of the environment but they also appraise ambiguous or unpleasant events more benignly than do younger adults. Older adults perceive daily stressors as less severe and less threatening than do younger adults in a sample ranging from 25 to 74 years (Charles & Almeida, 2007). They appraise aversive stimuli less negatively than do younger adults, and are more likely to focus their thoughts away from the negative content (Charles & Carstensen, 2008). Even when asked about their diagnosis and treatment of cancer, older adults report lower levels of threat appraisals (e.g., the amount of tension people experience in response to their illness) and are more likely than younger adults to frame the event as a challenge rather than a threat (Hart & Charles, 2013).

The Positivity Effect Is Malleable

SST maintains that—all things being equal—chronically activated goals differ for older and younger people. However, because positivity reflects top-down motivational shifts as opposed to bottom-up brain-driven shifts, the effect is theoretically malleable. Younger people sometimes pursue emotional goals, and older adults sometimes pursue informational goals. In circumstances where stakes are high, younger and older people may pursue goals that direct attention toward or away from emotional material accordingly. Indeed, Löckenhoff and Carstensen (2007) examined whether experimental instructions provided to participants would eliminate age differences. In an initial phase of the study, participants were simply asked to review information about a variety of alternative health care plans. In this condition, older people focused more on positive than on negative characteristics. In a second condition, researchers explicitly asked older adults to focus on informational aspects of the plans; that is, they gave the participants a specific goal, namely, to pursue information. And in this condition, age differences were eliminated. Reed and Carstensen (2013) recently reviewed the positivity effect literature and concluded that positivity preferences are strongest during controlled processing when motivations are hypothesized to play a strong role (for discussion, see Kensinger, 2004).

Whether or not positivity in cognitive processing is adaptive or maladaptive depends on the task. Certainly there are situations where selective attention to positivity may disadvantage performance. Decision making likely benefits from evenhanded processing of advantages and disadvantages associated with options. Focusing on the positive interpersonal qualities of a charming conman could have disastrous financial consequences. Ignoring negative information or appraising a situation as less negative than it is may lead to short-term benefits to well-being but longer term problems that lead to distress (Scheibe & Carstensen, 2010). Focusing on positive aspects and avoiding negative realities of fiscal concerns may lead to "optimistic, but not necessarily realistic, financial planning" (Weierich et al., 2011, p. 197). Ignoring negative information may contribute to misremembering negative information as more positive than it actually was (Shamaskin, Mikels, & Reed, 2010).

Finally, there is some evidence that not only emotional valence but also arousal contribute to age differences in positivity. Grühn and Scheibe (2008) found that older people perceived positive pictures in the International Affective Picture System (IAPS) as *more* positive and *less* arousing than did younger adults. Keil and Freund (2009) later observed that older adults *prefer* less arousing positive and negative images and words than do younger adults, and Streubel and Kunzmann (2011) reported that the subjective experience of positivity in older adults is reduced under high arousal. Using experience sampling data, Scheibe, English, Tsai, and Carstensen (2013) recently examined the effect of age differences in the value placed on high- versus low-arousal states and concluded that older people placed more value on low-arousal positive states and were more successful in achieving them than their younger counterparts. Taken together, these

findings suggest that older adults may orient to positive material that is more calming and peaceful than to positive material associated with excitement and surgency.

Social Partners and Emotion Regulation

This review has focused largely on age-related changes in cognitive processes that serve emotion regulation goals. SST also makes specific predictions about how age-related behaviors, and explicitly changes in social partner preferences and interaction patterns, serve emotion regulatory goals. Older adults have smaller and more carefully pruned social networks that contain larger percentages of emotionally close social partners than do those of younger adults (Lang & Carstensen, 1994). Younger peoples' networks, by way of contrast, often include many social partners who were not chosen freely but have been incorporated because of relationships to work or offspring (Lang & Carstensen, 1994; Lansford, Sherman, & Antonucci, 1998). Thus, the social networks of older adults may be easier to navigate emotionally. Because most strong emotions occur in social contexts, careful selection of social partners is arguably a highly effective strategy. When faced with interpersonal conflict in their networks, older adults engage in more conflict avoidance, with the goal of preserving harmony in their relationships, than do middle-aged and younger adults (Blanchard-Fields, Mienaltowski, & Seay, 2007).

Responding to Negative Events

The previously discussed literature points to older adults using—either knowingly or not—thoughts and behaviors that fall under the category of antecedent-focused emotion regulation strategies. Some researchers argue, however, that older adults' greater well-being reflects not actions by the older adults but instead age-related differences in environmental demands (see review by Lawton, 2001). According to this view, the end of child-rearing responsibilities and the onset of retirement decreases the frequency of negative events more in the lives of older than younger adults (see review by Lawton, 2001; but see Villamil et al., 2006). On the other hand, older people face many challenges that younger people are less likely to face, such as chronic health problems, loss of loved ones, and age discrimination.

Although questions about environmental demands are important and there is some evidence that changes in life circumstances may contribute to the decreased number of stressors older adults report in their daily lives (e.g., Charles & Almeida, 2007), there is evidence that older adults are also active agents in influencing their well-being. Compared to younger people, older adults report better control over their emotions (Gross et al., 1997), enhanced abilities to regulate emotions in general (Kessler & Staudinger, 2009), as well as more success at calming strategies and other thoughts to control their feelings of anger (Phillips, Henry, Hosie, & Milne, 2008). Although the veracity of subjective reports alone may be questioned, they are consistent with low levels of psychiatric disease and reported experience, and behavioral evidence in the literature. For example, when people ranging in age from 18 to 94 years reported the emotions they were experiencing at five random times each day throughout the course of a week, the probability of continuing to feel a negative emotion from one reporting interval to the next was lower with advancing age (Carstensen et al., 2000). Researchers have discussed how older adults select and are adept at using emotion regulation strategies given their decrements in others area of their lives, such as cognitive functioning (Urry & Gross, 2010).

Questions remain, however, as to how well older adults regulate their emotions when they cannot avoid or mitigate exposure to a negative event. SAVI posits that when older adults cannot avoid experiencing high and sustained levels of emotional distress, this arousal reduces any age-related benefits previously observed for regulation abilities. One study explicitly compared age differences in affective outcomes in two situations: one in which people disengaged from a negative event and another in which they did not (Charles, Piazza, Luong, & Almeida, 2009). When older adults encountered an unpleasant situation in which they chose to avoid further conflict, they reported lower levels of affective reactivity than did younger adults. When they engaged in a conflict, no

age differences in reactivity were apparent (Charles et al., 2009).

Other studies also suggest that high levels of arousal reduce age-related benefits in emotional outcomes and may even show age-related decrements. When older and younger adults report stressors of equally high severity, older adults display greater affective reactivity than do younger adults (Mroczek & Almeida, 2004). Longitudinal data confirm this age-related pattern, in that when people report experiencing a stressor, affective reactivity increases with age (Sliwinski, Almeida, Smyth, & Stawski, 2009).

The studies described earlier use information gathered from the daily lives of adults. Researchers attempting to capture emotion regulation in the laboratory face the challenge of devising paradigms that elicit responses sufficiently strong to demand active and sustained regulatory efforts. Although use of weak stimuli, such as pictures of facial expressions, has become commonly accepted, we strongly question the validity of this approach when researchers are interested in participants' efforts to regulate emotional responses actively. Stronger inductions are possible with film clips, personal memories, music, and interactions with confederates or spouses. Of course, a tradeoff exists between carefully controlled elicitors and responsivity. Only a handful of such studies that focus on age differences in regulation effectiveness have been published to date.

In one study, subjective reports of distress were compared across groups of younger (18 to 40 years old) and older adults (60 to 88 years old) who watched emotional film clips with instructions to react naturally, suppress all facial expressions, or positively refocus on a pleasant memory if they felt any negative emotions (Phillips et al., 2008). Results indicate that older adults reported less intense negative affect in the positive reappraisal condition compared to the other conditions. When comparing across age groups, older and younger adults had similar ratings in the positive reappraisal condition, but younger adults reported less negative affect than older adults in the conditions where they were instructed to react naturally or to suppress their facial expressions.

Another study examined the baseline, reactivity, and recovery ratings of negative affect in older (60 to 75 years old) and younger (20 to 30 years old) adults in response to a film clip that elicited disgust (Scheibe & Blachard-Fields, 2009). After having viewed the film clips, participants were given instructions to attempt to change the negative feelings into positive ones or to continue feeling their negative emotions and not try to change them, or they were given no instructions. Results indicated no significant age differences in the reported affect during the recovery period. However, older adults showed less deterioration in performance on one of the three trials of a *n*-back task following the induction, suggesting that fewer resources were required for regulation.

A third study examined three types of regulation strategies in older (60–69 years), middle-aged (40–49 years) and younger adults (20–29 years) in response to emotional film clips (Shiota & Levenson, 2009). The three strategies were detached appraisal, in which participants were told to watch objectively in order to feel less negative emotion; positive reappraisal, in which they were told to think about positive aspects of what they were viewing so that they would feel less negative emotion; or emotional suppression, in which they were asked to hide negative facial expressions. When comparing subjective reports across different conditions within each age group, researchers found that detached appraisal resulted in significantly lower levels of distress among the younger adults, marginally significant lower levels of distress in middle-aged adults, but the same level of distress in older adults when compared to the "just watch" condition. Only older adults showed significantly lower levels of subjective distress in the positive reappraisal condition compared to the "just watch" condition. When examining mean levels of the target emotions (sadness or disgust) across age groups, subgroups reporting the lowest mean levels were the youngest and middle-aged adults in the detached appraisal group (3.25 and 4.08, respectively), and the two groups with the highest reported target emotions were the middle-aged and oldest adults in the positive reappraisal groups (5.42 and 5.20, respectively).

One recent study compared younger (18- to 30-year-olds) and older (60- to 80-year-olds) participants' responses as they viewed

negative and neutral images in three conditions: a "just watch" condition, in which they were told simply to look at the image; a condition in which each image was preceded by a string of digits they need to remember immediately after their 10-second exposure to the image; and a condition in which they were instructed to reinterpret the picture in such a way that they would feel less negative affect (Tucker, Feuerstein, Mende-Siedlecki, Ochsner, & Stern, 2012). After each image, participants rated how much negative emotion they felt. Findings indicate that younger adults reported less negative reactivity in the reappraisal condition than did the older adults, but age groups were similar in levels of reactivity in the distraction condition.

In summary, few studies have examined emotion regulation strategies that target decreasing subjective distress when people are exposed to unpleasant stimuli under controlled laboratory conditions. Findings from the handful of existing studies do not point to consistent evidence for age-related improvements or impairment in emotion regulation. As we note below, however, we believe that more of this type of research is needed.

Physical Costs of Emotion Regulation

SAVI further posits that circumstances marked by higher levels of sustained arousal will have greater physiological consequences for older adults. Support for this tenet comes from literature examining both cardiovascular and HPA reactivity responses to emotional events in younger and older adults. For example, blood pressure reactivity is more pronounced in older adults relative to younger adults in response to stressors of daily life and laboratory-based stressors such as social evaluative threat (Ong, Rothstein, & Uchino, 2012; Uchino et al., 2010) and when dealing with cognitively complex stressors of daily life (Wrusz, Müller, Wagner, Lindenberger, & Riediger, 2013). In addition, the effect of a more prolonged negative experience—loneliness—is more strongly related to blood pressure recovery in older than in younger adults (Ong et al., 2012).

Age-related changes in HPA reactivity have been studied less frequently. Although researchers document prolonged HPA reactivity in animal studies, they have conducted fewer studies using human samples (Björntorp, 2002). Still, findings suggest that negative affect is related more strongly to cortisol output for older than for younger adults. For example, one study found that high levels of negative affect were related to higher level of daily cortisol output for relatively older participants (54 to 74 years old), but unrelated to this biomarker for people 53 and younger (Piazza, Charles, Stawski, & Almeida, 2013).

When Avoiding Negative Emotions Does Not Occur: Aging with Negative Affect

SAVI posits that affective well-being remains relatively stable and may increase with age, with one important caveat. When older people find themselves in unavoidable circumstances that elicit sustained physiological arousal, the consequences for well-being will be exacerbated. Researchers have long discussed the powerful role of the environment in shaping emotion-related outcomes and how age is related to different life circumstances. These circumstances are posited to explain why not all groups of older adults show continuing, positive trajectories of emotional experience. Loss of social belonging, prolonged caregiving, and neurological dysregulation may result in an upturn in depressive symptoms and negative affect in the last decades of life (Charles, 2010). For example, in the last years of life—often characterized by neurological and physiological dysregulation—there are reliable declines in emotional well-being. Indeed, Gerstorf and colleagues (2010) found that terminal decline is better predicted by closeness to death than by chronological age. On the one hand, age and closeness to death are strongly coupled in the population, but Gerstorf and his colleagues found that years from birth may be less important than the circumstances that define the years to death in predicting emotional well-being.

In addition, experiencing chronically high levels of trait-negative affect also makes people more vulnerable to emotional distress with age. For example, two studies have found that declines in subjective reports of negative affect do not continue into old age for those high in neuroticism (Charles et al., 2001; Ready, Akertedt, & Mroczek, 2012).

Future Directions and Conclusions

Empirical findings amassed over the past 30 years have dramatically changed the landscape of research on emotion and aging. Among healthy adults, emotional experiences and emotion regulation do not decline across adulthood but are instead well-maintained and, in some ways, improve. We review below the aims for future studies we recommended 5 years ago.

We stated that future studies will need to link hypothesized emotion regulation strategies (e.g., attention allocation) directly to emotion regulation. This area has begun to receive some attention (e.g., Isaacowitz & Blanchard-Fields, 2012), but more work and conceptual clarity are needed. SST, for example, focuses on selection as the key mechanism involved in greater well-being with age, and views selective attention and memory as consequences of changes in goals. In the theoretical context of SST, the positivity effect, rather than being a coping strategy, reflects goal-directed attention. Whether or not older people also deplore positive attention in response to distress remains an open question. SAVI posits that when selection is not possible, age advantages disappear or even reverse. Further study is needed to examine how emotion regulation strategies and effectiveness change with age, relative to the way age reflects both birth cohort and age-associated experience. In addition, we mentioned the need to study multiple response-focused regulation strategies and include conditions specifying maintenance, exacerbation, and attenuation of emotional experience in the investigations. As we reviewed earlier, several studies have begun to address different types of strategies among people of different ages (e.g., Shiota & Levenson, 2009). We hope that researchers will continue to investigate age differences in online emotion regulation tasks.

We mentioned the need to examine the importance of the arousal of emotional stimuli when studying age differences (Wurm, Labouvie-Vief, Aycock, Rebucal, & Koch, 2004), as well as the complexity of emotional stimuli (Charles, 2005). Researchers who take into account the role of arousal in processing and regulating emotions find that more arousing conditions may be more challenging for older adults (e.g., Ong et al., 2012). It will be important to consider both valence and arousal. We mentioned the need to examine discrete emotions in the previous edition of this volume, and provided the example that no large study to date has reported increases in levels of anger with age, but one study did observe age differences in reports of depressive symptoms (e.g., Teachman, 2006) although others have not. A growing number of studies has examined discrete emotions (Grühn et al., 2010) and especially emotion recognition (e.g., Slessor, Miles, Bull, & Phillips, 2010), yet theories still focus mostly on positive and negative valence and arousal when examining emotion regulation processes (see Charles & Carstensen, 2010).

One methodological issue that frequently arises in lifespan developmental research is the issue of cohort effects. Cross-sectional research points to potentially interesting developmental phenomena, but only longitudinal research can confirm developmental processes postulated by socioemotional selectivity theory. Researchers have made great strides in longitudinal investigation of more complex emotional stimuli in the past 5 years (e.g., Carstensen et al., 2011; Gerstorf et al., 2010; Sliwinski et al., 2009) and the maintenance of large longitudinal datasets promises to illuminate the field in this important area.

Methodological innovations in the past 5 years have advanced the field in several ways. For example, functional imaging of brain activity has illuminated associations between behavior and brain activity, and promises to provide insights into the neurological correlates of age differences. As this area grows, the study of emotion and aging will continue to benefit. A second innovation that will shape the future of psychological science is the growing area of health psychology and new techniques allowing for affordable assessments of cardiovascular, neuroendocrine, and immunological responses in the laboratory and in daily life. With these studies of physiological stability and reactivity, researchers will be able to examine how thoughts and behaviors are related to physiological arousal and reactivity accompanying emotional experience. In addition, smart phones provide the ability to assess cognitive functioning rapidly, along with momentary sampling of emotion across

the course of the days, which provides more online collection of cognitive and emotional experiences (see Riediger, Schmiedek, Wagner, & Lindenberger, 2009).

Finally, psychologists have long recognized the importance of the environment in shaping behavior (see review by Hess, 2005), but we need to continue to recognize the importance of context when interpreting age differences. Although notable exceptions exist, such as studies examining age differences in social cognition (e.g., Hess, 2005) and in health-related contexts (e.g., Consedine & Magai, 2006), further studies are needed to understand how similar behaviors or cognitions may have different outcomes according to age-specific contexts. Perceptions are guided by experiences, and these experiences may alter the meaning and interpretation of emotional stimuli. A picture of a surgical procedure, for example, may elicit more sadness in older adults but more disgust in younger adults (e.g., Kunzmann & Grühn, 2005). The death of a parent is often an imagined event for a college student and a long past reality for an older adult. To this end, the study of emotions and aging is an examination of how cognition and behavior shape, and are shaped by, the interplay between physiological processes and the environment across the lifespan.

In conclusion, emotions arouse physiological systems and direct attention, and they motivate people to action. Emotions are critical for successful adaptation across the lifespan, but developmental processes alter multiple facets of emotional experience, including cognitive appraisals, behavioral responses, physiological reactivity, and environmental context. Research on emotion and aging has revealed that shifts in cognitive processing can offset physiological decline, such that even though biological functioning peaks relatively early in life, emotional experience remains vital and well maintained into late adulthood.

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References

Alea, N., Bluck, S., & Semegon, A. B. (2004). Young and older adults' expression of emotional experience: Do autobiographical narratives tell a different story? *Journal of Adult Development, 11*, 235–250.

Björntorp, P. (2002). Alterations in the ageing corticotropic stress-response axis. In Novartis Foundation Symposium (Ed.), *Novartis Foundation Symposium 242—Endocrine facets of ageing* (pp. 46–58). London: Wiley.

Blanchard-Fields, F., Mienaltowski, A., & Seay, R. B. (2007). Age differences in everyday problem-solving effectiveness: Older adults select more effective strategies for interpersonal problems. *Journals of Gerontology: Psychological Science, 62*, P61–P64.

Carstensen, L. L. (1992). Social and emotional patterns in adulthood: Support for socioemotional selectivity theory. *Psychology and Aging, 7*, 331–338.

Carstensen, L. L. (2006). The influence of a sense of time on human development. *Science, 312*, 1913–1915.

Carstensen, L. L., Isaacowitz, D. M., & Charles, S. T. (1999). Taking time seriously: A theory of socioemotional selectivity. *American Psychologist, 54*, 165–181.

Carstensen, L. L., Fung, H., & Charles, S. (2003). Socioemotional selectivity theory and the regulation of emotion in the second half of life. *Motivation and Emotion, 27*, 103–123.

Carstensen, L. L., & Mikels, J. A. (2005). At the intersection of emotion and cognition: Aging and the positivity effect. *Current Directions in Psychological Science, 14*, 117–121.

Carstensen, L. L., Pasupathi, M., Mayr, U., & Nesselroade, J. R. (2000). Emotional experience in everyday life across the adult lifespan. *Journal of Personality and Social Psychology, 79*, 644–655.

Carstensen, L. L., Turan, B., Scheibe, S., Ram, N., Ersner-Hershfield, H., Samanez-Larkin, G. R., et al. (2011). Emotional experience improves with age: Evidence based on over 10 years of experience sampling. *Psychology and Aging, 26*, 21–33.

Carstensen, L. L., & Turk-Charles, S. (1994). The salience of emotion across the adult lifespan. *Psychology and Aging, 9*, 259–264.

Charles, S. T. (2005). Viewing injustice: Age differences in emotional experience. *Psychology and Aging, 20*, 159–164.

Charles, S. T. (2010). Strength and Vulnerability Integration (SAVI): A model of emotional well-being in later adulthood. *Psychological Bulletin, 136*, 1068–1091.

Charles, S. T., & Almeida, D. M. (2007). Genetic and environmental effects on daily life stressors: More evidence for greater variation in later life. *Psychology and Aging, 22*, 231–240.

Charles, S. T., & Carstensen, L. L. (2008). Unpleasant situations elicit different emotional responses in younger and older adults. *Psychology and Aging, 23*, 495–504.

Charles, S. T., & Carstensen, L. L. (2010). Social and emotional aspects of aging. *Annual Review of Psychology, 61*, 383–409.

Charles, S. T., Mather, M., & Carstensen, L. L. (2003). Aging and emotional memory: The forgettable nature of negative images for older adults. *Journal of Experimental Psychology: General, 132*, 310–324.

Charles, S. T., & Piazza J. R. (2009). Age differences in affective well-being: Context matters. *Social and Personality Psychology Compass, 3*, 1–14.

Charles, S. T., Piazza, J., Luong, G., & Almeida, D. M. (2009). Now you see it, now you don't: Age differences in affective reactivity to social tensions. *Psychology and Aging, 24*, 645–653.

Charles, S. T., Reynolds, C. A., & Gatz, M. (2001). Age-related differences and change in positive and negative affect over 23 years. *Journal of Personality and Social Psychology, 80*, 136–151.

Consedine, N. S., & Magai, C. (2006). Emotion development in adulthood: A developmental functionalist review and critique. In C. Hoare (Ed.), *The Oxford handbook of adult development and learning* (pp. 209–244). New York: Oxford University Press.

Davidson, R. J., Jackson, D. C., & Kalin, N. H. (2000). Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience [Special issue]. *Psychological Bulletin, 126*, 890–909.

Davis, S. W., Dennis, N. A., Daselaar, S. M., Fleck, M. S., & Cabeza, R. (2008). Que PASA?: The posterior–anterior shift in aging. *Cerebral Cortex, 18*, 1201–1209.

Diener, E., & Suh, E. M. (1998). Subjective well-being and age: An international analysis. *Annual Review of Gerontology and Geriatrics, 17*, 304–324.

Emery, L., & Hess, T. M. (2011). Cognitive consequences of expressive regulation in older adults. *Psychology and Aging, 26*, 388–396.

Gerstorf, D., Ram, N., Mayraz, G., Hidajat, M., Lindenberger, U., Wagner, G. G., et al. (2010). Late-life decline in well-being across adulthood in Germany, the United Kingdom, and the United States: Something is seriously wrong at the end of life. *Psychology and Aging, 25*, 577–485.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gross, J. J., Carstensen, L. L., Pasupathi, M., Tsai, J., Götestam Skorpen, C., & Hsu, A. Y. C. (1997). Emotion and aging: Experience, expression, and control. *Psychology and Aging, 12*, 590–599.

Grühn, D., Kotter-Grühn, D., & Röcke, C. (2010). Discrete affects across the lifespan: Evidence for multidimensionality and multidirectionality of affective experiences in young, middle-aged and older adults. *Journal of Research in Personality, 44*, 492–500.

Grühn, D., & Scheibe, S. (2008). Age-related differences in valence and arousal ratings of pictures from the International Affective Picture System (IAPS): Do ratings become more extreme with age? *Behavior Research Methods, 40*, 512–521.

Hart, S. L., & Charles, S. T. (2013). Age-related patterns in negative affect and appraisals about colorectal cancer over time. *Health Psychology, 32*, 302–310.

Hess, T. M. (2005). Memory and aging in context. *Psychological Bulletin, 131*, 383–406.

Isaacowitz, D. M., & Blanchard-Fields, F. (2012). Linking process and outcome in the study of emotion and aging. *Perspectives on Psychological Science, 7*, 3–17.

Isaacowitz, D. M., Toner, K., Goren, D., & Wilson, H. R. (2008). Looking while unhappy: Mood congruent gaze in young adults, positive gaze in older adults. *Psychological Science, 22*, 147–159.

Keil, A., & Freund, A. M. (2009). Changes in the sensitivity to appetitive and aversive arousal across adulthood. *Psychology and Aging, 24*, 668–680.

Kensinger, E. A. (2004). Remembering emotional experiences: The contribution of valence and arousal. *Reviews in the Neurosciences, 15*(4), 231–307.

Kennedy, Q., Mather, M., & Carstensen, L. L. (2004). The role of motivation in the age-related positivity effect in autobiographical memory. *Psychological Science, 15*, 208–214.

Kessler, E., & Staudinger, U. M. (2009). Affective experience in adulthood and old age: The role of affective arousal and perceived affect regulation. *Psychology and Aging, 24*, 349–362.

Kliegel, M., Jäger, T., & Phillips, L. H. (2007). Emotional development across adulthood: Differential age-related emotional reactivity and emotion regulation in a negative mood induction procedure. *International Journal of Aging and Human Development, 64*, 217–244.

Kobau, R., Safran, M. A., Zack, M. M., Moriarty, D. G., & Chapman, D. (2004). Sad, blue, or depressed days, health behaviors and health-related quality of life, Behavioral Risk Factor Surveillance System, 1995–2000. *Health and Quality of Life Outcomes, 2*, 40.

Kunzmann, U., & Grühn, D. (2005). Age differences in emotional reactivity: The sample case of sadness. *Psychology and Aging, 20*, 47–59.

Kunzmann, U., Little, T. D., & Smith, J. (2000). Is age-related stability of subjective well-being a paradox?: Cross-sectional and longitudinal evidence from the Berlin aging study. *Psychology and Aging, 15*, 511–526.

Kunzmann, U., & Richter, D. (2009). Emotional reactivity across the adult life span: The cognitive pragmatics make a difference. *Psychology and Aging, 24*, 879–889.

Lambrecht, L., Kreifelts, B., & Wildgruber, D. (2012). Age-related decrease in recognition of emotional facial and prosodic expressions. *Emotion, 12*, 529–539.

Lang, F. R., & Carstensen, L. L. (1994). Close emotional relationships in late life: Further support for proactive aging in the social domain. *Psychology and Aging, 9*, 315–324.

Lansford, J. E., Sherman, A. M., & Antonucci, T. C. (1998). Satisfaction with social networks: An examination of socioemotional selectivity theory across cohorts. *Psychology and Aging, 13*, 544–552.

Lawton, M. P. (2001). Emotion in later life. *Current Directions in Psychological Science, 10*, 120–123.

Leclerc, C. M., & Kensinger, E. A. (2011). Neural processing of emotional pictures and words: A comparison of young and older adults. *Developmental Neuropsychology, 26*, 519–538.

Levenson, R. W., Carstensen, L. L., & Gottman, J. M. (1994). The influence of age and gender on affect, physiology, and their interrelations: A study of long-term marriages. *Journal of Personality and Social Psychology, 67*, 56–68.

Levine, L. J., & Pizarro, D. A. (2004). Emotion and memory research: A grumpy overview. *Social Cognition, 22*, 530–554.

Löckenhoff, C. E., & Carstensen, L. L. (2007). Aging, emotion, and health-related decision strategies: Motivational manipulations can reduce age differences. *Psychology and Aging, 22*, 134–146.

Magai, C., Consedine, N. S., Krivoshekova, Y. S., Kudadjie-Gyamfi, E., & McPherson, R. (2006). Emotion experience and expression across the adult life span: Insights from a multimodal assessment study. *Psychology and Aging, 21*, 303–317.

Mather, M., Canli, T., English, T., Whitfield, S., Wais, P., Ochsner, K., et al. (2004). Amygdala responses to emotionally valenced stimuli in older and younger adults. *Psychological Science, 15*, 259–263.

Mather, M., & Carstensen, L. L. (2003). Aging and attentional biases for emotional faces. *Psychological Science, 14*, 409–415.

Mather, M., & Carstensen, L. L. (2005). Aging and motivated cognition: The positivity effect in attention and memory. *Trends in Cognitive Sciences, 9*, 496–502.

Mather, M., Johnson, M. K., & De Leonardis, D. M. (1999). Stereotype reliance in source monitoring: Age differences and neuropsychological test correlates. *Cognitive Neuropsychology, 16*, 437–458.

McRae, K., Gross, J. J., Weber, J., Robertson, E. R., Solkol-Hessner, P., Ray, R. D., et al. (2012). The development of emotion regulation: An fMRI study of cognitive reappraisal in children, adolescents and young adults. *Scan, 7*, 11–22.

Mikels, J. A., Larkin, G. R., Reuter-Lorenz, P., & Carstensen, L. L. (2005). Divergent trajectories in the aging mind: Changes in working memory for affective versus visual information with age. *Psychology and Aging, 20*, 542–553.

Mroczek, D. K., & Almeida, D. M. (2004). The effect of daily stress, personality, and age on daily negative affect. *Journal of Personality, 72*, 355–378.

Mroczek, D. K., & Kolarz, C. M. (1998). The effect of age on positive and negative affect: A developmental perspective on happiness. *Journal of Personality and Social Psychology, 75*, 1333–1349.

Nikitin, J., & Freund, A. M. (2011). Age and motivation predict gaze behavior for facial expressions. *Psychology and Aging, 26*, 695–700.

Ong, A. D., Rothstein, J. D., & Uchino, B. N. (2012). Loneliness accentuates age differences in cardiovascular responses to social evaluative threat. *Psychology and Aging, 27*, 190–198.

Petrican, R., Moscovitch, M., & Schimmack, U. (2008). Cognitive resources, valence, and memory retrieval of emotional events in older adults. *Psychology and Aging, 23*, 585–594.

Phillips, L. H., Henry, J. D., Hosie, J. A., & Milne, A. B. (2008). Effective regulation of the experience and expression of negative affect in old age. *Journals of Gerontology B: Psychological Sciences and Social Sciences, 63*, P138–P145.

Piazza, J., & Charles, S. T. (2006). Mental health and the baby boomers. In S. K. Whitbourne & S. L. Willis (Eds.), *The baby boomers at midlife: Contemporary perspectives on middle age* (pp. 111–148). Mahwah, NJ: Erlbaum.

Piazza, J. R., Charles, S. T., Stawski, R. S., & Almeida, D. M. (2013). Age and the association between negative affective states and diurnal cortisol. *Psychology and Aging, 28*, 47–56.

Piguet, O., Connally, E., Krendl, A. C., Huot, J. R., & Corkin, S. (2008). False memory in aging: Effects of emotional valence on word recognition accuracy. *Psychology and Aging, 23*, 85–92.

Ready, R. E., Akerstedt, A. M., & Mroczek, D. K. (2012). Emotional complexity and emotional well-being in older adults: Risks of neuroticism. *Aging and Mental Health, 16*, 17–26.

Reed, A., & Carstensen, L. L. (2012) The theory behind the age-related positivity effect. *Frontiers in Cognitive Science, 3*, 1–9.

Riediger, M., Schmiedek, F., Wagner, G., & Lindenberger, U. (2009). Seeking pleasure and seeking pain: Differences in prohedonic and contra-hedonic motivation from adolescence to old age. *Psychological Science, 20*, 1529–1535.

Samanez-Larkin, G. R., Gibbs, S. E. B., Khanna, K., Nielsen, L., Carstensen, L. L., & Knutson, B. (2007). Anticipation of monetary gain but not loss in healthy older adults. *Nature Neuroscience, 10*, 787–791.

Scheibe, S., & Blanchard-Fields, F. (2009). Effects of regulating emotions on cognitive performance: What is costly for young adults is not so costly for older adults. *Psychology and Aging, 24*, 217–223.

Scheibe, S., & Carstensen, L. L. (2010). Emotional aging: Recent findings and future trends. *Journals of Gerontology B: Psychological and Social Sciences, 65*, 135–144.

Scheibe, S., English, T., Tsai, J. L., & Carstensen, L. L. (2013). Striving to feel good: Ideal affect, actual affect, and their correspondence across adulthood. *Psychology and Aging, 28*, 160–171.

Schryer, E., & Ross, M. (2012). Evaluating the valence of remembered events: The importance of age and self-relevance. *Psychology and Aging, 27*, 237–242.

Seider, B. H., Shiota, M. N., Whalen, P., & Levenson, R. W. (2011). Greater sadness reactivity in late life. *Social Cognitive and Affective Neuroscience, 6*, 186–194.

Shamaskin, A. M., Mikels, J. A., & Reed, A. E. (2010). Getting the message across: Age differences in the positive and negative framing of health care messages. *Psychology and Aging, 25*, 746–751.

Shiota, M. N., & Levenson, R. W. (2009). Effects of aging on experimentally instructed detached reappraisal, positive reappraisal, and emotional behavioral suppression. *Psychology and Aging, 24*, 890–900.

Slessor, G., Miles, L. K., Bull, R., & Phillips, L. H. (2010). Age-related changes in detecting happiness: Discriminating between enjoyment and nonenjoyment smiles. *Psychology and Aging, 25*, 246–250.

Sliwinski, M. J., Almeida, D. M., Smyth, J., & Stawski, R. S. (2009). Intraindividual change and variability in daily stress processes: Findings from two measurement-burst diary studies. *Psychology and Aging, 24*, 828–840.

St. Jacques, P., Dolcos, F., & Cabeza, R. (2009). Effects of aging on functional connectivity of the amygdala during subsequent memory for negative pictures: A network analysis of fMRI data. *Psychological Science, 20*, 74–84.

St. Jacques, P., Dolcos, F., & Cabeza, R. (2010). Effects of aging on functional connectivity of the amygdala during negative evaluation: A network analysis of fMRI data. *Neurobiology of Aging, 31*, 315–331.

St. Jacques, P. L., Bessette-Symons, B., & Cabeza, R. (2009). Functional neuroimaging studies of aging and emotion: Fronto-amygdalar differences during emotional perception and episodic memory. *Journal of the International Neuropsychological Society, 15*, 819–825.

Stone, A. A., Schwartz, J. E., Broderick, J. E., & Deaton, A. (2010). A snapshot of the age distribution of psychological well-being in the United States. *Proceedings of the National Academy of Sciences USA, 107*(22), 9985–9990.

Story, T. N., Berg, C. A., Smith, T. W., Beveridge, R., Henry, N. J. M., & Pearce, G. (2007). Age, marital satisfaction, and optimism as predictors of positive sentiment override in middle-aged and older married couples. *Psychology and Aging, 22*, 719–727.

Streubel, B., & Kunzmann, U. (2011). Age differences in emotional reactions: Arousal and age-relevance count. *Psychology and Aging, 26*, 966–978.

Teachman, B. A. (2006). Aging and negative affect: The rise and fall and rise of anxiety and depressive symptoms. *Psychology and Aging, 21*, 201–207.

Tsai, J. L., Levenson, R. W., & Carstensen, L. L. (2000). Autonomic, expressive and subjective responses to emotional films in younger and older adults of European American and Chinese descent. *Psychology and Aging, 15*, 684–693.

Tucker, A. M., Feuerstein, R., Mende-Siedlecki, P., Ochsner, K. N., & Stern, Y. (2012). Double dissociation: Circadian off-peak times increase emotional reactivity; Aging impairs emotion regulation via reappraisal. *Emotion, 12*, 869–874.

Uchino, B. N., Birmingham, W., & Berg, C. A. (2010). Are older adults less or more physiologically reactive?: A meta-analysis of age-related differences in cardiovascular reactivity to laboratory tasks. *Journals of Gerontology B: Psychological Sciences and Social Sciences, 65*, P154–P162.

Urry, H. L., & Gross, J. J. (2010). Emotion regulation in older age. *Current Directions in Psychological Science, 19*, 352–357.

Villamil, E., Huppert, F. A., & Melzer, D. (2006). Low prevalence of depression and anxiety is linked to statutory retirement ages rather than personal work exit: A national survey. *Psychological Medicine, 36*, 999–1009.

Weierich, M. R., Kensinger, E. A., Munnell, A. H., Sass, S. T., Dickerson, B. D., Wright, C. I., et al. (2011). Older and wiser?: An affective science perspective on age-related challenges in financial decision making. *Scan, 6*, 195–206.

Westlye, L. T., Walhovd, K., B., Dale, A. M., Bjørnerud, A., Due-Tønnessen, P., Engvig, A., et al. (2010). Life-span changes of the human brain white matter: Diffusion tensor imaging (DTI) and volumetry. *Cerebral Cortex, 20*, 2055–2068.

Wrzus, C., Müller, V., Wagner, G. G., Lindenberger, U., & Riediger, M. (2013). Affective and cardiovascular responding to unpleasant events from adolescence to old age: Complexity of events matters. *Developmental Psychology, 49*, 384–397.

Wurm, L. H., Labouvie-Vief, G., Aycock, J., Rebucal, K. A., & Koch, H. E. (2004). Performance in auditory and visual emotional Stroop tasks: A comparison of older and younger adults. *Psychology and Aging, 19*, 523–535.

Zulfiqar, U., Jurivich, D. A., Gao, W., & Singer, D. H. (2010). Relation of high heart rate variability to healthy longevity. *American Journal of Cardiology, 105*, 1181–1185.

PART V

SOCIAL ASPECTS

CHAPTER 14

Social Baseline Theory and the Social Regulation of Emotion

James A. Coan
Erin L. Maresh

To survive and reproduce, organisms must take in more energy than they expend, a principle of behavioral ecology called *economy of action* (Krebs & Davies, 1993). *Social baseline theory* (SBT), a framework based on this principle, organizes decades of observed links between social relationships, health, and well-being, in order to understand how humans utilize each other as resources to *optimize* individual energy expenditures. This general strategy helps us manage the costs of our very long period of ontogenetic development and, we argue, the many behavioral and psychological capabilities of our uniquely powerful and costly brain (Smith, 2003). Below, we review evidence that social relationships serve the energy-saving functions that we claim and describe the reasons SBT refers to social proximity as a "baseline" condition. For this chapter we place special emphasis on the role of social proximity in the regulation of emotion. In our view, the social regulation of emotion serves, in the aggregate and on average, to decrease the cost of coping with many of life's difficulties (Cohen & Hoberman, 1983; Cohen & McKay, 1984)—a function that involves the brain's ability to use both internal and external information to make "bets" about how to deploy its own resources in light of expected returns. This perspective (1) highlights the importance of emotion as a source of information about current and predicted resources, and (2) entails a view of psychological measurement that extends beyond the individual to systemic processes within dyads and groups (Schilbach et al., in press).

Social Proximity and the Economy of Action

The economy of action suggests that adaptations to prevailing environmental conditions must, at the very least, ensure that more energy is acquired than lost (Krebs & Davies, 1993). The alternative—more energy out than in—leads ultimately to death. But because environments are rife with danger and competition, the economy of action tends toward optimization, where resources are both acquired *and conserved* whenever possible. For example, foraging animals optimize energy intake per unit of time by abandoning even plentiful food sources if the amount of energy (e.g., calories) acquired is not optimal (MacArthur & Pianka, 1966; Schmidhempel, Kacelnik, & Houston, 1985). Optimization strategies

are likely mediated through changes in sensory perception. So simply wearing a heavy backpack can make distances appear farther away and hills seem steeper (Stefanucci, Proffitt, Banton, & Epstein, 2005). Indeed, it is increasingly apparent that perception is influenced by not only the properties of sensory stimuli but also emotions—emotions that are themselves embodied instantiations of prevailing circumstances, goals, and physiological states (Stefanucci, Proffitt, Clore, & Parekh, 2008).

SBT is rooted in the proposition that, for humans, social proximity and interaction powerfully optimize energy expenditures (e.g., actual and perceived effort) devoted to navigating potentially dangerous environments. Moreover, the best evidence suggests these effects are mediated through relatively automatic, bottom-up, unconditioned perceptual mechanisms that modulate—and regulate—emotional responding, as opposed to mechanisms often associated with the *self*-regulation of emotion (e.g., the dorsolateral [dlPFC] and ventromedial prefrontal cortex [vmPFC]). SBT invokes the economy of action here, too, by suggesting that perceptual mechanisms of the central nervous system are "inexpensive"[1] when compared to more effortful, self-regulatory, vigilant, "future-thinking" processes associated with the operation of the PFC—a difference that leads to both phylogenetic and ontogenetic pressures to achieve regulatory ends by perceptual and hence social means. We suggest that the ability to depend on others for as many costly processes as possible leads to (1) decreased overall cost in dealing with an uncertain and potentially deadly environment, and (2) pressure—throughout the lifespan and over the course of evolution—to form and maintain close social relationships.

Before going any further, we would like to emphasize three important points. First, our view of social emotion regulation does *not* imply that any and all effects an individual's behavior may have on the emotional behavior of another constitute *regulatory* effects. If one individual insults or compliments another, either behavior could cause the receiver to activate an emotional response. This would not, however, constitute an instance of emotion regulation. An individual regulates another's emotions when his or her behavior (which might encompass social history with the other person, an offer of aid, or simply close physical proximity) influences another's emotional response to some *additional* current or potential stimulus or situation. Second, we acknowledge that people vary in the extent to which they both seek out and derive benefit from social contact (leading to individual differences in the broad processes described here). But on average and in the aggregate, it is probably the case that for humans and other social mammals, proximity to social resources reduces the net cost of survival. Third, although most of the empirical evidence we review entails the regulatory impact of social proximity and interaction on the regulation of negative affect, we do not intend to argue that negative affect is the only broad form of emotional responding that can be regulated. Indeed, below we address some instances of the social regulation of positive emotion.

Our Social Baseline

There is little doubt that supportive social behaviors can modify or quell our subjective, behavioral, and physiological responses to stress (Holt-Lunstad, Smith, & Layton, 2010). Such effects are likely rooted in human phylogeny. Indeed, Beckes and Coan (2011) and others (Berscheid, 2003; Brewer & Caporeal, 1990) have suggested that the dominant ecology to which humans are adapted is not any one terrain, diet, or climate, but rather *each other*. One of the defining features of human beings—our adaptability—is yoked to our ability to cooperate with others, an ability that was likely shaped by periods of great environmental instability (Richerson, Bettinger, & Boyd, 2005). Cooperative behavior was likely selected as a means of managing the energy costs of our exceedingly expensive bodies, brains, and activities (Hill et al., 2011; McNally, Brown, & Jackson, 2012; Moll & Tomasello, 2007). So the first sense in which SBT refers to a social *baseline* is that social relatedness and its psychological correlates constitute the normal, baseline ecology of the functional human brain. That is, the human brain is designed to operate within a relatively predictable network of social relationships characterized by famil-

iarity, shared intentionality, and interdependence (Tomasello, Carpenter, Call, Behne, & Moll, 2005).

The other sense in which SBT refers to the social "baseline" results from work using functional magnetic resonance imaging (fMRI; Coan, Schaefer, & Davidson, 2006), which suggests that social proximity and interaction regulate many aspects of the brain's response to potential threats. Specifically, the authors subjected 16 married women to the threat of mild electric shock under three conditions, counterbalanced across subjects: while alone in the scanner, while holding a stranger's hand, and while holding her husband's hand. The brain was maximally active while facing the threat alone, exhibiting a set of responses replicating a large body of work on the brain's response to threat, fear, stress, and pain (Bishop, Duncan, Brett, & Lawrence, 2004; Brooks, Nurmikko, Bimson, Singh, & Roberts, 2002; Dedovic et al., 2005; Stark et al., 2003). As levels of contact with perceived social resources increased, however, many of these activations either decreased in intensity or vanished altogether.

For example, although both spouse and stranger handholding caused decreased threat responding in regions associated with the regulation of bodily arousal and the mobilization of behavioral action plans—such as the ventral anterior cingulate cortex, supramarginal and postcentral gyri, and posterior cingulate—only spouse hand-holding was specifically associated with additional attenuation in circuits associated with effortful emotion regulation and threat-related homeostatic functions, such as the right dlPFC and superior colliculus. Most strikingly, women inhabiting the highest quality relationships realized dramatic attenuations in all the aforementioned threat-responsive regions in addition to those supporting subjective suffering and the release of stress hormones, such as the right anterior insula and hypothalamus. Putting it all together, there appeared to be a relatively linear, monotonic decrease in the degree of threat-related neural processing as one progressed from being alone to being with anyone (stranger or spouse), to being with a spouse, to being with a spouse in a very high-quality relationship. This suggests that the perception of threat decreases as a function of not only proximity to social resources but also the perceived *quality* of those resources.

From the perspective of SBT, the alone condition used in the hand-holding paradigm described earlier is not a "baseline" condition against which the experimental hand-holding conditions are compared. On the contrary, the reverse is true—the hand-holding conditions are closer to the environmental conditions to which the human brain is adapted, and the alone condition is farther away. This conclusion forms part of the basis of SBT—that social resources decrease the perceived cost of managing a threatening environment, providing a primary and largely unconditional opportunity for economizing both neural and behavioral activity.

Importantly, these effects are not dependent upon physical touch per se. For example, there is less hypothalamic activity during social rejection among individuals asked to imagine an attachment figure (Karremans, Heslenfeld, van Dillen, & Van Lange, 2011). Moreover, imagining a strong attachment figure seems to have resulted in not more but less activity in frontal regions supporting self-regulatory effort, suggesting that even imagining the presence of a secure attachment figure may regulate one's emotions in such a way as to conserve perceived resources.

Social Systems Regulate the Emotions of Individuals

Critically, what we experience and categorize as *emotion* powerfully impacts the way we perceive and engage with the world (Stefanucci, Gagnon, & Lessard, 2011). Emotional processes carry vital, often implicit, and embodied information about the goodness and badness of things (Clore & Tamir, 2002)—information that modifies perception and guides action (Barrett & Bar, 2009; Schwarz & Clore, 1983). It follows that emotions are indicators of, among other things, the tension between perceived personal resources and perceived environmental demands (Moore, Vine, Wilson, & Freeman, 2012; Tomaka, Blascovich, Kelsey, & Leitten, 1993). In this way, emotion is probably at or near the center of decisions that economize activity (Bechara, 2011;

Stefanucci et al., 2008). Accordingly, SBT suggests that the brain uses affective, experiential, conceptual, and contextual knowledge—in effect, emotion (Barrett, 2006; Lindquist & Barrett, 2008)—to bias perception in ways that guide actions toward more favorable outcomes (Beckes & Coan, 2011; Coan, 2008).

Emotion has been identified by others as a key link between social relationships and health. For example, the *social buffering hypothesis* suggests that social relationships can provide support that is tangible, such as material support when facing loss of income, appraisal-based support, such as when familiar others reduce the incidence or intensity of threat-related appraisals, or emotional support, such as when familiar others repair threats to self-esteem or increase a sense of felt belonging (Cohen & McKay, 1984). It has been suggested further that social relationships grow into part of the brain's understanding of itself and its available resources (Beckes, Coan, & Hasselmo, 2012). Moreover, as it makes predictions about the potential cost of coping with a given situation, the brain follows Bayesian rules of inference (Friston, 2010; Knill & Pouget, 2004), in which judgments about the level of personal resources to deploy are based on (1) the current situation (particularly constraints, risks, and opportunities), (2) predicted possible future situations, (3) situational goals, (4) current energy states, and (5) expected future energy states (Salinas, 2011).

For example, hills appear to be less steep when standing next to a friend (Schnall, Harber, Stefanucci, & Proffitt, 2008), and this effect is correlated with friendship duration: the longer the friendship, the less steep the hill appears to be. The perception that the hill is less steep serves as a marker that one has, in effect, more bioenergetic capital in one's budget—capital that can either be spent more freely walking up the hill, or conserved over time. Diverse studies hint at similar conclusions regarding the brain's Bayesian properties. For example, Kraus, Huang, and Keltner (2010) have observed that more frequent physical touching among professional basketball players corresponds with increased late-season performance, even after accounting for player status, preseason expectations, and early season performance. Others have observed that one of the best predictors of collective IQ is not the IQs of individual group members but rather the degree to which each member is sensitive to social cues expressed on the face (Woolley, Chabris, Pentland, Hashmi, & Malone, 2010). From the perspective of SBT, each of these findings is attributable to the way social resources help to conserve costly vigilance and self-regulation efforts, either by imposing less interference with motor and perceptual activity (in the case of players in the NBA), or by devoting those resources to solving other kinds of problems (in the case of collective IQ). But the question of *why* social resources economize human neural and behavioral activity remains. We propose at least two broad, distal mechanisms by which this is accomplished: risk distribution and load sharing.

Risk Distribution

In group settings, many species adjust the level of their energy expenditure in accordance with the size of the group they inhabit (Krause & Ruxton, 2002). This benefit of group living is called *risk distribution* (Coan, 2008), in that environmental risk is probabilistically distributed across the group rather than being concentrated on one individual. A clear example of this can be seen in *optimal foraging theory*, which proposes that animals forage in a way that maximizes energy intake per unit of time (MacArthur & Pianka, 1966). For example, animals that forage must spend a certain amount of energy maintaining vigilance for predators. Foraging in a larger group decreases the burden of vigilance on any one member, and indeed, in many species, the number of individuals that are vigilant for predators at any given time decreases as group size increases (Elgar, 1989; Roberts, 1996). With regard to the social regulation of emotion, we can construe threat vigilance as a form of negative affect—or at least as the result of a negative affect process that reflects the demand for vigilance. If true, then social proximity can be viewed as decreasing vigilance behavior via the regulation of negative affect. Evidence for this can even be seen in the modulation by group size of physi-

ological indicators of stress in nonhuman animals. For example, salivary cortisol concentration in sheep is higher when the flock size is smaller (Michelena et al., 2012). Risk distribution suggests further that the number of proximal conspecifics decreases negative affect, because doing so conserves effort that would otherwise be spent either on vigilance, self-regulation, or both. This is exemplified in humans in the hand-holding study discussed earlier, by the finding that holding any hand—even that of a stranger—reduces neural activity related to threat (Coan et al., 2006), especially in regions supporting responses to acute threats (Mobbs et al., 2007).

Interestingly, the individual benefits of group living described earlier often benefit the group as well, such that as each individual's vigilance effort decreases, total group vigilance actually increases (Bertram, 1978; Pulliam, Pyke, & Caraco, 1982) and time to detect predators decreases (Siegfried & Underhill, 1975). It is important to note, however, that group living introduces certain risks and stressors as well, such as increased competition for resources, increased likelihood of disease transmission, and increased conspicuousness (Alexander, 1974). Groups therefore iteratively move toward an optimal size given the availability of food and other resources (Higashi & Yamamura, 1993). Note that risk distribution concerns only the number of members in a group and makes no reference to the nature and quality of the relationships between the group members. Coan (2008) has speculated that the brain is sensitive to risk distribution as a general purpose strategy that employs Bayesian-like calculations to assess the cost-effectiveness of affective behaviors at any given time.

Load Sharing

Load sharing builds on the benefits of risk distribution by adding information that increases certainty about the availability of a given social resource—namely, familiarity, reliability, and interdependence. The presence of an individual with whom one has established a close relationship not only probabilistically reduces one's risk of environmental threat but also signals access to a person who will use his or her own resources on one's behalf, thus further reducing the need to expend one's own energy, and commensurably decreasing the need, for example, for negative affect. In other words, a close companion can be trusted to share an interest in one's well-being. He or she may provide additional vigilance not only in some general sense but also specifically, with one's own welfare in mind (Davis, 1984). A close companion may share resources (e.g., Rogers & DeBoer, 2001), help care for young (e.g., Ehrenberg, Gearing-Small, Hunter, & Small, 2001), and help when one is sick or injured (e.g., Townsend & Franks, 1995). These additional benefits provide a much greater opportunity for economizing neural and behavioral activity, as can be seen in the enhanced effect of holding hands with a close friend or a spouse (Coan, Beckes, & Allen, 2013; Coan et al., 2006).

SBT maintains that load sharing impacts decisions about personal resource budgeting in part by altering the way the brain encodes what constitutes the "self," with implications for the level of resources the self is able to access. This is consistent with the observation that the neural representations of threats directed at the self are highly correlated with those directed at friends, implying a kind of "involuntary breach of individual separateness" (Langer, 1974, p. 129) that does not seem to generalize to strangers (Beckes et al., 2012). In this way, an individual's social support system can be construed as an extension of the self and, in turn, of how one perceives and interacts with the world.

Capitalization

Importantly, social proximity and interaction serve not only to down-regulate negative emotions, but also to maintain and increase positive, approach-related emotions. Admittedly, our research has focused on the social regulation of response to threat, but we believe the basic ideas behind SBT are generalizable to positive emotions as well. People frequently expend energy to induce, maintain, or even dampen experiences of positive emotion, and this is reflected in neural activity in prefrontal areas similar to those seen in the self-regulation of negative emotion (Kim & Hamann, 2007). Social

relationships may act to conserve effortful positive self-regulation, possibly by reducing reliance on prefrontal activity and increasing activity in reward circuits. Interestingly, neural activity in the medial PFC decreases in mothers observing pictures of their children, and in adults observing pictures of their romantic partner, as compared to when they are observing pictures of a friend (Bartels & Zeki, 2000, 2004). Moreover, positive responses by a relational partner to one's own positive news, a process known as *capitalization*, may be a mechanism by which social relationships enhance positive emotions in ways that are less individually effortful. Capitalization increases daily positive affect over and above the impact of the positive event itself (Gable, Reis, Impett, & Asher, 2004; Langston, 1994). Furthermore, capitalization may strengthen trust and prosocial behavior, even when one is interacting with a stranger (Reis et al., 2010). Following the SBT model, then, receiving supportive, positive feedback from a partner may signal an increase in both personal and social resources. It is interesting to note that the perceived responsiveness of one's partner during positive events may be more strongly linked with the relationship's well-being and longevity than perceived responsiveness during negative events (Gable, Gonzaga, & Strachman, 2006).

Importantly, SBT views all forms of social emotion regulation as systemic and dynamic, challenging the widely held assumption that the basic unit of analysis in human psychology is the single individual (Beckes & Coan, 2011). By extending the unit of analysis to the dyad or group, social interaction and proximity emerge as adaptive strategies rooted in the sharing of efforts that, left only to each individual member of a dyad or group, would be redundant, costly, and inefficient. Ultimately, it may be that dyads or groups benefit from social emotion regulation at least as much as the individuals who inhabit them (McComb, Moss, Durant, Baker, & Sayialel, 2001). In considering mechanisms for these dyadic or group effects, social emotion regulation can be mediational, reflecting a direct intervention on an emotional process by social resources, or moderational, modifying the perception of potentially emotional situations. In the following sections, we briefly describe and propose potential mechanisms for the distinction.

Mediating Mechanisms of Social Emotion Regulation

As mediators of emotion regulation (Baron & Kenny, 1986), social resources serve as a proximal mechanism through which emotion regulation effects are achieved. Attachment theory provides the quintessential example of socially regulated emotion in its description of mother–child attachment interactions, in which infants seek attachment figures during periods of distress and are soothed by their caregiver's presence (Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1969/1982). A common example might be a child's vaccination procedure, where the child—distressed at the prospect of receiving a shot—calls for the mother's support. In response, the mother may engage in soothing behaviors like hand-holding and offering reassuring words in a calm tone of voice. Here, the mother's behavior provides an obvious mechanism through which the child's distress is attenuated. Specifically, the child identifies a potential threat (the needle), and this threat causes two reactions, distress and the seeking of support from the mother. In turn, the mother's support behavior exerts a down-regulatory influence on the distress response.

The mother–child dynamic is a particularly powerful example of how social resources can mediate emotion regulation in part because the child in the example is developmentally limited in his or her self-regulatory abilities, a limitation that is itself rooted in the slow development of neural systems that will eventually serve the child's self-regulatory needs (Coan, 2008). That the mother's soothing works is obvious and well documented (Cassidy & Shaver, 2008). Less obvious is that the child is relatively *incapable* of emotion regulation without the mother. Thus, we can view the mother and child as an interactive system that is intimately linked in an emotion activation–regulation dynamic. Importantly, however, the mother–child behavioral and experiential dynamics described by attachment theorists play out similarly within adult relationships (Mikulincer & Shaver, 2007), where stressful situations motivate social proximity, and

social proximity lowers autonomic arousal, attenuates hypothalamic–pituitary–adrenal (HPA) axis activity, and improves immune function (Baron, Cutrona, Hicklin, Russell, & Lubaroff, 1990; Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Of interest with regard to adult relationships is that, unlike mother–infant dyads, two or more adults each possess fully developed and functioning brains. Thus, although children require maternal support for emotion regulation, adults appear to utilize each other for similar purposes more strategically.

A major focus of our work has been the identification of proximal neural mechanisms linking social relationships to decreased threat responding and, in turn, increased health and well-being. Although many candidate mechanisms have been postulated, few or none have been definitively identified. Recently, Eisenberger and colleagues (2011) provided what they characterized as mediational evidence for the social regulation of emotion by portions of the vmPFC. In this work, participants were shown pictures of a romantic partner, pictures of strangers, or pictures of objects, while receiving painful stimulation. Putative pain-related activations in regions such as the dorsal anterior cingulate cortex (dACC) and anterior insula were less active while participants viewed images of a romantic partner. Moreover, the vmPFC was *more* active while participants viewed romantic partners, even during pain stimuli, and vmPFC activation was positively associated with relationship duration and perceived partner support, while negatively associated with both subjective pain ratings and pain-related neural activity. In a similar study, Younger, Aron, Parke, Chatterjee, and Mackey (2010) collected brain images from 15 participants as they experienced moderate levels of thermal pain while they viewed pictures of their romantic partner, pictures of attractive strangers, or engaged in a distraction task, hypothesizing that pictures of romantic partners would attenuate pain processing via putative "reward systems." Indeed, viewing pictures of romantic partners both reduced subjective pain reports and attenuated circuits associated with pain processing. Moreover, subjective pain reports were inversely correlated during romantic partner picture viewing with the activation of neural circuits associated with reward processing (e.g., the caudate nucleus, the nucleus accumbens) and effortful self-regulation (e.g., the dlPFC).

Thus, emerging evidence for proximal mechanisms supporting socially mediated emotion regulation appears to include regions associated with both the automatic and effortful *self*-regulation of emotion (vmPFC and dlPFC, respectively), as well as putative reward circuits (e.g., the nucleus accumbens). However, our laboratory has not been able to produce similar results, in our original study of hand-holding by married couples (Coan et al., 2006), in a more recent study of hand-holding by platonic friends (Coan et al., 2013), or in a study of the regulation of threat responses in children by the presence of adult caregivers (Conner et al., 2012). Indeed, in the latter study, we observed decreased activation specifically in the vmPFC and ventrolateral PFC (vlPFC), where we used a region of interest (ROI) approach for the purpose of addressing the question of prefrontal mediation of social support. Ultimately, we have thus far failed to reliably identify *any* regions of the brain that are normatively more active under threat conditions during supportive presence or hand-holding than while alone. Moreover, none of the regions identified by Eisenberger et al. (2011) and Younger et al. (2010) are negatively correlated with any of the threat-responsive circuits apparently downregulated in our work by hand-holding.

What may account for these apparently inconsistent results? The answer, we think, has to do with the difference between touch and picture viewing. Specifically, there is ample evidence that touch acts as an unconditioned or primary stimulus in social animals, not least humans (Francis et al., 1999), who may also use touch to exchange important social information (Gazzola et al., 2012; Morrison, Löken, & Olausson, 2010). By contrast, picture viewing likely involves associative learning, particularly involving the expected value of the photographic image—a task often mediated through vmPFC activity (Kable & Glimcher, 2007). Thus, vmPFC activity may be necessary when utilizing photographs of loved ones to regulate emotion. But a careful reading of Younger et al. (2010) reveals that, in addition to showing their participants pictures

of loved ones, they instructed participants to "focus on the picture and think about the displayed person" (p. 2), effectively encouraging self-mediated rather than socially mediated emotion regulation. Ultimately, we would argue that both the Eisenberger et al. (2011) and Younger et al. (2010) experiments manipulated self-regulatory systems and are not best understood as examples of socially mediated emotion regulation at all.

Moderating Mechanisms of Social Emotion Regulation

Strictly speaking, the hand-holding study discussed earlier may also provide an example of socially *moderated* emotion regulation, in part because the provision of social support preceded the presentation of threat cues, thus potentially altering the perception of those threat cues and obviating the activation of portions of the threat response. This illustrates one of the many ways in which social proximity and interaction can be viewed as moderating emotional responses and self-regulation needs, acting as third variables that modify the conditions under which emotional responses and self-regulation strategies are either called upon or maximally effective. Taking the example of the vaccination procedure discussed earlier, it may be the case that, as in our hand-holding experiments, the mother holds the child's hand not in response to the child's obvious distress but in anticipation of the child's potential distress. In this case, maternal hand-holding may cause relaxation of the child's vigilance processing, rendering the appearance of the needle less threatening when it does arrive.

Past social experiences also impact an individual's self-regulation requirements and capabilities (Allen, Moore, Kuperminc, & Bell, 1998; Prinstein & La Greca, 2004; Weaver et al., 2004). For example, ongoing current daily social support may moderate the perception of threats encountered while alone, which may in turn reduce the perceived need for engaging self-regulation capabilities (Eisenberger, Taylor, Gable, Hilmert, & Lieberman, 2007). Similarly, mental representations of loved ones may be used as methods by which one can exert self-regulatory strategies related to distraction or reappraisal. Any or all of these possibilities can be modeled by SBT, such that past social experiences or even prevailing social conditions alter an individual's perception of potential threats and rewards in his or her immediate environment even when he or she is alone at the occasion of measurement.

A number of researchers have argued that early childhood experiences critically influence the ways in which individuals view social relationships later in life. For humans and many other species, *filial bonding*—bonding with siblings and caregivers—occurs rapidly, unconditionally, and during a period of neural development that may hold powerful consequences for subsequent functioning (Bowlby, 1969/1982; Coan, 2008). In infants, the PFC is particularly underdeveloped in comparison to that of adults, and its development takes approximately two decades to complete. The human brain in general grows exponentially during just the first 2 years of life (Franceschini et al., 2007)—growth reflected in the brain's consumption of glucose, which continues to be approximately double that of adults until 10 years of age (Chugani, 1998). Throughout this early development, the brain is in effect adapting itself to environmental contingencies through a process of axonal, dendritic, and synaptic "pruning"—a process that is itself dependent upon neural activity associated with environmental stimulation (Cancedda et al., 2004; Reichardt, 2006). Put another way, the brain in early development adheres roughly to the colloquialism "use it or lose it"; environmental demands shape a developing brain prepared for a wide range of behavioral possibilities. By the process of pruning, the brain's responses to prevailing contextual demands become more fixed and hence more rapid—it becomes adapted ontogenetically to its expected environment (Hebb, 1949; Posner & Rothbart, 2007).

During this development, interactions between social experiences, self-regulatory activity and socially bound activations in medial prefrontal, temporoparietal, and posterior temporal cortices, as well as in the amygdala, ventral tegmentum, nucleus accumbens, periaqueductal gray and elsewhere, may shape expectations about the availability of social resources, opportunities for affiliation, expectations about the reliability of close relational partners, and so on (Lieberman, 2007). This in turn is likely

to shape individual differences in the way social resources are approached and maintained, perhaps especially in situations that blend social proximity with potential stressors. Indeed, it is just this sort of experience-based developmental divergence that early attachment researchers described as manifesting in "working models" of attachment figures, a theoretical perspective expressed in more recent terms as *attachment styles* (Ainsworth et al., 1978; Main, 1996; Mikulincer & Shaver, 2007).

A large and growing body of research has documented effects consistent with early psychosocial brain development. For example, primates are highly sensitive to early proximity to and interaction with caregivers (Harlow, 1958). In rats, maternal grooming may influence the methylation of glucocorticoid receptor genes throughout the hippocampus and possibly elsewhere, influencing in turn the stress reactivity of the pups throughout their lives and even into subsequent generations (Weaver et al., 2004). Evidence suggests a similar methylation process may occur in humans (McGowan et al., 2009; van IJzendoorn, Bakermans-Kranenburg, & Ebstein, 2011). Hofer (2006) has proposed that early interactions with caregivers slowly evolve from the indirect regulation of sensorimotor, thermal, and nutrient functions via responses to expressed emotion. At first the infant is totally dependent upon caregivers for access to primary reinforcers (food, water, warmth, touch). The only way for the infant to access these resources is to get the caregiver's attention with expressed emotion. A predictable pattern emerges where the infant cries out when a basic resource is needed, followed by the caregiver's response, after which the infant stops crying. As the infant develops, his or her emotional repertoire expands to include preferences beyond simple metabolic or thermal needs, but the process of caregiver responsiveness continues in much the same way, until it can be said that the emotional feelings and expressions are themselves the targets of the caregiver's responses.

Attachment theorists have had perhaps the most to say about how social experiences contribute to individual differences in the social regulation of emotion (Mikulincer & Shaver, 2005; Mikulincer, Shaver, & Pereg, 2003). Specifically, attachment theory posits the existence of trait-like expectations—the aforementioned attachment styles—regarding the availability of social resources. Dominant views of attachment style can be expressed as two independent axes representing tendencies toward social anxiety and social avoidance. Individuals low in both are said to be *secure*, in that their appraisals of potential threats and views of emotional disclosure tend to be less negative (Mikulincer & Florian, 1998; Mikulincer & Orbach, 1995). Critically, such individuals are likelier to seek out, utilize, and benefit from emotional support from others (Fraley & Shaver, 2000; Larose, Bernier, Soucy, & Duchesne, 1999; Rholes, Simpson, & Grich Stevens, 1998). Secure individuals may even benefit more from capitalization attempts: Shallcross, Howland, Bemis, Simpson, and Frazier (2011) found that insecurely attached individuals more often underestimated the responsiveness of their partners when sharing a positive event than did securely attached individuals. Although neuroscientific investigations of attachment style are rare, Gillath, Bunge, Shaver, Wendelken, and Mikulincer (2005) have observed that greater trait attachment anxiety is associated with more activity in the dACC and temporal pole, areas associated with alarm and negative affect—and decreased activity in the orbitofrontal cortex—when participants are asked to think about a threatening relationship scenario.

Future Directions

We feel these perspectives on social relationships and social emotion regulation have implications that extend both to basic and applied domains of psychological science, and that the ecological framework of SBT may be useful for understanding a number of important new findings. Clinically, we have recently noted that SBT holds implications for how we understand the frontolimbic dysfunction and emotional dysregulation that characterize borderline personality disorder (Hughes, Crowell, Uyeji, & Coan, 2012), and we believe that SBT may be relevant to the development and maintenance of other psychopathologies as well. For example, although speculative, it is possible that the disruptions in social relationships

that characterize autism spectrum disorders result in an overreliance on self-regulation, leading to chronic depletion and the use of self-regulatory strategies associated with small children. We suggest further that SBT may be useful for understanding emerging approaches to relationship therapy that are proving to be efficacious for a number of problems traditionally treated by individually oriented psychotherapies alone (Monson et al., 2012; Naaman, Radwan, & Johnson, 2009).

Ultimately, we emphasize that human life is in many ways defined by social relationships. According to SBT, a key function of social relationships is the social regulation of emotion. Emotions provide rapid, embodied information about current states and contextual demands (Clore & Tamir, 2002), guiding decision making and modifying perception (Bechara, 2011; Stefanucci et al., 2008). The modification of perception via emotional responding is itself yoked to the management of bioenergetic resources for the purpose of economizing action (Stefanucci et al., 2011). Because emotion is powerfully regulated by social proximity (Coan et al., 2013; Coan et al., 2006), all of the above is controlled by proximity to social resources (Schnall et al., 2008). Thus, the impact of social relationships ripples through virtually everything humans do, because social relationships are tightly linked to emotional processes, and emotional processes inform, bias, and direct many, if not most, of the brain's activities. This may not always work in ways that are broadly socially desirable. For example, the social regulatory processes described throughout this chapter may contribute to phenomena such as social loafing (Karau & Williams, 1993), and the "outsourcing" of health behaviors (Fitzsimons & Finkel, 2011). The impact of social relationships—manifest as perceived proximity, interaction, and history—is thus likely to be felt within any specific subdomain of psychological science, suggesting that any such subdomain that does not account for social processes will be limited in important ways. On the other hand, the extent to which social processes impact psychological phenomena opens wide the possibilities for new testable hypotheses and exciting programs of research.

Note

1. Accumulating evidence from a diverse collection of laboratories suggests the psychological functions supported by the cortex—including the PFC—are computationally, and hence bioenergetically, expensive, especially relative to more computationally basic perceptual processes (Dietrich, 2009; Halford, Wilson, & Phillips, 1997, 1998). At the very least, psychological processes supported by the PFC are often subjectively experienced as effortful (Muraven & Baumeister, 2000; Sheppes, Catran, & Meiran, 2009). Sustained prefrontally mediated behaviors (e.g., attention, self-control, vigilance, behavioral inhibition) cause subjective exhaustion and a steady decrease in the ability to perform those very behaviors (Baumeister, Vohs, & Tice, 2007; Vohs & Heatherton, 2000). Because the brain maintains a fairly constant metabolic rate regardless of task (Sokoloff, Mangold, Wechsler, Kennedy, & Kety, 1955), an extended reliance on prefrontally mediated processes may reduce resources (e.g., blood) available to other regions. Indeed, there is evidence of intrabrain competition for blood, even within the cortex (Dietrich, 2009). Although open to serious criticism (Kurzban, 2010), it may yet be possible that the PFC even places a higher metabolic demand on the brain's resources. For example, the ratio of glia to neurons—a ratio that covaries with metabolic demand—is higher in the PFC of humans than in other primates (Sherwood et al., 2006). The PFC is among the last neural systems to develop fully (Fuster, 2002) and among the first to show functional deficits in the case of extreme emotional stress, malnourishment, intoxication, addiction, old age, and even exercise (Arnsten, 2009; Del Giorno, Hall, O'Leary, Bixby, & Miller, 2010; Goldstein & Volkow, 2011; Lipina & Colombo, 2009; Paxton, Barch, Racine, & Braver, 2008). All of this is despite a general consensus that the human PFC is one of the defining characteristics of human phylogeny (Ehrlich & Ehrlich, 2008)—a resource shaped over evolutionary development that allows humans a level of abstraction, creativity, vigilance, self-control, and anticipatory judgment that is unprecedented in the animal kingdom and largely responsible for the globally dominant status that humans enjoy.

References

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation.* Hillsdale, NJ: Erlbaum.

Alexander, R. D. (1974). The evolution of social behavior. *Annual Review of Ecology and Systematics, 5*, 325–383.

Allen, J. P., Moore, C., Kuperminc, G., & Bell, K. (1998). Attachment and adolescent psychosocial functioning. *Child Development, 69*(5), 1406–1419.

Arnsten, A. F. T. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience, 10*(6), 410–422.

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*(6), 1173–1182.

Baron, R. S., Cutrona, C. E., Hicklin, D., Russell, D. W., & Lubaroff, D. M. (1990). Social support and immune function among spouses of cancer patients. *Journal of Personality and Social Psychology, 59*(2), 344–352.

Barrett, L. F. (2006). Solving the emotion paradox: Categorization and the experience of emotion. *Personality and Social Psychology Review, 10*, 20–46.

Barrett, L. F., & Bar, M. (2009). See it with feeling: Affective predictions during object perception. *Philosophical Transactions of the Royal Society B: Biological Sciences, 364*, 1325–1334.

Bartels, A., & Zeki, S. (2000). The neural basis of romantic love. *NeuroReport, 11*(17), 3829–3834.

Bartels, A., & Zeki, S. (2004). The neural correlates of maternal and romantic love. *NeuroImage, 21*(3), 1155–1166.

Baumeister, R. F., Vohs, K. D., & Tice, D. M. (2007). The strength model of self-control. *Current Directions in Psychological Science, 16*(6), 351–355.

Bechara, A. (2011). The somatic marker hypothesis and its neural basis: Using past experiences to forecast the future in decision making. In M. Bar (Ed.), *Predictions in the brain: Using our past to generate a future* (pp. 122–133). New York: Oxford University Press.

Beckes, L., & Coan, J. A. (2011). Social baseline theory: The role of social proximity in emotion and economy of action. *Social and Personality Psychology Compass, 5*, 976–988.

Beckes, L., Coan, J. A., & Hasselmo, K. (2012). Familiarity promotes the blurring of self and other in the neural representation of threat. *Social Cognitive and Affective Neuroscience.* [E-publication ahead of print]

Berscheid, E. (2003). The human's greatest strength: Other humans. In U. M. Staudinger (Ed.), *A psychology of human strengths: Fundamental questions and future directions for a positive psychology* (pp. 37–47). Washington, DC: American Psychological Association.

Bertram, B. C. R. (1978). Living in groups: Predators and prey. In J. R. Krebs & N. B. Davies (Eds.), *Behavioural ecology: An evolutionary approach* (pp. 64–96). Oxford, UK: Blackwell.

Bishop, S., Duncan, J., Brett, M., & Lawrence, A. D. (2004). Prefrontal cortical function and anxiety: Controlling attention to threat-related stimuli. *Nature Neuroscience, 7*, 184–188.

Bowlby, J. (1982). *Attachment and loss: Vol. 1. Attachment* (2nd ed.). New York: Basic Books. (Original work published 1969)

Brewer, M. B., & Caporeal, L. R. (1990). Selfish genes vs. selfish people: Sociobiology as origin myth. *Motivation and Emotion, 14*, 237–243.

Brooks, J. C. W., Nurmikko, T. J., Bimson, W. E., Singh, K. D., & Roberts, N. (2002). fMRI of thermal pain: Effects of stimulus laterality and attention. *NeuroImage, 15*, 293–301.

Cancedda, L., Putignano, E., Sale, A., Viegi, A., Berardi, N., & Maffei, L. (2004). Acceleration of visual system development by environmental enrichment. *Journal of Neuroscience, 24*, 4840–4848.

Cassidy, J., & Shaver, P. R. (2008). *Handbook of attachment: Theory, research, and clinical applications* (2nd ed.). New York: Guilford Press.

Chugani, H. T. (1998). A critical period of brain development: Studies of cerebral glucose utilization with PET. *Preventive Medicine, 27*, 184–188.

Clore, G. L., & Tamir, M. (2002). Affect as embodied information. *Psychological Inquiry, 13*, 37–45.

Coan, J. A. (2008). Toward a neuroscience of attachment. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 241–265). New York: Guilford Press.

Coan, J. A., Beckes, L., & Allen, J. P. (2013). Childhood maternal support and social capital moderate the regulatory impact of social relationships in adulthood. *International Journal of Psychophysiology, 88*(3), 224–231.

Coan, J. A., Schaefer, H. S., & Davidson, R. J. (2006). Lending a hand: Social regulation of the neural response to threat. *Psychological Science, 17*, 1032–1039.

Cohen, S., & Hoberman, H. M. (1983). Positive events and social supports as buffers of life change stress. *Journal of Applied Social Psychology, 13*(2), 99–125.

Cohen, S., & McKay, G. (1984). Social support, stress, and the buffering hypothesis: A theoretical analysis. *Handbook of Psychology and Health, 4*, 253–267.

Conner, O. L., Siegle, G. J., McFarland, A. M., Silk, J. S., Ladouceur, C. D., Dahl, R. E., et al. (2012). Mom—It helps when you're right here!: Attenuation of neural stress markers in anxious youths whose caregivers are present during fMRI. *PloS ONE, 7*(12), e50680.

Davis, L. S. (1984). Alarm calling in Richardson's ground squirrels (*Spermophilus richardsonii*). *Zeitschrift für Tierpsychologie, 66*(2), 152–164.

Dedovic, K., Renwick, R., Mahani, N. K., Engert, V., Lupien, S. J., & Pruessner, J. C. (2005). The Montreal Imaging Stress Task: Using functional imaging to investigate the effects of perceiving and processing psychosocial stress in the human brain. *Journal of Psychiatry and Neuroscience, 30*, 319–325.

Del Giorno, J. M., Hall, E. E., O'Leary, K. C., Bixby, W. R., & Miller, P. C. (2010). Cognitive function during acute exercise: A test of the transient hypofrontality theory. *Journal of Sport and Exercise Psychology, 32*(3), 312–323.

Dietrich, A. (2009). The transient hypofrontality theory and its implications for emotion and cognition. In T. McMorris, P. D. Tomporowski, & M. Audiffren (Eds.), *Exercise and cognitive function* (pp. 69–90). Hoboken, NJ: Wiley-Blackwell.

Ehrenberg, M. F., Gearing-Small, M., Hunter, M. A., & Small, B. J. (2001). Childcare task division and shared parenting attitudes in dual-earner families with young children. *Family Relations, 50*(2), 143–153.

Ehrlich, P. R., & Ehrlich, A. H. (2008). *The dominant animal: Human evolution and the environment*. Washington, DC: Island Press.

Eisenberger, N. I., Master, S. L., Inagaki, T. K., Taylor, S. E., Shirinyan, D., Lieberman, M. D., et al. (2011). Attachment figures activate a safety signal-related neural region and reduce pain experience. *Proceedings of the National Academy of Sciences, 108*, 11721–11726.

Eisenberger, N. I., Taylor, S. E., Gable, S. L., Hilmert, C. J., & Lieberman, M. D. (2007). Neural pathways link social support to attenuated neuroendocrine stress responses. *NeuroImage, 35*(4), 1601–1612.

Elgar, M. A. (1989). Predator vigilance and group-size in mammals and birds—A critical review of the empirical evidence. *Biological Reviews of the Cambridge Philosophical Society, 64*(1), 13–33.

Fitzsimons, G. M., & Finkel, E. J. (2011). Outsourcing self-regulation. *Psychological Science, 22*(3), 369–375.

Fraley, R. C., & Shaver, P. R. (2000). Adult romantic attachment: Theoretical developments, emerging controversies, and unanswered questions. *Review of General Psychology, 4*, 132–154.

Franceschini, M. A., Thaker, S., Themelis, G., Krishnamoorthy, K. K., Bortfeld, H., Diamond, S. G., et al. (2007). Assessment of infant brain development with frequency-domain near-infrared spectroscopy. *Pediatric Research, 61*, 546–551.

Francis, S., Rolls, E. T., Bowtell, R., McGlone, F., O'Doherty, J., Browning, A., et al. (1999). The representation of pleasant touch in the brain and its relationship with taste and olfactory areas. *NeuroReport, 10*, 453–459.

Friston, K. (2010). The free-energy principle: A unified brain theory? *Nature Reviews Neuroscience, 11*(2), 127–138.

Fuster, J. M. (2002). Frontal lobe and cognitive development. *Journal of Neurocytology, 31*(3), 373–385.

Gable, S. L., Gonzaga, G. C., & Strachman, A. (2006). Will you be there for me when things go right?: Supportive responses to positive event disclosures. *Journal of Personality and Social Psychology, 91*(5), 904–917.

Gable, S. L., Reis, H. T., Impett, E. A., & Asher, E. R. (2004). What do you do when things go right?: The intrapersonal and interpersonal benefits of sharing positive events. *Journal of Personality and Social Psychology, 87*(2), 228–245.

Gazzola, V., Spezio, M. L., Etzel, J. A., Castelli, F., Adolphs, R., & Keysers, C. (2012). Primary

somatosensory cortex discriminates affective significance in social touch. *Proceedings of the National Academy of Sciences, 109*, E1657–E1666.

Gillath, O., Bunge, S. A., Shaver, P. R., Wendelken, C., & Mikulincer, M. (2005). Attachment-style differences in the ability to suppress negative thoughts: Exploring the neural correlates. *NeuroImage, 28*, 835–847.

Goldstein, R. Z., & Volkow, N. D. (2011). Dysfunction of the prefrontal cortex in addiction: Neuroimaging findings and clinical implications. *Nature Reviews Neuroscience, 12*(11), 652–669.

Halford, G. S., Wilson, W. H., & Phillips, S. (1997). Abstraction: Nature, costs, and benefits. *International Journal of Educational Research, 27*(1), 21–35.

Halford, G. S., Wilson, W. H., & Phillips, S. (1998). Processing capacity defined by relational complexity: Implications for comparative, developmental, and cognitive psychology. *Behavioral and Brain Sciences, 21*(6), 803–831.

Harlow, H. F. (1958). The nature of love. *American Psychologist, 13*, 673–685.

Hebb, D. O. (1949). *The organization of behavior.* New York: Wiley.

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological Psychiatry, 54*(12), 1389–1398.

Higashi, M., & Yamamura, N. (1993). What determines animal group size?: Insider–outsider conflict and its resolution. *American Naturalist, 142*(3), 553–563.

Hill, K. R., Walker, R. S., Bozicevic, M., Eder, J., Headland, T., Hewlett, B., et al. (2011). Coresidence patterns in hunter–gatherer societies show unique human social structure. *Science, 331*(6022), 1286–1289.

Hofer, M. A. (2006). Psychobiological roots of early attachment. *Current Directions in Psychological Science, 15*, 84–88.

Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine, 7*, e1000316.

Hughes, A. E., Crowell, S. E., Uyeji, L., & Coan, J. A. (2012). A developmental neuroscience of borderline pathology: Emotion dysregulation and social baseline theory. *Journal of Abnormal Child Psychology, 40*, 21–33.

Kable, J. W., & Glimcher, P. W. (2007). The neural correlates of subjective value during intertemporal choice. *Nature Neuroscience, 10*, 1625–1633.

Karau, S. J., & Williams, K. D. (1993). Social loafing: A meta-analytic review and theoretical integration. *Journal of Personality and Social Psychology, 65*(4), 681–706.

Karremans, J. C., Heslenfeld, D. J., van Dillen, L. F., & Van Lange, P. A. (2011). Secure attachment partners attenuate neural responses to social exclusion: An fMRI investigation. *International Journal of Psychophysiology, 81*(1), 44–50.

Kim, S. H., & Hamann, S. (2007). Neural correlates of positive and negative emotion regulation. *Journal of Cognitive Neuroscience, 19*(5), 776–798.

Knill, D. C., & Pouget, A. (2004). The Bayesian brain: The role of uncertainty in neural coding and computation. *Trends in Neurosciences, 27*(12), 712–719.

Kraus, M. W., Huang, C., & Keltner, D. (2010). Tactile communication, cooperation, and performance: An ethological study of the NBA. *Emotion, 10*, 745–749.

Krause, J., & Ruxton, G. (2002). *Living in groups*. New York: Oxford University Press.

Krebs, J. R., & Davies, N. B. (1993). *An introduction to behavioural ecology* (3rd ed.). Malden, MA: Blackwell.

Kurzban, R. (2010). Does the brain consume additional glucose during self-control tasks? *Evolutionary Psychology, 8*(2), 244–259.

Langer, S. K. (1974). *Mind: An essay on human feeling* (Vol. 2). Baltimore: Johns Hopkins University Press.

Langston, C. A. (1994). Capitalizing on and coping with daily-life events: Expressive responses to positive events. *Journal of Personality and Social Psychology, 67*(6), 1112–1125.

Larose, S., Bernier, A., Soucy, N., & Duchesne, S. (1999). Attachment style dimensions, network orientation and the process of seeking help from college teachers. *Journal of Social and Personal Relationships, 16*, 225–247.

Lieberman, M. D. (2007). Social cognitive neuroscience: A review of core processes. *Annual Review of Psychology, 58*, 259–289.

Lindquist, K. A., & Barrett, L. F. (2008). Constructing emotion. *Psychological Science, 19*, 898–903.

Lipina, S. J., & Colombo, J. A. (2009). *Poverty and brain development during childhood: An*

approach from cognitive psychology and neuroscience. Washington, DC: American Psychological Association.

MacArthur, R. H., & Pianka, E. R. (1966). On optimal use of a patchy environment. *American Naturalist, 100*, 603–609.

Main, M. (1996). Introduction to the special section on attachment and psychopathology: 2. Overview of the field of attachment. *Journal of Consulting and Clinical Psychology, 64*, 237–243.

McComb, K., Moss, C., Durant, S. M., Baker, L., & Sayialel, S. (2001). Matriarchs as repositories of social knowledge in African elephants. *Science, 292*, 491–494.

McGowan, P. O., Sasaki, A., D'Alessio, A. C., Dymov, S., Labonté, B., Szyf, M., et al. (2009). Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience, 12*, 342–348.

McNally, L., Brown, S. P., & Jackson, A. L. (2012). Cooperation and the evolution of intelligence. *Proceedings of the Royal Academy B: Biological Sciences, 279*(1740), 3027–3034.

Michelena, P., Pillot, M. H., Henrion, C., Toulet, S., Boissy, A., & Bon, R. (2012). Group size elicits specific physiological response in herbivores. *Biology Letters, 8*(4), 537–539.

Mikulincer, M., & Florian, V. (1998). The relationship between adult attachment styles and emotional and cognitive reactions to stressful events. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 143–165). New York: Guilford Press.

Mikulincer, M., & Orbach, I. (1995). Attachment styles and repressive defensiveness: The accessibility and architecture of affective memories. *Journal of Personality and Social Psychology, 68*, 917–925.

Mikulincer, M., & Shaver, P. R. (2005). Attachment theory and emotions in close relationships: Exploring the attachment-related dynamics of emotional reactions to relational events. *Personal Relationships, 12*, 149–168.

Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York: Guilford Press.

Mikulincer, M., Shaver, P. R., & Pereg, D. (2003). Attachment theory and affect regulation: The dynamics, development, and cognitive consequences of attachment-related strategies. *Motivation and Emotion, 27*, 77–102.

Mobbs, D., Petrovic, P., Marchant, J. L., Hassabis, D., Weiskopf, N., Seymour, B., et al. (2007). When fear is near: Threat imminence elicits prefrontal–periaqueductal gray shifts in humans. *Science, 317*(5841), 1079–1083.

Moll, H., & Tomasello, M. (2007). Cooperation and human cognition: The Vygotskian intelligence hypothesis. *Philosophical Transactions of the Royal Society B: Biological Sciences, 362*(1480), 639–648.

Monson, C. M., Fredman, S. J., Macdonald, A., Pukay-Martin, N. D., Resick, P. A., & Schnurr, P. P. (2012). Effect of cognitive-behavioral couple therapy for PTSD: A randomized controlled trial. *Journal of the American Medical Association, 308*, 700–709.

Moore, L. J., Vine, S. J., Wilson, M. R., & Freeman, P. (2012). The effect of challenge and threat states on performance: An examination of potential mechanisms. *Psychophysiology, 49*(10), 1417–1425.

Morrison, I., Löken, L. S., & Olausson, H. (2010). The skin as a social organ. *Experimental Brain Research, 204*, 305–314.

Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin, 126*(2), 247–259.

Naaman, S., Radwan, K., & Johnson, S. (2009). Coping with early breast cancer: Couple adjustment processes and couple-based intervention. *Psychiatry: Interpersonal and Biological Processes, 72*(4), 321–345.

Paxton, J. L., Barch, D. M., Racine, C. A., & Braver, T. S. (2008). Cognitive control, goal maintenance, and prefrontal function in healthy aging. *Cerebral Cortex, 18*(5), 1010–1028.

Posner, M. I., & Rothbart, M. K. (2007). Research on attention networks as a model for the integration of psychological science. *Annual Review of Psychology, 58*, 1–23.

Prinstein, M. J., & La Greca, A. M. (2004). Childhood peer rejection and aggression as predictors of adolescent girls' externalizing and health risk behaviors: A 6-year longitudinal Study. *Journal of Consulting and Clinical Psychology, 72*, 103–112.

Pulliam, H. R., Pyke, G. H., & Caraco, T. (1982). The scanning behavior of juncos: A game–theoretical approach. *Journal of Theoretical Biology, 95*(1), 89–103.

Reichardt, L. F. (2006). Neurotrophin-regulated signalling pathways. *Philosophical Transactions of the Royal Society B: Biological Sciences, 361*, 1545–1564.

Reis, H. T., Smith, S. M., Carmichael, C. L.,

Caprariello, P. A., Tsai, F. F., Rodrigues, A., et al. (2010). Are you happy for me?: How sharing positive events with others provides personal and interpersonal benefits. *Journal of Personality and Social Psychology, 99*(2), 311–329.

Rholes, W. S., Simpson, J. A., & Grich Stevens, J. (1998). Attachment orientations, social support, and conflict resolution in close relationships. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 166–188). New York: Guilford Press.

Richerson, P. J., Bettinger, R. L., & Boyd, R. (2005). Evolution on a restless planet: Were environmental variability and environmental change major drivers of human evolution? In F. M. Wuketits & C. Antweiler (Eds.), *Handbook of evolution: The evolution of living systems* (Vol. 2, pp. 223–242). New York: Wiley.

Roberts, G. (1996). Why individual vigilance declines as group size increases. *Animal Behaviour, 51*, 1077–1086.

Rogers, S. J., & DeBoer, D. D. (2001). Changes in wives' income: Effects on marital happiness, psychological well-being, and the risk of divorce. *Journal of Marriage and Family, 63*(2), 458–472.

Salinas, E. (2011). Prior and prejudice. *Nature Neuroscience, 14*, 943–945.

Schilbach, L., Timmermans, B., Reddy, V., Costall, A., Bente, G., Schlicht, T., et al. (in press). Toward a second-person neuroscience. *Behavioral and Brain Sciences*.

Schmidhempel, P., Kacelnik, A., & Houston, A. I. (1985). Honeybees maximize efficiency by not filling their crop. *Behavioral Ecology and Sociobiology, 17*(1), 61–66.

Schnall, S., Harber, K. D., Stefanucci, J. K., & Proffitt, D. R. (2008). Social support and the perception of geographical slant. *Journal of Experimental Social Psychology, 44*(5), 1246–1255.

Schwarz, N., & Clore, G. L. (1983). Mood, misattribution, and judgments of well-being: Informative and directive functions of affective states. *Journal of Personality and Social Psychology, 45*, 513–523.

Shallcross, S. L., Howland, M., Bemis, J., Simpson, J. A., & Frazier, P. (2011). Not "capitalizing" on social capitalization interactions: The role of attachment insecurity. *Journal of Family Psychology, 25*(1), 77–85.

Sheppes, G., Catran, E., & Meiran, N. (2009). Reappraisal (but not distraction) is going to make you sweat: Physiological evidence for self-control effort. *International Journal of Psychophysiology, 71*(2), 91–96.

Sherwood, C. C., Stimpson, C. D., Raghanti, M. A., Wildman, D. E., Uddin, M., Grossman, L. I., et al. (2006). Evolution of increased glia–neuron ratios in the human frontal cortex. *Proceedings of the National Academy of Sciences, 103*(37), 13606–13611.

Siegfried, W. R., & Underhill, L. G. (1975). Flocking as an anti-predator strategy in doves. *Animal Behaviour, 23*, 504–508.

Smith, E. A. (2003). Human cooperation: Perspectives from behavioral ecology. In P. Hammerstein (Ed.), *Genetic and cultural evolution of cooperation* (pp. 401–427). Cambridge, MA: MIT Press.

Sokoloff, L., Mangold, R., Wechsler, R. L., Kennedy, C., & Kety, S. S. (1955). The effect of mental arithmetic on cerebral circulation and metabolism. *Journal of Clinical Investigation, 34*(7, Pt. 1), 1101–1108.

Stark, R., Schienle, A., Walter, B., Kirsch, P., Sammer, G., Ott, U., et al. (2003). Hemodynamic responses to fear and disgust-inducing pictures: An fMRI study. *International Journal of Psychophysiology, 50*, 225–234.

Stefanucci, J. K., Gagnon, K. T., & Lessard, D. A. (2011). Follow your heart: Emotion adaptively influences perception. *Social and Personality Psychology Compass, 5*, 296–308.

Stefanucci, J. K., Proffitt, D. R., Banton, T., & Epstein, W. (2005). Distances appear different on hills. *Attention, Perception, and Psychophysics, 67*, 1052–1060.

Stefanucci, J. K., Proffitt, D. R., Clore, G. L., & Parekh, N. (2008). Skating down a steeper slope: Fear influences the perception of geographical slant. *Perception, 37*, 321–323.

Tomaka, J., Blascovich, J., Kelsey, R. M., & Leitten, C. L. (1993). Subjective, physiological, and behavioral effects of threat and challenge appraisal. *Journal of Personality and Social Psychology, 65*, 248–260.

Tomasello, M., Carpenter, M., Call, J., Behne, T., & Moll, H. (2005). Understanding and sharing intentions: The origins of cultural cognition. *Behavioral and Brain Sciences, 28*, 675–690.

Townsend, A. L., & Franks, M. M. (1995). Binding ties: Closeness and conflict in adult children's caregiving relationships. *Psychology and Aging, 10*(3), 343–351.

Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review

with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin, 119*, 488–531.

van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., & Ebstein, R. P. (2011). Methylation matters in child development: Toward developmental behavioral epigenetics. *Child Development Perspectives, 5*, 305–310.

Vohs, K. D., & Heatherton, T. F. (2000). Self-regulatory failure: A resource-depletion approach. *Psychological Science, 11*(3), 249–254.

Weaver, I. C. G., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., et al. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience, 7*, 847–854.

Woolley, A. W., Chabris, C. F., Pentland, A., Hashmi, N., & Malone, T. W. (2010). Evidence for a collective intelligence factor in the performance of human groups. *Science, 330*, 686–688.

Younger, J., Aron, A., Parke, S., Chatterjee, N., & Mackey, S. (2010). Viewing pictures of a romantic partner reduces experimental pain: Involvement of neural reward systems. *PloS ONE, 5*, e13309.

CHAPTER 15

Adult Attachment and Emotion Regulation

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In the past 30 years, attachment theory (Bowlby, 1973, 1980, 1969/1982) has become one of the most influential conceptual frameworks for understanding emotion regulation. Although Bowlby did not devote much attention to abstract theorizing about emotion itself (he included only a single brief chapter about it in Volume 1 of his *Attachment and Loss* trilogy), his writings were motivated by clinical and ethological observations of humans and other primates who were experiencing, expressing, and regulating emotions such as affection, anxiety, anger, grief, and despair. He was especially interested in the anxiety-buffering function of close relationships and the capacity for dysfunctional relationships to generate negative emotions, and, in the extreme, to precipitate debilitating forms of psychopathology. Bowlby (1973, 1980) described and conceptualized the relatively stable individual differences in emotion regulation that emerge from prolonged reliance on particular *attachment figures*, people who provide either adequate or inadequate protection, safety, support, and guidance concerning emotions and emotion regulation.

With the accumulation of empirical knowledge about what Bowlby called the "attachment behavioral system" and about individual differences in attachment orientations in childhood and adulthood, Bowlby's ideas (as elaborated and tested initially by Ainsworth, Blehar, Waters, & Wall, 1978) have been expanded, tested, and organized by social–personality psychologists into a theoretical model of the attachment system in adulthood (e.g., Mikulincer & Shaver, 2007a; Shaver & Mikulincer, 2002). The model focuses on the emotion-regulatory function of the attachment system and explains many of the emotional correlates and consequences of normative and individual-differences aspects of attachment system functioning. In this chapter, we elaborate on these normative and individual-differences aspects, and review studies that have examined the links between attachment system activation and emotion regulation, and the ways in which individual differences in attachment are reflected in patterns of coping with attachment-irrelevant stressful events and attachment-related stressors (e.g., a partner's hurtful behavior, the breakup of a relationship, the death of a relationship partner).

Attachment Theory: Basic Concepts

Bowlby (1969/1982) claimed that human beings are born with an innate psychobiological system (the *attachment behavioral system*) that motivates them to seek proxim-

ity to significant others (*attachment figures*) in times of need. This system accomplishes basic regulatory functions (protection from threats and alleviation of distress) in human beings of all ages, but it is most directly observable during infancy and early childhood (Bowlby, 1988). According to Bowlby (1969/1982), attachment figures can provide a physical and emotional safe haven that facilitates the down-regulation of negative affective states, and a secure base that encourages positive affective states conducive to exploration and learning, thus supporting the development of knowledge and skills.

Bowlby (1973) also described important individual differences in attachment system functioning shaped by the responsiveness and supportiveness of attachment figures. Interactions with attachment figures who are responsive in times of need facilitate the optimal functioning of the attachment system and promote a *sense of attachment security*. This deep and pervasive sense of security is based on implicit beliefs that the world is generally safe, that attachment figures are helpful when called upon, and that it is possible to explore the environment curiously and to engage enjoyably with other people without undue fear. This sense of security is rooted in positive mental representations of self and others, which Bowlby called *internal working models*.

Unfortunately, when attachment figures are not reliably available and supportive, and when they fail to provide adequate relief from distress, children who are dependent on them may form negative working models of self and others and develop defensive *secondary attachment strategies* that involve hyperactivation or deactivation of the attachment system (e.g., Cassidy & Kobak, 1988). *Hyperactivation* (which Bowlby, 1982, called "protest") is characterized by intense efforts to attain proximity to attachment figures and insistent attempts to induce a relationship partner, viewed as insufficiently available or responsive, to provide more satisfying and reassuring care and support. Hyperactivating strategies include clinging, controlling, and coercive behaviors; cognitive and behavioral efforts to establish physical contact and a sense of "oneness"; and up-regulation of negative affective states (Shaver & Mikulincer, 2002). In contrast, *deactivation* involves the inhibition of proximity-seeking inclinations and actions, suppression or discounting of threats that might activate the attachment system, down-regulation of both negative and positive affective states, and determination to handle stressors alone (a defensive stance that Bowlby [1969/1982] called "compulsive self-reliance"). People who rely on these strategies tend to maximize autonomy and distance from relationship partners and experience discomfort with closeness and intimacy.

When testing this theory in studies of adults, most researchers have focused on the organized pattern of relational expectations, emotions, and behavior that results from a history of interactions with attachment figures—often called "attachment style" by social psychologists (e.g., Fraley & Shaver, 2000). Psychometric research (e.g., Brennan, Clark, & Shaver, 1998) has shown that attachment styles can be measured in terms of two independent dimensions: attachment anxiety and attachment-related avoidance. A person's position on the attachment anxiety dimension indicates the degree to which he or she worries that a partner will not be responsive in times of need. A person's position on the avoidance dimension indicates the extent to which he or she distrusts relationship partners' goodwill and their willingness and capacity to help without causing further distress. People who score low on both dimensions are said to be secure with respect to attachment. A person's location in the continuous two-dimensional anxiety-by-avoidance space can be measured with reliable and valid self-report scales (e.g., Brennan et al., 1998) and is associated in theoretically predictable ways with many aspects of psychological adjustment (see Mikulincer & Shaver, 2007a, for a review).

Attachment styles begin to develop in interactions with primary caregivers during early childhood, as a large body of research demonstrates (see Cassidy & Shaver, 2008, for an anthology of reviews), but Bowlby (1988) claimed that relational experiences throughout life can move a person from one region to another in the anxiety-by-avoidance conceptual space. Moreover, although attachment style is often measured as a single global orientation toward close relationships, a person's orientation to

attachment is rooted in a complex cognitive and affective associative neural network that includes both secure and insecure episodic and semantic memories and mental representations (Mikulincer & Shaver, 2007a). Indeed, a growing body of research shows that attachment style can change, subtly or dramatically, depending on current context and recent relational experiences (see Mikulincer & Shaver, 2007b, for a review). For example, we have found that priming thoughts of an available and supportive attachment figure leads people who score relatively high on scales tapping attachment insecurities momentarily to behave like more secure people (e.g., Mikulincer & Shaver, 2001).

In the next two sections, we present both theoretical ideas and empirical evidence concerning the link between attachment and emotion regulation. In the first section, we focus on normative aspects of this link and the emotion regulation functions of the attachment behavioral system. That is, we discuss and review empirical evidence concerning the normative activation of the attachment system in response to threats and the down-regulation of distress caused by optimal functioning of the system. In the second section, we focus on individual differences and review empirical evidence concerning the up-regulation of negative affect caused by anxious attachment, and the ways in which avoidant attachment leads to down-regulation of both negative and positive affective states.

Normative Aspects of the Link between Attachment and Emotion Regulation

According to attachment theory (Bowlby, 1969/1982), the attachment system evolved because it increased the likelihood that offspring would survive until they were old enough to reproduce and also taught children how to regulate emotions effectively. This system is automatically activated by external threats or internal sources of distress and, when it functions appropriately, leads to emotional security. Its optimal functioning is associated with a relational if–then script, which Waters and Waters (2006) called a secure-base script, paraphrased by us as follows: "If I encounter an obstacle and/or become distressed, then I can approach a significant other for help. He or she is likely to be available and supportive. I will experience relief and comfort as a result of proximity to this person, and I can then return to other activities." Once activated, this script can, by itself, mitigate distress, promote optimism and hope, and help a person cope effectively with life's inevitable difficulties (Mikulincer, Shaver, Sapir-Lavid, & Avihou-Kanza, 2009).

Adult attachment researchers have designed experimental procedures to examine these normative regulatory properties of the attachment system. For example, in a series of experiments, Mikulincer, Gillath, and Shaver (2002) showed that mental representations of attachment figures (e.g., names of security-enhancing attachment figures) are automatically activated in a person's mind when he or she is exposed to threatening stimuli, even if that exposure is unconscious (i.e., primed subliminally). Specifically, when a threat-related word (e.g., *death*) was presented subliminally on a computer screen, participants were faster to detect the name of one of their attachment figures when it appeared on the screen and slower to name the color in which such names were printed on the screen—an indication that the names had been automatically and unconsciously activated in memory (Mikulincer et al., 2002). In other words, threats, even when arising unconsciously, can automatically activate mental representations of security providers. Similar findings have been obtained in experiments examining automatic activation of representations of symbolic sources of security, such as a person's pet or God, following a threat (Granqvist, Mikulincer, Gurwitz, & Shaver, 2012; Zilcha-Mano, Mikulincer, & Shaver, 2011).

In another set of studies of what we call *security priming*, Mikulincer, Hirschberger, Nachmias, and Gillath (2001) showed that activation of representations of security-enhancing attachment figures can automatically infuse a previously neutral stimulus with positive affect. For example, subliminal presentation of the names of people who were nominated by study participants as attachment figures, compared with others who were not nominated as attachment figures, led to greater liking of previously unfamil-

iar stimuli. Moreover, subliminal exposure to names of attachment figures eliminated the detrimental effects that threats otherwise had on liking for previously neutral stimuli. These effects of security priming on positive affect have been replicated in subsequent studies (see Mikulincer & Shaver, 2007b, for a review). In addition, Eisenberger and colleagues (2011; Master et al., 2009) found that viewing a photograph of one's romantic partner (vs. a stranger or an object) reduced one's subjective experience of pain while receiving thermal stimulation at levels slightly higher than the pain threshold. Moreover, Selcuk, Zayas, Günaydin, Hazan, and Kross (2012) found that both explicit and implicit priming of attachment figure representations speeded up emotional recovery and reduced negative thoughts after recalling an upsetting experience.

Considering all such findings, we conclude that people automatically search for internal representations of security-enhancing attachment figures during times of stress, and mental activation of these representations produces positive emotions (e.g., relief, satisfaction, gratitude, love) that facilitate effective coping and restore emotional equanimity. That is, actual or symbolic interactions with available and supportive attachment figures, and the resulting sense of safety, can be viewed as psychological resources for dealing with problems and adversities, and sustaining well-being and mental health (Mikulincer & Shaver, 2007a).

Attachment-Related Individual Differences in Emotion Regulation

Theoretical Account

According to Bowlby (1973), disruptions in the sense of attachment security are viewed as risk factors for emotional problems and psychopathology. Although secondary attachment strategies (anxious hyperactivation and avoidant deactivation) are initially adaptive, in the sense that they adjust a child's behavior to the requirements of an inconsistently available or consistently distant or unavailable attachment figure, they are maladaptive when used in later relationships in which support seeking and relational interdependence could be rewarding and help a person maintain a sense of well-being even in stressful conditions or difficult periods of life (Shaver & Mikulincer, 2002). Moreover, these attachment strategies encourage repeated activation and suppression of negative emotions and continued reliance on negative or distorted mental representations of self and others, which can erode and destroy mental health (Mikulincer & Shaver, 2007a).

The early attachment experiences of insecure people (whether anxious, avoidant, or both) often involve unstable and inadequate distress regulation (Bowlby, 1973), which interferes with the development of inner resources needed for coping successfully with stressors. This impairment is particularly likely during prolonged, highly demanding stressful experiences that require active support seeking and actual confrontation with a problem (Berant, Mikulincer, & Shaver, 2008). In such cases, anxious hyperactivating strategies can become extreme, damaging not only a person's own mental health but that of key relationship partners. Moreover, avoidant defenses can collapse, resulting in a marked decline in psychological well-being.

When regulating their emotions, avoidant people attempt to block or inhibit any emotional state that is incongruent with the goal of keeping their attachment system deactivated (Mikulincer & Shaver, 2007a). These inhibitory efforts are directed mainly at fear, anxiety, anger, sadness, shame, guilt, and distress, because these emotions are associated with threats and feelings of vulnerability. In addition, anger often implies emotional involvement or investment in a relationship, and such involvement is incongruent with avoidant people's preference for independence and self-reliance (Cassidy, 1994). Avoidant individuals also attempt to block or inhibit emotional reactions to potential or actual threats to attachment figure availability (rejection, betrayal, separation, loss), because such threats are direct triggers of attachment system activation. Like secure people, avoidant ones attempt to down-regulate threat-related emotions. But whereas secure people's regulatory attempts usually promote communication, compromise, and relationship maintenance, avoidant people's efforts are aimed mainly at keeping the attachment system deactivated,

regardless of the deleterious effects this can have on a relationship.

Deactivating strategies cause people to avoid noticing their own emotional reactions. Avoidant individuals often deny or suppress emotion-related thoughts and memories, divert attention from emotion-related material, suppress emotion-related action tendencies, or inhibit or mask verbal and nonverbal expressions of emotion (Mikulincer & Shaver, 2007a). By averting the conscious experience and expression of unpleasant emotions, avoidant individuals make it less likely that emotional experiences will be integrated into their cognitive structures and that such feelings and mental structures will be used effectively in information processing and social behavior. Bowlby (1980) described this strategy as "defensive exclusion" and the creation of "segregated mental systems."

Unlike secure and avoidant people, who tend to view negative emotions as goal-incongruent states that should either be managed effectively or suppressed, anxiously attached individuals tend to perceive these emotions as congruent with attachment goals, and they may seek to sustain and even exaggerate them. Attachment-anxious people are guided by an unfulfilled wish to cause attachment figures to pay more attention and provide more reliable protection and support (Cassidy & Kobak, 1988; Mikulincer & Shaver, 2007a). Therefore, they tend to exaggerate the presence and seriousness of threats and to overemphasize their sense of helplessness and vulnerability, because signs of weakness and neediness can sometimes elicit attachment figures' attention and care (Cassidy & Berlin, 1994).

How is anxious hyperactivation sustained? One method is to exaggerate the appraisal process, perceptually heightening the threatening aspects of even fairly benign events, to hold pessimistic beliefs about one's ability to manage distress, and to attribute threat-related events to uncontrollable causes or global personal inadequacies (Mikulincer & Shaver, 2007a). Another method is to attend to internal indicators of distress (Cassidy & Kobak, 1988). This includes hypervigilant attention to the physiological aspects of emotional states, heightened recall of threat-related experiences, and rumination on real and potential threats (Mikulincer & Shaver, 2007a). Another hyperactivating strategy is to intensify negative emotions by favoring an approach, counterphobic orientation toward threatening situations or making self-defeating decisions and taking ineffective actions that are likely to end in failure. All of these strategies create a self-amplifying cycle of distress even after a threat objectively recedes.

Empirical Evidence

There is now a large body of evidence supporting the hypothesized links between attachment-related avoidance and emotional inhibition, and between attachment anxiety and distress intensification. In particular, theory-congruent findings have been found in studies of responses to both attachment-irrelevant stressful events and attachment-related stressors (e.g., a partner's hurtful behavior, a relationship breakup, or the death of a relationship partner).

Attachment Orientations and Responses to Stressful Events

Several studies have examined attachment style differences in the ways people appraise, cope with, and emotionally and physiologically react to attachment-irrelevant stressful events (i.e., events that have no direct implications for one's close relationships or attachment figures). These attachment style differences have been examined in response to a wide variety of stressful experiences, such as combat training (e.g., Mikulincer & Florian, 1995), transition to college (e.g., Lopez & Gormley, 2002), abortion (e.g., Cozzarelli, Sumer, & Major, 1998), infertility (e.g., Amir, Horesh, & Lin-Stein, 1999), pregnancy (e.g., Mikulincer & Florian, 1999), workload (e.g., Raskin, Kummel, & Bannister, 1998), financial problems (e.g., Bartley, Head, & Stansfeld, 2007), and job loss (e.g., Hobdy et al., 2007). They have also been examined in studies that did not focus on a specific stressful event but asked participants to report on any major stressors recently experienced (e.g., Holmberg, Lomore, Takacs, & Price, 2011). In addition, the link between attachment orientations and stress-related reactions has been examined in response to several laboratory-induced stressors, such as aversive noise (e.g.,

Quirin, Pruessner, & Kuhl, 2008), the Trier Social Stress Test (e.g., Smeets, 2010), and difficult cognitive tasks (e.g., Kidd, Hamer, & Steptoe, 2011).

With regard to the cognitive appraisal of stressful events, attachment anxiety has been associated with distress-intensifying appraisals—in which stressful events are appraised as threats rather than challenges and one's coping resources are viewed as deficient (e.g., Cozzarelli et al., 1998; Mikulincer & Florian, 1998). For avoidant attachment, however, the findings are more complex. Whereas avoidant attachment is associated with appraising stressful events in more threatening terms, it is not associated with appraisals of coping resources (e.g., Williams & Riskind, 2004). This dissociation might reflect avoidant individuals' reluctance to recognize that they are vulnerable and weak, and therefore need to rely on others for assistance and support (Mikulincer & Shaver, 2007a).

There is also theoretically consistent evidence concerning attachment-related differences in self-reports of ways of coping with stressful events. Whereas attachment anxiety is associated with emotion-focused coping, such as wishful thinking, self-blame, and distress-related rumination, avoidant attachment is associated with reliance on distancing strategies, such as stress denial, diversion of attention, and behavioral or cognitive disengagement (e.g., Holmberg et al., 2011; Zhang & Labouvie-Vief, 2004).

Several adult attachment studies have involved asking participants to report on their psychological distress or well-being during and following stressful events. Overall, findings indicate that attachment insecurities—anxiety, avoidance, or both—are associated with self-reports of heightened distress and deteriorated well-being (see Mikulincer & Shaver, 2007a, for a review). Moreover, some of the studies compared the emotional reactions of secure and insecure people undergoing stressful experiences with those of controls, revealing that stressful events arouse distress mainly among insecurely attached people. For secure people, there is often no notable difference in emotion between neutral and stressful situations (e.g., Amir et al., 1999). That is, secure people seem to be relatively calm under stressful conditions.

Recent studies have found that attachment-related differences in coping are reflected in physiological responses to stressful events. For example, two experiments (Diamond, Hicks, & Otter-Henderson, 2006; Maunder, Lancee, Nolan, Hunter, & Tannenbaum, 2006) exposed participants to various laboratory stressors (e.g., recalling a stressful situation, performing demanding math tasks) and found that self-reports of avoidant attachment were associated with heightened physiological reactivity: decreased heart rate variability, increased skin conductance, and heightened blood pressure. With regard to attachment anxiety, Maunder et al. found that attachment-anxious people reported higher levels of distress but did not exhibit heightened physiological reactivity, perhaps suggesting, as mentioned earlier, that anxious people exaggerate their own distress.

In addition, four studies have assessed attachment style differences in the activity of the hypothalamic–pituitary–adrenal (HPA) axis, which can be measured by salivary cortisol levels during and following a laboratory-induced stressor. One study found that avoidant attachment was associated with increased levels of salivary cortisol (Kidd et al., 2011); another study (Quirin et al., 2008) found a positive association between attachment anxiety and heightened cortisol reactivity; and two studies found no significant association between attachment orientations and cortisol levels (Ditzen et al., 2008; Smeets, 2010). These inconsistencies might have been due, in part, to variations in the laboratory-induced stressors (e.g., aversive noise, the Trier Social Stress Test) and participants' age (young adults, midlife adults). More systematic research is needed to determine the presence or absence of links between attachment insecurities and HPA dysregulation following stressful events.

There is also preliminary evidence that an association between attachment insecurities and psychological distress following stressful events can be observed in brain responses. Using event-related functional magnetic resonance imaging (fMRI), Lemche et al. (2006) found that self-reports of attachment anxiety or avoidance were associated with heightened activation in bilateral amygdalae to a stressful stimulus. That is, less secure people tended to react to

stress with increased amygdala activity—a neural indication of distress-related arousal.

Considering longer-term neural effects of attachment insecurities, Quirin, Gillath, Pruessner, and Eggert (2010) found that self-reports of attachment anxiety and avoidance were associated with reduced hippocampal cell density, which was associated with poorer emotion regulation. In particular, attachment-related avoidance was associated with bilateral hippocampal cell reduction, and attachment anxiety was significantly related to reduced cell concentration in the left hippocampus. These findings are compatible with a neurotoxic model of stress-induced cell reduction in the hippocampus, contributing to poorer emotion regulation abilities in individuals with insecure attachment orientations.

Attachment Orientations and Responses to a Partner's Hurtful Behaviors

Explicit or implicit signs of a partner's disapproval, criticism, rejection, or betrayal can evoke hurt feelings, arouse hostility toward a partner, and even destroy a relationship. However, the ways people react to these relational stressors seem to depend on their attachment orientation. There is accumulating evidence that insecure people tend to react more intensely than secure people—cognitively, emotionally, and behaviorally—to actual or imagined partner disapproval, criticism, rejection, or betrayal (e.g., Besser & Priel, 2009, 2010; Carnelley, Israel, & Brennan, 2007). For example, Dewitte, De Houwer, Goubert, and Buysse (2010) found that whereas attachment anxiety was related to greater physiological arousal and stronger negative affect following relational stress, attachment-related avoidance predicted withdrawal responses.

Using Rusbult, Verette, Whitney, Slovik, and Lipkus's (1991) typology of accommodation responses to a partner's negative behavior, several studies (e.g., Gaines et al., 1997; Scharfe & Bartholomew, 1995) found that, compared with insecure people, secure ones were more likely to rely on "voice" (an active, relational approach to solving a problem) and "loyalty" (understanding the temporary nature of a partner's behavior and working with him or her to improve)—the two more accommodative responses to a partner's transgressions. Moreover, whereas secure individuals tended to respond to rejection threats by adopting approach-oriented goals that improved relationship quality, less secure people tended to respond to such threats by decreasing approach motivation and emphasizing avoidance goals (Park, 2010). In a test of responses to a hurtful interaction, Perunovic and Holmes (2008) found that more secure individuals reported inhibition of negative impulses and the adoption of relationship-enhancing behavior even under time pressure, implying that for them accommodation was automatic.

Dewitte and De Houwer (2008; Dewitte, Koster, De Houwer, & Buysse, 2007) used a classic dot-probe task and found that both attachment anxiety and avoidance were associated with attentional avoidance of signs of a partner's hurtful behavior (attachment-threat words and angry faces). In addition, using the same dot-probe task, Dewitte, De Houwer, Koster, and Buysse (2007) found that attachment anxiety was associated with hypervigilance regarding attachment figures' names in both threatening and positive relational situations. This finding is conceptually similar to Mikulincer et al.'s (2002) finding of faster cognitive processing of attachment figure's names regardless of context.

Attachment insecurities also tend to inhibit forgiveness—one of the most effective accommodation responses for improving relationship quality and reestablishing relational harmony following a partner's negative behavior (e.g., Burnette, Taylor, Worthington, & Forsyth, 2007; Yárnoz-Yaben, 2009). Mikulincer, Shaver, and Slav (2006) found that less secure people were more inclined to report intense feelings of vulnerability or humiliation and a strong sense of relationship deterioration when "forgiving" a partner. In other words, attachment insecurities were associated with a more negative conception and experience of forgiveness. Burnette, Davis, Green, Worthington, and Bradfield (2009) provided evidence concerning potential mediators of such effects: Whereas the link between attachment anxiety and forgiveness was mediated by excessive rumination on relational injuries, the link between attachment-related avoidance and forgiveness was mediated by lack of empathy.

Beyond these associations between dispositional measures of attachment insecurities and deficient forms of forgiveness, there is increasing evidence that state-like senses of security or insecurity can alter the tendency to forgive a hurtful partner. For example, Finkel, Burnette, and Scissors (2007) experimentally enhanced attachment anxiety or measured weekly fluctuations in attachment anxiety for 6 months and found that heightened attachment anxiety reduced forgiveness of a partner's offenses. In addition, Hannon, Rusbult, Finkel, and Kumashiro (2010) found that, following an act of betrayal, the offending partner's provision of a sense of security to the injured partner (by genuinely expressing interest in being responsive to the victim's needs) promoted forgiveness and restoration of relational harmony. Karremans and Aarts (2007) found that subliminal security priming (with the name of a loving other), compared with neutral priming, elicited more automatic forgiveness responses to interpersonal transgressions.

Following this line of reasoning, Cassidy, Shaver, Mikulincer, and Lavy (2009) showed that experimentally heightening the sense of attachment security can reduce detrimental effects of dispositional attachment insecurities on cognitive and emotional reactions to hurtful experiences. Participants wrote a description of an incident in which a close relationship partner criticized, disapproved, rejected, or ostracized them. They then completed a computerized task in which they were repeatedly exposed subliminally (for 22 milliseconds on each occasion) to either a security-enhancing prime word (*love, secure, affection*) or a neutral prime word (*lamp, staple, building*). Immediately after the priming trials, participants were asked to think again about the hurtful event they had described and to rate how they would react to such an event if it happened in the future—for example, how rejected they would feel, and how they would feel about themselves.

In the neutral priming condition, the findings confirmed the usual deactivating and hyperactivating strategies of avoidant and anxious people. Whereas avoidance scores were associated with more defensive/hostile reactions, attachment anxiety was associated with more intense feelings of rejection, more crying, and more negative emotions. These typical correlational findings were dramatically reduced in size (most approached zero) in the security-priming condition. In other words, security priming reduced the tendency of avoidant people to rely on cool hostility or denial, and the tendency of anxious people to react histrionically to a partner's hurtful behaviors. Overall, research indicates that (1) people who are dispositionally secure can deal constructively with hurtful relational events, and (2) contextual activation of the sense of security can soften the typical maladaptive responses of dispositionally insecure people.

Responses to Separations and Relationship Breakups

Several studies have shown that attachment orientations predict the intensity and duration of distress following a romantic relationship breakup (e.g., Sbarra, 2006; Sbarra & Emery, 2005), divorce (Birnbaum, Orr, Mikulincer, & Florian, 1997), wartime separation from a marital partner (e.g., Medway, Davis, Cafferty, Chappell, & O'Hearn, 1995), or temporary separations from a romantic partner (e.g., Diamond, Hicks, & Otter-Henderson, 2008). In these studies, distress intensification was a common response of anxiously attached people, whereas attachment security was associated with faster emotional recovery and adjustment. For example, Sbarra (2006) collected daily emotion data for 4 weeks from a sample of young adults who had recently experienced a romantic relationship breakup and found that attachment anxiety was associated with slower recovery from sadness and anger. In a 21-day diary study, Diamond et al. (2008) found that attachment anxiety was associated with sleeping problems, physical symptoms, and higher levels of salivary cortisol during and following days of physical separation from a romantic partner brought about by work-related travel. Using fMRI to observe brain responses to the recall of a painful separation, Gillath, Bunge, Shaver, Wendelken, and Mikulincer (2005) found that attachment anxiety was associated with higher activation of the left anterior temporal pole and left hippocampus (areas associated with recall of sad thoughts) and lower activation of the orbitofrontal cortex (an area associated with emotional control).

For avoidant individuals, the findings depended on the nature of the separation (Mikulincer & Shaver, 2007a). Avoidance was associated with higher levels of distress following divorce and wartime separations but lower levels of distress and greater relief following temporary separations from, or permanent breakups with, dating partners. It seems that avoidant people who can handle the distress of brief separations or the dissolution of casual bonds are less successful in dealing with major separations requiring reorganization of relational routines, goals, and plans. This fits with other evidence, including that from experiments (e.g., Mikulincer, Dolev, & Shaver, 2004), indicating that avoidant defenses collapse under pressure.

There is also evidence that people with different attachment orientations differ in the ways they cope with separation. For example, following divorce (Birnbaum et al., 1997) and temporary separations from a dating partner (Feeney, 1998), attachment-anxious individuals were more likely to rely on emotion-focused coping strategies, and avoidant individuals, on distancing strategies. Similar coping strategies were noted by Davis, Shaver, and Vernon (2003) in a survey of more than 5,000 Internet respondents who described romantic relationship breakups. Avoidant respondents were less likely to seek support and more likely to cope with the breakup alone, while avoiding new romantic involvements. Anxious respondents reacted with angry protests, heightened sexual attraction to the former partner, intense preoccupation with the lost partner, a damaged sense of identity, and interference with school and work activities. In addition, as a means of coping with separation, both anxious and avoidant individuals used alcohol and drugs, which is not generally an effective coping strategy.

Studies that induced thoughts about hypothetical or actual separations also provide important information about attachment-related differences in regulating the distress resulting from these thoughts. For example, using a thought suppression task, Fraley and Shaver (1997) showed that avoidant people were highly effective in suppressing separation-related thoughts, as indicated by less frequent thoughts of loss following the suppression task and lower skin conductance during the task. However, Mikulincer et al. (2004) found that avoidant people's ability to suppress separation-related thoughts was disrupted when a cognitive load—remembering a 7-digit number—was added to the experimental task. Under a high cognitive load, avoidant individuals suddenly evinced high availability of thoughts of separation and negative self-traits. That is, the suppressed material resurfaced in experience and behavior when a high cognitive demand was imposed. We suspect that a similar resurfacing occurs when a high emotional demand is imposed, as in the studies reviewed earlier that dealt with life-threatening traumatic events (e.g., Berant et al., 2008). Recently, Ehrenthal, Friederich, and Schauenburg (2011) found that avoidant attachment was associated with impaired blood pressure recovery following recall of a painful separation, further emphasizing the fragility of avoidant defenses.

Reactions to the Death of a Close Relationship Partner

Few studies have directly examined associations between attachment orientations and grief reactions following the death of a close relationship partner. However, these studies have consistently found that attachment anxiety is associated with complicated grief reactions (e.g., Fraley & Bonanno, 2004; Jerga, Shaver, & Wilkinson, 2011). For example, Field and Sundin (2001) found that anxious attachment, assessed 10 months after the death of a spouse, predicted higher levels of psychological distress 14, 25, and 60 months after the loss, and Fraley and Bonanno (2004) found that attachment anxiety assessed 4 months after the loss of a spouse predicted higher levels of anxiety, depression, grief, trauma-related symptoms, and alcohol consumption 18 months following the loss.

With regard to avoidant attachment, researchers have generally found no significant association between this attachment pattern and depression, grief, or distress following the death of a relationship partner (e.g., Field & Sundin, 2001; Fraley & Bonanno, 2004). However, Wayment and Vierthaler (2002) found that avoidance was associated with increased somatic symptom levels following the death of a spouse.

Recently, Jerga et al. (2011) found that avoidant attachment was positively associated with prolonged grief symptoms but not with typical or normative grief symptoms. That is, people who are generally avoidant in close relationships experience long-term difficulties adjusting to the death of a relationship partner, even though they do not necessarily experience more intense typical grief symptoms.

Jerga et al. (2011) also found that relationship-specific avoidance was negatively associated with both typical and prolonged grief symptoms. However, other findings indicated that this association disappeared when measures of relationship closeness and strength were statistically controlled, suggesting that avoidant individuals may maintain relatively weak and emotionally distant relationships with the deceased, which in turn leaves them with less to grieve about. In other words, it may not be avoidant attachment per se that protects avoidant individuals from grief symptoms; it may be the weakness of the emotional bonds they have to contend with when a relationship partner dies.

Conclusions

In summarizing recent research on adult attachment patterns and its implications for emotion regulation, we have shown that attachment security is associated with appraisals and regulation efforts that are compatible with a balanced, open mind, generally low levels of stress and distress, and constructive approaches to relationship maintenance. The two major dimensions of insecure attachment are associated with characteristic strategies for dealing with painful experiences in previous attachment relationships.

Avoidance and attachment system deactivation are reactions to important relationships in which attachment figures, often beginning with one or both parents, reacted negatively to expressions of need, vulnerability, and negative emotions. To cope with that powerfully painful relationship influence, avoidant people have learned to downplay threats (i.e., trying not to appraise events as threatening), suppress or deny feelings of vulnerability and negative emotions, and view themselves as superior, autonomous, and properly unemotional. This does not keep them from reacting to relationship partners with frustration, hostility, and denigration or from boosting their own self-esteem in the face of relationship losses by focusing disproportionately on their own strengths and other people's weaknesses.

Attachment anxiety and attachment system hyperactivation are reactions to important relationships in which attachment figures, often beginning with one or both parents, reacted inconsistently to a person's expressions of need, vulnerability, and negative emotions, sometimes providing support but at other times being frustrating or inattentive (often because of their own self-focused anxiety). This caregiver regimen causes a person to believe that constant vigilance, worry, and expressions of need, vulnerability, and retaliatory anger pay off, because they sometimes do capture a relationship partner's attention. Unfortunately, they can also alienate a person from his or her initially favorable and loving relationship partners and evoke exactly what the anxious person fears: rejection or abandonment. Thus, what began as a response to a partial reinforcement schedule of attention and support, and what became a pattern of noisy negativity, seems to the anxious person to lead to outcomes that confirm his or her expectations and worst fears.

These different patterns of emotional reaction and defense have been documented in a remarkable variety of studies using experimental, interview, and observational research methods. They are now being illuminated further by neuroscientific studies. They provide strong specific support for Bowlby and Ainsworth's attachment theory and its extension into the realm of adult relationships (Mikulincer & Shaver, 2007a). However, more research is needed if we are to understand better (1) the specific hyperactivating or deactivating strategies a particular insecure person will use in particular situations, (2) the link between attachment orientations and the relative balance of implicit and explicit emotion-regulation attempts, and (3) the ways people scoring high on both attachment anxiety and avoidance regulate their emotions. In addition, future studies should attempt to integrate Bowlby's (1988) notion that effec-

tive therapeutic interventions amount to providing a secure base and help with emotion regulation—precisely what was missing in a client's previous attachment relationships—with the findings we have reviewed here concerning normative and individual-differences aspects of links between attachment orientations and emotion regulation strategies. Further development and evaluation of attachment-related therapeutic interventions will foster greatly improved lives and close relationships.

References

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: Assessed in the Strange Situation and at home.* Hillsdale, NJ: Erlbaum.

Amir, M., Horesh, N., & Lin-Stein, T. (1999). Infertility and adjustment in women: The effects of attachment style and social support. *Journal of Clinical Psychology in Medical Settings, 6*, 463–479.

Bartley, M., Head, J., & Stansfeld, S. (2007). Is attachment style a source of resilience against health inequalities at work? *Social Science and Medicine, 64*, 765–775.

Berant, E., Mikulincer, M., & Shaver, P. R. (2008). Mothers' attachment style, their mental health, and their children's emotional vulnerabilities: A seven-year study of children with congenital heart disease. *Journal of Personality, 76*, 31–66.

Besser, A., & Priel, B. (2009). Emotional responses to a romantic partner's imaginary rejection: The roles of attachment anxiety, covert narcissism, and self-evaluation. *Journal of Personality, 77*, 287–325.

Besser, A., & Priel, B. (2010). Grandiose narcissism versus vulnerable narcissism in threatening situations: Emotional reactions to achievement failure and interpersonal rejection. *Journal of Social and Clinical Psychology, 29*, 874–902.

Birnbaum, G. E., Orr, I., Mikulincer, M., & Florian, V. (1997). When marriage breaks up: Does attachment style contribute to coping and mental health? *Journal of Social and Personal Relationships, 14*, 643–654.

Bowlby, J. (1973). *Attachment and loss: Vol. 2. Separation: Anxiety and anger.* New York: Basic Books.

Bowlby, J. (1980). *Attachment and loss: Vol. 3. Sadness and depression.* New York: Basic Books.

Bowlby, J. (1982). *Attachment and loss: Vol. 1. Attachment* (2nd ed.). New York: Basic Books. (Original work published 1969)

Bowlby, J. (1988). *A secure base: Clinical applications of attachment theory.* London: Routledge.

Brennan, K. A., Clark, C. L., & Shaver, P. R. (1998). Self-report measurement of adult attachment: An integrative overview. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 46–76). New York: Guilford Press.

Burnette, J. L., Davis, D. E., Green, J. D., Worthington, E. L., & Bradfield, E., Jr. (2009). Insecure attachment and depressive symptoms: The mediating role of rumination, empathy, and forgiveness. *Personality and Individual Differences, 46*, 276–280.

Burnette, J. L., Taylor, K., Worthington, E. L., Jr., & Forsyth, D. R. (2007). Attachment working models and trait forgivingness: The mediating role of angry rumination. *Personality and Individual Differences, 42*, 1585–1596.

Carnelley, K. B., Israel, S., & Brennan, K. (2007). The role of attachment in influencing reactions to manipulated feedback from romantic partners. *European Journal of Social Psychology, 37*, 968–986.

Cassidy, J. (1994). Emotion regulation: Influence of attachment relationships [Special issue]. *Monographs of the Society for Research in Child Development, 59*, 228–249.

Cassidy, J., & Berlin, L. J. (1994). The insecure/ambivalent pattern of attachment: Theory and research. *Child Development, 65*, 971–981.

Cassidy, J., & Kobak, R. R. (1988). Avoidance and its relationship with other defensive processes. In J. Belsky & T. Nezworski (Eds.), *Clinical implications of attachment* (pp. 300–323). Hillsdale, NJ: Erlbaum.

Cassidy, J., & Shaver, P. R. (Eds.). (2008). *Handbook of attachment: Theory, research, and clinical applications* (2nd ed.). New York: Guilford Press.

Cassidy, J., Shaver, P. R., Mikulincer, M., & Lavy, S. (2009). Experimentally induced security influences responses to psychological pain. *Journal of Social and Clinical Psychology, 28*, 463–478.

Cozzarelli, C., Sumer, N., & Major, B. (1998). Mental models of attachment and coping with abortion. *Journal of Personality and Social Psychology, 74*, 453–467.

Davis, D., Shaver, P. R., & Vernon, M. L. (2003). Physical, emotional, and behavioral reactions to breaking up: The roles of gender, age, emotional involvement, and attachment style. *Personality and Social Psychology Bulletin, 29*, 871–884.

Dewitte, M., & De Houwer, J. (2008). Adult attachment and attention to positive and negative emotional face expressions. *Journal of Research in Personality, 42*, 498–505.

Dewitte, M., De Houwer, J., Goubert, L., & Buysse, A. (2010). A multi-modal approach to the study of attachment-related distress. *Biological Psychology, 85*, 149–162.

Dewitte, M., De Houwer, J., Koster, E. H. W., & Buysse, A. (2007). What's in a name?: Attachment-related attentional bias. *Emotion, 7*, 535–545.

Dewitte, M., Koster, E. H. W., De Houwer, J., & Buysse, A. (2007). Attentive processing of threat and adult attachment: A dot-probe study. *Behaviour Research and Therapy, 45*, 1307–1317.

Diamond, L. M., Hicks, A. M., & Otter-Henderson, K. A. (2006). Physiological evidence for repressive coping among avoidantly attached adults. *Journal of Social and Personal Relationships, 23*, 205–229.

Diamond, L. M., Hicks, A. M., & Otter-Henderson, K. A. (2008). Every time you go away: Changes in affect, behavior, and physiology associated with travel-related separations from romantic partners. *Journal of Personality and Social Psychology, 95*, 385–403.

Ditzen, B., Schmidt, S., Strauss, B., Nater, U. M., Ehlert, U., & Heinrichs, M. (2008). Adult attachment and social support interact to reduce psychological but not cortisol responses to stress. *Journal of Psychosomatic Research, 64*, 479–486.

Ehrenthal, J. C., Friederich, H. C., & Schauenburg, H. (2011). Separation recall: Psychophysiological response-patterns in an attachment-related short-term stressor. *Stress and Health, 27*, 251–255.

Eisenberger, N. I., Master, S. L., Inagaki, T. K., Taylor, S. E., Shirinyan, D., Lieberman, M. D., et al. (2011). Attachment figures activate a safety signal-related neural region and reduce pain experience. *Proceedings of the National Academy of Sciences USA, 108*, 11721–11726.

Feeney, J. A. (1998). Adult attachment and relationship-centered anxiety: Responses to physical and emotional distancing. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 189–219). New York: Guilford Press.

Field, N. P., & Sundin, E. C. (2001). Attachment style in adjustment to conjugal bereavement. *Journal of Social and Personal Relationships, 18*, 347–361.

Finkel, E. J., Burnette, J. L., & Scissors, L. E. (2007). Vengefully ever after: Destiny beliefs, state attachment anxiety, and forgiveness. *Journal of Personality and Social Psychology, 92*, 871–886.

Fraley, R., & Bonanno, G. A. (2004). Attachment and loss: A test of three competing models on the association between attachment-related avoidance and adaptation to bereavement. *Personality and Social Psychology Bulletin, 30*, 878–890.

Fraley, R. C., & Shaver, P. R. (1997). Adult attachment and the suppression of unwanted thoughts. *Journal of Personality and Social Psychology, 73*, 1080–1091.

Fraley, R. C., & Shaver, P. R. (2000). Adult romantic attachment: Theoretical developments, emerging controversies, and unanswered questions. *Review of General Psychology, 4*, 132–154.

Gaines, S. O. Jr., Reis, H. T., Summers, S., Rusbult, C. E., Cox, C. L., Wexler, M. O., et al. (1997). Impact of attachment style on reactions to accommodative dilemmas in close relationships. *Personal Relationships, 4*, 93–113.

Gillath, O., Bunge, S. A., Shaver, P. R., Wendelken, C., & Mikulincer, M. (2005). Attachment-style differences in the ability to suppress negative thoughts: Exploring the neural correlates. *NeuroImage, 28*, 835–847.

Granqvist, P., Mikulincer, M., Gurwitz, V., & Shaver, P. R. (2012). Experimental findings on God as an attachment figure: Normative processes and moderating effects of internal working models. *Journal of Personality and Social Psychology, 103*, 804–818.

Hannon, P. A., Rusbult, C. E., Finkel, E. J., & Kumashiro, M. A. (2010). In the wake of betrayal: Perpetrator amends, victim forgiveness, and the resolution of betrayal incidents. *Personal Relationships, 17*, 253–278.

Hobdy, J., Hayslip, B., Kaminski, P. L., Crowley, B. J., Riggs, S., & York, C. (2007). The role of attachment style in coping with job loss and the empty nest in adulthood. *International Journal of Aging and Human Development, 65*, 335–371.

Holmberg, D., Lomore, C. D., Takacs, T. A., & Price, E. L. (2011). Adult attachment styles

and stressor severity as moderators of the coping sequence. *Personal Relationships, 18*, 502–517.

Jerga, C., Shaver, P. R., & Wilkinson, R. B. (2011). Attachment insecurities and identification of at-risk individuals following the death of a loved one. *Journal of Social and Personal Relationships, 28*, 891–914.

Karremans, J. C., & Aarts, H. (2007). The role of automaticity in the inclination to forgive close others. *Journal of Experimental Social Psychology, 43*, 902–917.

Kidd, T., Hamer, M., & Steptoe, A. (2011). Examining the association between adult attachment style and cortisol responses to acute stress. *Psychoneuroendocrinology, 36*, 771–779.

Lemche, E., Giampietro, V. P., Surguladze, S. A., Amaro, E. J., Andrew, C. M., Williams, S. C., et al. (2006). Human attachment security is mediated by the amygdala: Evidence from combined fMRI and psychophysiological measures. *Human Brain Mapping, 27*, 623–635.

Lopez, F. G., & Gormley, B. (2002). Stability and change in adult attachment style over the first-year college transition: Relations to self-confidence, coping, and distress patterns. *Journal of Counseling Psychology, 49*, 355–364.

Master, S. L., Eisenberger, N. I., Taylor, S. E., Naliboff, B. D., Shirinyan, D., & Lieberman, M. D. (2009). A picture's worth: Partner photographs reduce experimentally induced pain. *Psychological Science, 20*, 1316–1318.

Maunder, R. G., Lancee, W. J., Nolan, R. P., Hunter, J. J., & Tannenbaum, D. (2006). The relationship of attachment insecurity to subjective stress and autonomic function during standardized acute stress in healthy adults. *Journal of Psychosomatic Research, 60*, 283–290.

Medway, F. J., Davis, K. E., Cafferty, T. P., Chappell, K. D., & O'Hearn, R. E. (1995). Family disruption and adult attachment correlates of spouse and child reactions to separation and reunion due to Operation Desert Storm. *Journal of Social and Clinical Psychology, 14*, 97–118.

Mikulincer, M., Dolev, T., & Shaver, P. R. (2004). Attachment-related strategies during thought-suppression: Ironic rebounds and vulnerable self-representations. *Journal of Personality and Social Psychology, 87*, 940–956.

Mikulincer, M., & Florian, V. (1995). Appraisal of and coping with a real-life stressful situation: The contribution of attachment styles. *Personality and Social Psychology Bulletin, 21*, 406–414.

Mikulincer, M., & Florian, V. (1998). The relationship between adult attachment styles and emotional and cognitive reactions to stressful events. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 143–165). New York: Guilford Press.

Mikulincer, M., & Florian, V. (1999). Maternal–fetal bonding, coping strategies, and mental health during pregnancy: The contribution of attachment style. *Journal of Social and Clinical Psychology, 18*, 255–276.

Mikulincer, M., Gillath, O., & Shaver, P. R. (2002). Activation of the attachment system in adulthood: Threat-related primes increase the accessibility of mental representations of attachment figures. *Journal of Personality and Social Psychology, 83*, 881–895.

Mikulincer, M., Hirschberger, G., Nachmias, O., & Gillath, O. (2001). The affective component of the secure base schema: Affective priming with representations of attachment security. *Journal of Personality and Social Psychology, 81*, 305–321.

Mikulincer, M., & Shaver, P. R. (2001). Attachment theory and intergroup bias: Evidence that priming the secure base schema attenuates negative reactions to out-groups. *Journal of Personality and Social Psychology, 81*, 97–115.

Mikulincer, M., & Shaver, P. R. (2007a). *Attachment in adulthood: Structure, dynamics, and change*. New York: Guilford Press.

Mikulincer, M., & Shaver, P. R. (2007b). Boosting attachment security to promote mental health, prosocial values, and inter-group tolerance. *Psychological Inquiry, 18*, 139–156.

Mikulincer, M., Shaver, P. R., Sapir-Lavid, Y., & Avihou-Kanza, N. (2009). What's inside the minds of securely and insecurely attached people?: The secure-base script and its associations with attachment-style dimensions. *Journal of Personality and Social Psychology, 97*, 615–633.

Mikulincer, M., Shaver, P. R., & Slav, K. (2006). Attachment, mental representations of others, and gratitude and forgiveness in romantic relationships. In M. Mikulincer & G. S. Goodman (Eds.), *Dynamics of romantic love: Attachment, caregiving, and sex* (pp. 190–215). New York: Guilford Press.

Park, L. E. (2010). Responses to self-threat: Linking self and relational constructs with approach and avoidance motivation. *Social*

and Personality Psychology Compass, 4, 201–221.

Perunovic, M., & Holmes, J. G. (2008). Automatic accommodation: The role of personality. *Personal Relationships, 15*, 57–70.

Quirin, M., Gillath, O., Pruessner, J. C., & Eggert, L. D. (2010). Adult attachment insecurity and hippocampal cell density. *Social Cognitive and Affective Neuroscience, 5*, 39–47.

Quirin, M., Pruessner, J. C., & Kuhl, J. (2008). HPA system regulation and adult attachment anxiety: Individual differences in reactive and awakening cortisol. *Psychoneuroendocrinology, 33*, 581–590.

Raskin, P. M., Kummel, P., & Bannister, T. (1998). The relationship between coping styles, attachment, and career salience in partnered working women with children. *Journal of Career Assessment, 6*, 403–416.

Rusbult, C. E., Verette, J., Whitney, G. A., Slovik, L. F., & Lipkus, I. (1991). Accommodation processes in close relationships: Theory and preliminary empirical evidence. *Journal of Personality and Social Psychology, 60*, 53–78.

Sbarra, D. A. (2006). Predicting the onset of emotional recovery following nonmarital relationship dissolution: Survival analyses of sadness and anger. *Personality and Social Psychology Bulletin, 32*, 298–312.

Sbarra, D. A., & Emery, R. E. (2005). The emotional sequelae of nonmarital relationship dissolution: Analysis of change and intraindividual variability over time. *Personal Relationships, 12*, 213–232.

Scharfe, E., & Bartholomew, K. (1995). Accommodation and attachment representations in couples. *Journal of Social and Personal Relationships, 12*, 389–401.

Selcuk, E., Zayas, V., Günaydin, G., Hazan, C., & Kross, E. (2012). Mental representations of attachment figures facilitate recovery following upsetting autobiographical memory recall. *Journal of Personality and Social Psychology, 103*, 362–378.

Shaver, P. R., & Mikulincer, M. (2002). Attachment-related psychodynamics. *Attachment and Human Development, 4*, 133–161.

Smeets, T. (2010). Autonomic and hypothalamic–pituitary–adrenal stress resilience: Impact of cardiac vagal tone. *Biological Psychology, 84*, 290–295.

Waters, H. S., & Waters, E. (2006). The attachment working models concept: Among other things, we build scriptlike representations of secure base experiences. *Attachment and Human Development, 8*, 185–198.

Wayment, H. A., & Vierthaler, J. (2002). Attachment style and bereavement reactions. *Journal of Loss and Trauma, 7*, 129–149.

Williams, N. L., & Riskind, J. H. (2004). Adult romantic attachment and cognitive vulnerabilities to anxiety and depression: Examining the interpersonal basis of vulnerability models. *Journal of Cognitive Psychotherapy, 18*, 7–24.

Yárnoz-Yaben, S. (2009). Forgiveness, attachment, and divorce. *Journal of Divorce and Remarriage, 50*, 282–294.

Zhang, F., & Labouvie-Vief, G. (2004). Stability and fluctuation in adult attachment style over a 6-year period. *Attachment and Human Development, 6*, 419–437.

Zilcha-Mano, S., Mikulincer, M., & Shaver, P. R. (2011). Pets as safe havens and secure bases: The moderating role of pet attachment orientations. *Journal of Research in Personality, 45*, 345–357.

CHAPTER 16

Attitudes, Evaluation, and Emotion Regulation

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Research and theory in the domain of attitudes and evaluation are relevant to emotion due to their many overlaps and parallels. Most fundamentally, they share at their core the concept of valence. Attitudes encode the valence of an object: that is, goodness or badness, desirability or undesirability, pleasure or pain. Valence is similarly integral to the concept of emotion; some theories argue that all emotions, at their core, can be distilled to whether they are positive or negative (e.g., Russell, 2003; Watson & Tellegen, 1985). Valence's function is to guide appropriate action. In the case of attitudes, that functionality lies in the ability of valence to provide a rapid, default assessment of an object's value; we need not constantly judge the world anew.

Yet this tool that may even be lifesaving in times of peril can also be a liability. Reflexively acting in accordance with one's feelings is not always appropriate; indeed, the feeling itself may be deemed problematic. For example, prejudiced group attitudes and evaluations may conflict with social norms or one's values, leading to reconciliatory processes of justification or suppression (Crandall, Eshleman, & O'Brien, 2002). We also argue that evaluation entails a process of affect regulation in and of itself. Even in the absence of a particular desire to change one's evaluation, regulation is occurring. Increasingly, contemporary perspectives on attitudes view evaluations as the product of dynamic processes that are shaped by the demands of the immediate situation. Relatively stable mental representations underlie the very earliest evaluative processing; however, in a way that can be viewed as fundamentally self-regulatory (e.g., Carver & Scheier, 1982), evaluations are sensitive to various goals and standards for thoughts, feelings, and behavior.

Regarding the volitional self-regulation of evaluation, it will be uncontroversial to note that people are not merely passive receivers of their psychological experiences; valence may be not only experienced but also assessed and modified. Though evaluation has some characteristics of cued responding grounded by preexisting structure, people do not respond to the same cue in an invariable fashion. One may generally dislike something (say, golf) but temper that affective response, or even reverse it, when the situation demands. For example, when offered the opportunity to golf with a work supervisor, one might be motivated to deemphasize the object's disliked features and emphasize its liked ones (e.g., defining golf more by its aesthetic value than by, say, its early-morning tee times), or reevaluate its features (e.g., considering how golf's difficulty might constitute a virtue rather than

an annoyance). Evaluations are often shaped to conform to appropriate action. As such, when considering the use of attitudes to guide behavior, the regulation of affect is important. One type of evaluative regulation entails the inhibition and reshaping of "gut reactions" of liking in accordance with their perceived appropriateness. Beyond this, we emphasize processes within even quite rapid and spontaneous evaluations (those very gut reactions) that evidence self-regulatory flexibility in experienced object-directed affect. Relatedly, Gross and Barrett (2011) suggest the possibility that emotion generation and regulation can be considered the same process: Information may be simultaneously processed as affectively relevant and informed by top-down processes that take contextual cues (e.g., situational appropriateness) into account. Similarly, Todd, Cunningham, Anderson, and Thompson (2012) have suggested that emotion can be simultaneously shaped and regulated through early attentional biases. In making this argument, we hope to highlight the potential for greater integration between the literatures on affect regulation and attitudes, which as yet have stood largely separate.

At the outset, some clarification of our usage of the terms *attitude* and *evaluation* will be useful. Some prominent definitions have defined *attitudes* generally: predispositions to respond positively or negatively to an object (e.g., Allport, 1935; Eagly & Chaiken, 1993), or as a constellation of affective, cognitive, and behavioral responses associated with an object (Zanna & Rempel, 1988). In contrast, in a more specific and limited definition, Fazio (2007) defines *attitudes* as associations in memory between objects and summated valenced information about those objects. This approach is the one we take here, defining attitudes as *representational*, which means that attitudes signify valenced information stored in memory that has the potential to be activated. We do, however, distinguish representational attitudes from evaluation—a *process* by which valence is constructed in both bottom-up and top-down fashion. In this way, we integrate a representational definition, with the approach to attitudes typically considered most antithetical to it: the constructivist approach (e.g., Schwarz & Bohner, 2001). This approach tends not to deny the existence of mental representations but chooses to deemphasize them, focusing instead on contextual factors that shape experiences of liking and disliking. In summary, we use the term *attitude* to refer to a structural entity in memory, and the term *evaluation* to refer to an appraisal process informed by an attitude that unfolds over time in a particular context. Thus, evaluation can be considered a process of affect regulation in the moment, fostering context appropriateness. Given our terminology, attitudes may also be subject to self-regulation, such as when one attempts to change how much one generally likes something. We primarily focus on the former.

It is the interplay of attitudes and evaluation that provides true functionality. A rigid retrieval system would be insensitive to context and beholden to even limited past experience. A fully constructed system would be slow, inefficient, and too unresponsive to lessons learned. Instead, we have both capabilities. The transformation of attitudes into evaluations is a form of emotion regulation in which situational factors can play a role even in immediate responses. We argue that an attitude is a triggered affective response shaped into a contextually appropriate evaluation via a process of iterative reprocessing (Cunningham & Zelazo, 2007). This updating occurs as the situation unfolds; thus, affect regulation is critical to all stages of the attitude-to-evaluation transformation—from the processes typically considered automatic to the processes that are typically considered controlled. Thus, although the effortful inhibition of evoked attitudes is a major topic, our conceptualization of regulation goes beyond this to include a variety of processes that shape adaptive responding. This broader view of emotion regulation is consistent with evolving views on self-regulation generally, in which inhibition is not a *sine qua non* (e.g., Fujita, 2011).

In this chapter, we review contemporary attitude theories that entail an emphasis on regulation. Following a discussion of two foundational models to most recent approaches, we focus in detail on contemporary treatments of attitude and evaluation that illustrate the self-regulatory shaping of rapid response. We highlight the iterative reprocessing (IR) model (Cunningham & Zelazo, 2007), which not only addresses such processes descriptively but also provides a perspective from neuroscience regarding their underlying brain substrates. We use the

IR model as a framework to address differences in theory and empirical controversy that characterize this literature.

Two Foundational Attitude Models

Models of attitudes commonly emphasize some distinction between implicit and explicit attitudes or evaluative processes. Undoubtedly, this is in large part due to the introduction of reaction-time-based implicit attitude measures, such as evaluative priming (Fazio, Jackson, Dunton, & Williams, 1995) and the Implicit Association Test (IAT; Greenwald, McGhee, & Schwarz, 1998). Researchers had long hoped to find a solution to the problem of dishonest responding to attitude measures out of social desirability concerns. These implicit measures offered the best solution to date by engaging participants in a task that might reveal attitudes without actually calling on participants to express them.

Interestingly, the new implicit attitude measures appear to diverge from explicit measures (self-report) particularly when participants' self-presentational concerns are high (e.g., Fazio et al., 1995; Olson, Fazio, & Hermann, 2007), yet the measures also diverge even when participants' incentives to misrepresent themselves are minimal. For example, Karpinski and Hilton (2001) found null relations between IAT and self-report measures of preference for insects versus flowers and apples versus candy bars, seemingly noncontroversial attitude objects. Furthermore, it appeared that social influence (not necessarily intentional misrepresentation) was especially evident on the *implicit* measure rather than the explicit measure. That is, the IAT suggested a greater affinity for the healthy (i.e., socially valued) alternative than was evident in explicit behavior. This example highlights the finding that dissociations between implicit and explicit measures are common and complex (for a meta-analytic review, see Hofmann, Gawronski, Gschwendner, Le, & Schmitt, 2005), and that these dissociations cannot be attributed exclusively to misrepresentation on self-report measures or measurement error and lack of structural fit (Payne, Burkley, & Stokes, 2008).

Models need to account for these dissociations, and incorporating some distinction between implicit and explicit attitudes or evaluations is a common way of doing so. However, something like this distinction has been long recognized. For example, Hovland, Janis, and Kelley (1953, cited in Petty & Briñol, 2006, p. 740) distinguished attitudes ("implicit responses" that are "sometimes unconscious") from opinions ("verbal answers that one covertly expresses"). The recent accumulated evidence from implicit measures can be understood as validation of the long-appreciated observation that rapid, unintentional responses (what one might call "gut reactions") can differ a great deal from carefully considered evaluations. Accounting fully for the processes that distinguish them is a central issue in contemporary attitude theory and obviously important for self-regulation. We first discuss two influential models that have been recently built upon to explain differences between immediate and subsequent responses.

The MODE Model

The acronym MODE refers to *M*otivation and *O*pportunity as *DE*terminants of whether the attitude-to-behavior process is relatively spontaneous or deliberative in nature. The MODE model (Fazio, 1990; Olson & Fazio, 2009) posits a single attitude representation, and defines an *attitude* as the association in memory between an object and a summary evaluation thereof (see Fazio, 2007). The model is largely agnostic about the nature of attitude representation. It is readily understood in terms of a "schema plus tag" model, in which summary evaluations are a special form of tag associated with an attitude object. However, it is also consistent with more recent dynamic and connectionist models if the term *summary* is understood to refer to distributed information in memory (Eiser, Fazio, Stafford, & Prescott, 2003). Regardless, the MODE model is primarily concerned with how and when attitudes determine behavior.

The MODE model predicts whether the attitude-to-behavior process is relatively spontaneous or deliberative in nature. The model notes that the primary function of attitudes is to provide a rapid assessment of objects in the environment to facilitate appropriate behavior in a timely fashion. Attitudes can be activated automatically, and the likelihood of automatic attitude

activation is a function of the associative strength between the attitude object and the associated evaluation (see Fazio, 2001). If there is little *motivation* for further consideration of the object, for whatever reason, or low *opportunity* (e.g., time pressure or cognitive load) to do so, attitudes should guide behavior in a spontaneous, largely automatic fashion, in which they promote congruent behaviors afforded by the situation. Controlled deliberation about the attitude and behavior is likely only when the motivation *and* opportunity to do so are present. This deliberation might lead to the alteration or rejection of an attitude and the behavior it facilitates, but it could also affirm it. In fact, the model suggests that affirmation is most typical, because the activated attitude fosters biased processing. Alteration or rejection of the attitude depends on various factors, including contextual influences on the object's utility, social influence, object reappraisal, and so on.

In summary, the MODE model posits a single attitude associated with each discrete representation of an attitude object that may be retrieved from memory, often automatically. That attitude is likely to shape subsequent judgment and behavior; however, the attitude's influence is especially likely to be direct for spontaneous behaviors. When the motivation and opportunity to deliberate are present, the attitude might be disregarded, adjusted for reasons including context or apparent appropriateness, or validated by deliberation. The MODE model's implications for emotion regulation are straightforward. The model applies directly to circumstances in which one wishes to control automatic emotional responses to a particular object. It points to motivation and opportunity as broad categories encompassing many specific variables and emphasizes that any major impediment to motivation or ability drastically reduces the likelihood of deliberative regulation.

To account for discrepancies between implicit and explicit attitudes, the MODE perspective suggests that implicit measures largely reflect automatically activated attitudes, while explicit measures capture deliberative evaluations, which may or may not reflect attitudes for the reasons described. Another way of explaining these differences is to evoke two different attitude *representations* to which the measures are differentially sensitive. The notion of an implicit attitude is often attributed to Greenwald and Banaji (1995), who wrote: "*Implicit attitudes* are introspectively unidentified (or inaccurately identified) traces of past experience that mediate favorable or unfavorable feeling, thought, or action toward social objects" (p. 8). It was not until Wilson, Lindsey, and Schooler's (2000) dual-attitudes model, however, that a major process model of dual attitudes was articulated.

The Dual-Attitudes Model

In the original conception of "implicit attitudes," such attitudes, by definition, were considered to be unknown to the evaluator (Greenwald & Banaji, 1995). The absence of awareness is most directly the facet of automaticity (see Bargh, 1994) referred to by the term *implicit*. However, the term has been used in a wide variety of ways, leading some to argue that because *implicit* de facto has become a catchall descriptor of (relatively) automatic processes, it should be understood as such (De Houwer & Moors, 2007). Wilson and colleagues (2000), for example, do not define *implicit attitudes* as being characterized by unawareness. They suggested that "people are often aware, at least fleetingly, of [implicit attitudes]" (p. 105). What, then, is an implicit attitude in this model?

According to the dual-attitudes model (Wilson et al., 2000), implicit attitudes are, in a word, old (or prior). That is, when an attitude representation changes, it is not fully supplanted by a new, altered attitude. Rather, both representations can coexist in memory. Thus, an implicit attitude is a prior, often deeply ingrained attitude. By virtue of its precedence and having been rehearsed, the implicit attitude is strongly associated with the attitude object. Thus, it will likely be automatically activated upon encountering an attitude object. Similar to the MODE model, the dual-attitudes model posits that "capacity and motivation" are required to retrieve the newer, explicit attitude effortfully. Retrieval of the explicit attitude is expected to lead to its application, especially for controlled behaviors. The simultaneous activation of implicit and explicit attitudes may also demonstrate unintended influence of the implicit attitude, especially on unmonitored or uncontrollable nonverbal

responses (e.g., Dovidio, Kawakami, Johnson, Johnson, & Howard, 1997).

Wilson et al. (2000) further distinguish a typology of four kinds of implicit attitudes, which have different characteristics due to their differing origins. Briefly, an implicit attitude might arise from repression, in which an anxiety-provoking implicit attitude is expelled from consciousness and is thus implicit in the sense of being unknown. Another implicit attitude arises from motivated overriding, when an attitude is unwanted and effortfully suppressed. A third implicit attitude arises from automatic overriding, when the process of overriding such an implicit attitude itself becomes automatized through repetition. In this case, capacity and motivation are not actually required for the explicit rather than implicit attitude to predominate. Finally, the dual-attitudes model suggests that implicit attitudes may differ from explicit attitudes simply by virtue of having an entirely separate neurocognitive substrate (i.e., independent systems; see Lieberman, 2007, for such a view) but does not elaborate much on this possibility, which distinguishes some more recent models.

In some ways, the implications of the dual-attitudes model for emotion regulation are similar to those of the MODE model. Certainly, the necessity of motivation and ability/capacity for any sort of controlled emotion regulation is again highlighted. The different types of implicit attitudes described, however, are uniquely relevant. The case of repression, though its very existence is controversial, poses an interesting problem. In this case, one's own affective reactions to an object are anxiety provoking, instigating defensive reactions that suppress and exclude them from consciousness. Motivated overriding essentially describes a phenomenon of successful affective regulation, but it does not elaborate much about *how* this occurs, especially under difficult circumstances, with the exception that automatizing a regulatory response may occur through repetition. This is an interesting possibility in the realm of emotion regulation.

Summary of Historical Perspectives

To summarize, these models in their early incarnations posited implicit or activated attitudes that were largely unregulated. Schema-plus-tag or implicit-attitude-as-habitual-response conceptualizations of attitude share the consequence of rapid responses that are quite inflexible and context-free. Affective regulation is then possible if and only if one is motivated and able to suppress, adjust, enhance, or replace (i.e., effortfully retrieve an explicit attitude) an evaluation. However, the strong form of this view is inconsistent with findings that are indicative of self-regulatory responding even in rapid responses evident on implicit measures. For example, Lowery, Hardin, and Sinclair (2001) demonstrated that racial attitudes as measured by an IAT were sensitive to social tuning—that is, they were influenced by the presumed attitudes of other individuals present. Increasingly, recent models address adaptive flexibility in processes occurring during the construction of an evaluation. Notably, a similar development has occurred in theorizing about stereotyping. Automatic stereotype activation was often viewed as inevitable (Bargh, 1999; Devine, 1989) and could only be overridden by controlled processes. Subsequent research in this domain suggested a much more optimistic view of flexibility and self-regulation at the earliest stages of processing (e.g., Jones & Fazio, 2010; Moskowitz, Gollwitzer, Wasel, & Schaal, 1999; Sassenberg & Moskowitz, 2005). In contemporary attitude models, we see a greater emphasis on malleability in rapid responses. This malleability can be viewed as demonstrating the fundamentally regulatory nature of evaluation. We turn now to recent models including several variations on this theme.

Contemporary Attitude Models

The Associative–Propositional Evaluation Model

Probably the most influential contemporary approach to attitudes and evaluation is the associative–propositional evaluation (APE) model, which originally was introduced to explain the complex literature on the impact of interventions that form or change attitudes on implicit and explicit measures (Gawronski & Bodenhausen, 2006). Since then, the APE model has been broadened to address attitudes and evaluation more generally (see Gawronski & Bodenhausen, 2011).

The APE model describes two interactive but distinct types of processes that underlie evaluation: associative and propositional processes.[1] *Associative* processes activate associated representations from memory. This activation is presumed to follow the principles of contiguity and similarity; *contiguity* refers to the spatiotemporal proximity of stimuli that determines the structure of memory, whereas *similarity* refers to the fit between encountered objects and mental representations. *Propositional* processes involve the validation of information activated by associative processes. Unlike associative processes, propositional processes entail subjective assessments of truth values (i.e., veridicality of information). They are posited to operate according to principles of logical consistency, though in the sense of perceived coherence rather than formal logic per se. Associative processes are argued to underlie implicit evaluations, and propositional processes are argued to underlie explicit evaluations. Thus, if implicit evaluations are perceived as valid, they will be used for explicit evaluations; if they are not, they are modified.

The APE model suggests that, following connectionist models of associative memory, information associated with a given attitude object is distributed across a network of weighted connections. The activation of a given piece of information associated with an object is typically probabilistic, in a manner that maximizes the likelihood of the most relevant information being activated, which can be viewed as regulatory. The particularities of the input stimulus, as well as incidentally active information due to recency of use, determine which subset of associations is activated. This allows for different implicit evaluations as a function of context, consistent with malleability often observed on implicit attitude measures (e.g., Barden, Maddux, Petty, & Brewer, 2004). These implicit evaluations provide the grist for propositional processing. If the propositional implications of an implicit evaluation are inconsistent with other salient propositions, an aversive state of cognitive dissonance is evoked. *Cognitive dissonance*, induced by the simultaneous activation of conflicting information, is an aversive state of arousal that motivates efforts to alleviate it (Festinger, 1957). Various methods of restoring balance and consistency have long been recognized, such as rejecting one of the propositions or introducing another proposition that reconciles or trivializes the apparent conflict.

Importantly, associative and propositional processes are argued to be interactive. In addition to the bottom-up processes in which activated associations determine the kinds of propositions that are evoked, there are also top-down processes that create or activate further associations (see Gawronski & Bodenhausen, 2011, for details). The APE model is useful for explaining the highly varied patterns of results that have been observed when implicit and explicit measures follow manipulations meant to form or change attitudes. Earlier models, particularly those that emphasize the stability of implicit evaluations (e.g., Wilson et al., 2000), have trouble explaining circumstances in which implicit measures are more sensitive to manipulation than explicit measures (e.g., Gawronski & LeBel, 2008; Karpinski & Hilton, 2001). The APE model, in contrast, can explain when information will recruit associative versus propositional processes.

Some types of interventions are likely to have direct effects on associative processes (Gawronski & Bodenhausen, 2011). For example, evaluative conditioning, a phenomenon in which an attitude toward an object forms or changes due to its co-occurrence with one or more valenced objects, is likely to have these types of direct associative effects (for reviews, see De Houwer, Thomas, & Baeyens, 2001; Jones, Olson, & Fazio, 2010). The repeated pairing of objects during conditioning procedures facilitates association formation and strengthening for obvious reasons. Other types of interventions are likely to have direct effects on propositional processes. Inductions of cognitive inconsistency (e.g., Gawronski & Strack, 2004) impact propositional processes directly but may leave relevant associations intact. Events might also directly influence both associative and propositional processes. For example, a persuasive message could change the propositions that are considered and also create new associations in memory.

Finally, events might directly influence one process and indirectly influence the

other. Any change in associative structure can potentially influence the propositions that are generated and assessed, the path taken to reduce cognitive dissonance, and so forth. The propositions an individual considers can create or change the associative strength of associations, even in the absence of external stimulation. A fuller account of the details of the APE model (see Gawronski & Bodenhausen, 2006, 2011) is beyond the scope of this chapter, but it should be noted that the APE model offers many specific hypotheses about the eliciting circumstances and processes underlying patterns of attitude change evident in implicit and explicit measures.

One issue addressed by the APE model that has interesting implications for affect regulation concerns negation. The act of negating a proposition may be insufficient to change the relevant associative structure in memory. Indeed, though negation may influence propositional processes in a straightforward manner (i.e., processing consistent with the negated proposition), the act of negation may have ironic effects at the associative level due to the coactivation of elements (e.g., Deutsch, Gawronski, & Strack, 2006). For example, telling oneself, "I am not afraid of giving a speech," may have some immediate positive consequences due to its consciously processed implication. However, this also risks increasing the (automatic) association in memory between fear and public speaking merely because of their simultaneous activation. Gawronski and Bodenhausen (2011) suggest that the overall success of regulating future affective reactions depends on whether unwanted propositions are negated ("I'm not afraid of public speaking") or preferable propositions are affirmed ("Public speaking is fun"), implying that affirmation works better than negation for emotion regulation.[2] For example, a study in which stereotypical associates were negated led to *increased* automatic stereotype activation and, significantly for the question of affect regulation, increased bias on an evaluative priming task (Gawronski, Deutsch, Mbirkou, Seibt, & Strack, 2008). Gawronski and Bodenhausen (2011) suggested that such ironic effects may bear some responsibility for the general superiority in emotion regulation (Gross, 1998) of an affirmative strategy of reappraisal as opposed to a negative strategy of suppression. However, other work suggests that generating or encountering negations may have positive influences on affect regulation. For example, Herbert, Deutsch, Sütterlin, Kübler, and Pauli (2011; see also Mauss, Evers, Wilhelm, & Gross, 2006) examined startle eyeblink responses to pleasant and unpleasant nouns that had or had not been negated. They found (with ample processing time) online effects of negation consistent with the logical meaning: reduced startle response to negated unpleasant words and increased response to negated pleasant words. Thus, the role of negation in affect regulation remains controversial.

The APE model suggests that the interplay of associative and propositional processes underlies evaluation. It is sometimes misunderstood to be a dual-system or dual-representation model but it explicitly is not (Gawronski & Bodenhausen, 2011, pp. 104–105). The APE model argues that all information is stored in the form of associations, and that there is not a separate store for propositions, thus rejecting the possibility of dual representations. The APE model does not explicitly endorse or reject a dual-system approach, which emphasizes separate mental systems (i.e., neurocognitive substrates).[3] Some researchers believe that the sometimes stark dissociations between observed implicit and explicit evaluations are best explained by their origination from separate systems. We turn now to a recent model of dual systems to explore this idea in greater detail.

The Systems of Evaluation Model

The systems of evaluation model (SEM) describes an *associative system* that produces implicit evaluations and a *rule-based system* that produces explicit evaluations (McConnell & Rydell, in press; Rydell & McConnell, 2006). It builds on prior dual-system models of cognition, particularly Sloman's (1996) model of fast and slow learning systems of reasoning. These partially independent systems operate in parallel and differ in the type of knowledge they use and the operations conducted on that knowledge. The associative system reflects associations governed by principles of similarity and contiguity, whereas the

rule-based system operates on symbolic representations (e.g., language) that are subjected to operations of logic and deductive reasoning. Because implicit measures tend to reflect construct activation and explicit measures tend to reflect symbolic reasoning, the measures differ in their sensitivity to the output of the two systems. This suggests that the primary distinction between these systems is similar to the APE model. However, a major difference is that the SEM emphasizes distinctive characteristics of the two systems that lead to differential rates of change. The SEM suggests that the explicit system is faster, while the implicit system is slower, both to form and to change attitudes. Explicit evaluations "can be formed and modified relatively quickly because logic and syllogism are responsive to one's deliberate goals and deductive reasoning processes," whereas implicit evaluations "typically are slower to form and change because they are based on accumulated attitude object-evaluation pairings in memory" (McConnell & Rydell, in press). In this way, the SEM bears some resemblance to the earlier dual-attitudes model of Wilson and colleagues (2000), insofar as an implicit attitude tends to be distinguished by stability. However, the SEM does not posit separate stores for "old" and "new" attitudes, nor does it consider prior attitude change necessarily responsible for divergence on implicit and explicit measures. Though implicit measures are sometimes quite labile, this can be reconciled with the SEM by noting that this often appears to be due to activation of different subset of associations from memory that influence an implicit measure, among other reasons, rather than a rapid change in particular associative representations.

The SEM is supported by research demonstrating strong dissociations between implicit and explicit measures, consistent with a dual-systems view. For example, Rydell and McConnell (2006, Experiment 2) presented participants with a series of behaviors performed by a target individual. Presenting a large number of positive or negative behaviors established positive or negative attitudes, respectively, that were evident on both implicit and explicit measures. This was followed by a series of counterattitudinal behaviors. When 100 initial behaviors were followed by 20 conflicting behaviors (i.e., positive followed by negative, or vice versa), explicit attitude measures showed considerable sensitivity to new information, while implicit measures were largely unaffected by it. When 100 counterattitudinal behaviors followed the initial 100, both types of measures reflected the new information. This is interpreted as being consistent with the presence of a slow-learning associative system and fast rule-based system. In other work supportive of the SEM, a series of subliminal primes was paired with target individuals (Rydell, McConnell, Mackie, & Strain, 2006). Because repeated subliminal priming might produce the gradual association of affect with an attitude object, it would be expected to influence the associative system. Moreover, by virtue of being subliminal, such a manipulation would not provide grist for the rule-based system. However, the rule-based system should be especially sensitive to the consciously accessible behavioral information that was presented, as evidenced by influences on explicit measures. Results in line with these hypotheses were obtained: Across several studies, implicit attitudes formed and changed in response to subliminal primes, and explicit attitudes formed and changed in response to consciously accessible information.

An interesting aspect of the SEM with wide implications for affect regulation is the study of the consequences of discrepancy between implicit and explicit evaluations. One consequence of implicit–explicit discrepancy involves the extent of information processing. It has been suggested that these discrepancies lead to increased information processing (e.g., Petty, Tormala, Briñol, & Jarvis, 2006). Moreover, recent work has suggested that this may be due to the arousal of cognitive dissonance caused by the conflicting outputs of the two systems (Rydell, McConnell, & Mackie, 2008). Seeking out information to resolve the discrepancy is a major route to dissonance reduction. However, the source of these aversive feelings can be mysterious to those experiencing them, and consequently, the feelings might be misattributed to inappropriate sources. Specifically, negative arousal caused by attitude formation with valence-inconsistent information led participants to report lower subjective well-being (Rydell & Durso, 2012). Thus, the SEM suggests that when

the systems of evaluation produce discrepant evaluations, negative affect and affective regulation tend to follow. Another relevant consequence of implicit–explicit discrepancy involves affective forecasting. It has also been suggested that thoughtful affective forecasting relies on explicit evaluations of objects, ignoring implicit evaluations (McConnell, Dunn, Austin, & Rawn, 2011). Consistent with this idea, implicit, but not explicit, attitudes predicted error in affective forecasting when considering future enjoyment of foods.

The Meta-Cognitive Model

Petty and colleagues' meta-cognitive model (MCM; Petty, 2006; Petty et al., 2006; Petty, Briñol, & DeMarree, 2007) has elements of most of the other models we have discussed. It focuses on how attitudes might be stored in memory, with particular emphasis on meta-cognitive perceptions of the validity of responses. Moreover, the MCM draws on research arguing that the neural substrates of positive and negative valence are distinct (e.g., Cacioppo, Gardner, & Berntson, 1997), suggesting that separate positive and negative evaluations of an object might be represented in memory.

According to the MCM, though many attitudes are predominantly univalent, it is not uncommon for an object to be associated with both positive and negative summary evaluations. Whether the positive or negative attitude associated with an object is activated depends on associative strength, recency of prior positive and negative experiences, and whether the current context is a better fit with the positive or negative association. Reflecting on these attitudes can lead them to become associated with a tag, marking them as either valid or invalid. The associated confidence indicator serves as a signal to utilize or disregard the attitude with minimal reflection required. However, the MCM notes that validity tags also vary in their associative strength to the evaluation and in accessibility, meaning that sometimes the tag may not be retrieved from memory when the attitude object is. Often, attitudes marked with an invalidity tag are prior assessments that have been changed but still may exert an influence (Petty et al., 2006). The MCM concurs with the APE in the assumption that the default response to association activation is acceptance, the presumption of validity (see also Gilbert, 1991). Therefore, it tends to matter more when an *in*validity tag is not retrieved, in which case a rejected evaluation is likely to influence judgment and behavior. The MCM distinguishes between explicit ambivalence, in which both positive and negative evaluations are associated with validity tags, and implicit ambivalence, in which one association is validated and the other is not. Explicit ambivalence is perceived as subjectively conflicting when it is experienced, but implicit ambivalence is not, due to the conscious rejection of either the positive or negative evaluation.

In summary, the MCM suggests that the creation and retrieval of validity tags serve a self-regulatory function by marking which associations are (in)appropriate for guiding action. Unlike the other models discussed, the MCM particularly may be seen as clearly concerning the regulation of attitudes per se. Validity tags concern the associative network of an attitude and the adaptive regulation of relatively generalized responses that can guide later action. Though all models concern the regulation of attitudes insofar as the process of evaluation not only recruits but also changes attitudes in memory, the MCM is particularly useful for considering how attitude structure changes over time.

The Iterative Reprocessing Model and Dynamism

Our final contemporary perspective emphasizes dynamism in evaluation and regulation. *Dynamism* refers to the notion that mental representations are constantly evolving states in which partial representations are cascading, competing, and interactive. The dynamical approach to processing (e.g., Spivey, 2007) has been applied to multiple social psychological concepts, including categorization (Freeman, Ambady, Rule, & Johnson, 2008), stereotyping (Freeman & Ambady, 2009), and evaluation (Wojnowicz, Ferguson, Dale, & Spivey, 2009). Wojnowicz and colleagues argue that explicit evaluations are "merely the end result of a complex, non-linear, time-dependent process of multiple less-explicit attitudes competing with one another over hundreds of milliseconds" (p. 1428). In early processing, mul-

tiple patterns of evoked activity are partially consistent with various evaluations. Over time, "a continuous accrual of information causes the distributed pattern to dynamically 'sharpen' into a confident (selected) interpretation, forcing other, partially activated, competing alternative[s] . . . to gradually die out" (p. 1429), an idea consistent with the connectionist modeling notion of an "attractor state" (e.g., Conrey & Smith, 2007). Selection is spurred by a cyclical processing loop between higher-order integrative brain regions and lower-level informational sources, in which the former enact representation competition that promotes or inhibits alternatives. Wojnowicz and colleagues (2009) drew on research on racial attitudes (e.g., Fazio et al., 1995) suggesting that negative partial evaluations of black targets would be common early in participants' processing streams but would be subsumed by more positive evaluations due to the motivation to control prejudice. They anticipated that the dynamic evolution of an evaluation would be evident in subtle behavioral traces as participants moved a cursor to associate "black" with "like" or "dislike." Their theory specifically predicted a curvature in trajectory toward the eventually unchosen alternative ("dislike") when expressing liking for the target "black" that would not be evident when the target was "white." This is what they observed.

The IR model (Cunningham & Zelazo, 2007) considers such dynamism central in regulating evaluative responses. As noted, the terms *attitude* and *evaluation* are often used interchangeably, but we believe that it is useful to refer to them as qualitatively different. Whereas an attitude is a relatively stable set of representations of a stimulus, an evaluation reflects one's current appraisal of the stimulus. Evaluative processes transform attitudes into evaluations. In other words, to generate an evaluation, one uses preexisting attitudes to retrieve useful affective information about a stimulus, but the evaluation also takes into consideration information about the environment and context, as well as current goals (see Figure 16.1). Furthermore, because information is distributed, not all aspects of the attitude are activated; as such, only the currently active aspects of attitude can shape an evaluation. Critically, because active representations shape perception and construal of a situation, and because categories and construals shape which attitude aspects are foregrounded and made more active, stimuli initiate an iterative sequence of evaluative processes (the evaluative cycle) through which the stimuli are interpreted and reinterpreted in light of an increasingly rich set of contextually meaningful representations. Such a view blurs the traditional distinctions found in dual-systems or dual-process models of attitudes and attitude regulation. Indeed, although evaluations that are based on few iterations of the evaluative cycle may be thought to be relatively automatic or implicit, they can be shaped by higher-order processes before they are ever encountered (Cunningham, Van Bavel, Arbuckle, Packer, & Waggoner, 2012; Todd et al., 2012). Furthermore, although evaluations that are based on additional iterations may become more integrated, they do not necessarily require or use new representations to generate ongoing evaluations.

Given the iterative nature of the model, evaluations are proposed to be the dynamic result of an integrated set of distributed processes, each of which responds to and resolves specific computational problems (see Cunningham & Johnson, 2007). Evaluation is an emergent property of multiple processes that unfold over time. Critically, the model proposes that a common set of processes is consistently involved in generating current evaluations that are shaped by both bottom-up and top-down influences. Thus, evaluative processes are part of an iterative cycle: With every iteration, the current evaluation of a stimulus is adjusted in light of additional contextual and motivational information in order to create an updated evaluation. Information is continually passed back from relatively higher-order to relatively lower-order processes, and the evaluation is recalculated. This "reseeding" of information allows for the foregrounding of relevant (and backgrounding of irrelevant) attitude representations and contextual information in order to develop incorporate current goals and standards, and allows for the regulation of an evaluation response to come into line with situational or motivational constraints. At each iteration, the current evaluation serves as input for ongoing evaluative processing; as such, earlier evaluations are likely to bias subsequent evaluative processing by

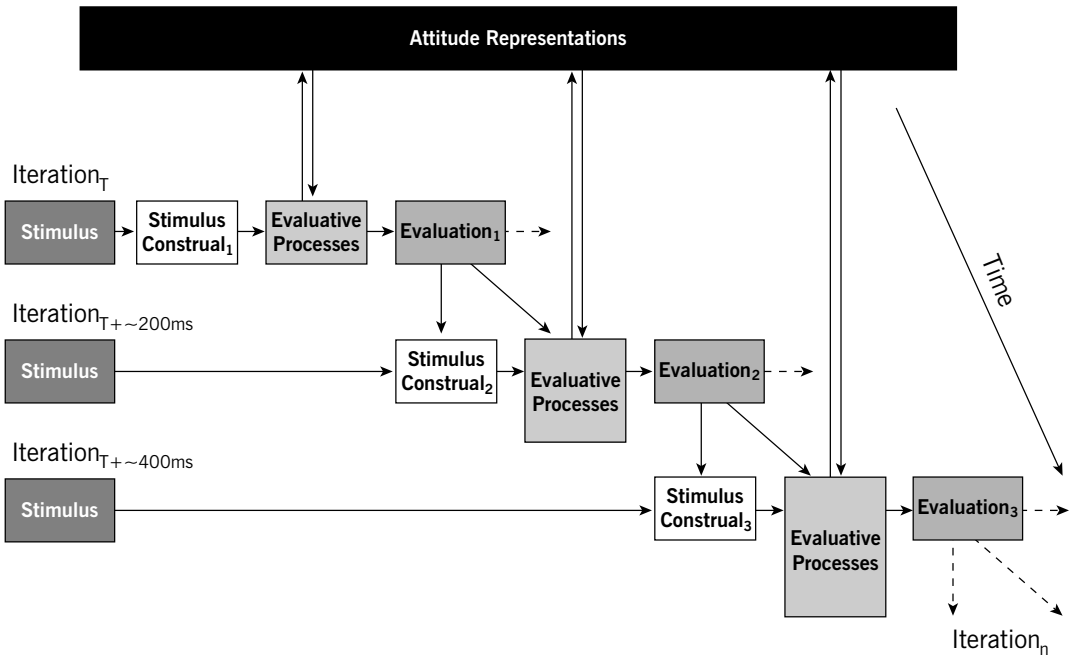

FIGURE 16.1. The iterative reprocessing model. From Cunningham, Zelazo, Packer, and Van Bavel (2007). Copyright 2007 by The Guilford Press. Reprinted by permission.

influencing attention, information seeking, stimulus construal, and so forth. Importantly, while conscious deliberation exerts an influence on evaluative processing, information about the valence and arousal value of a stimulus continues to be represented in subcortical structures.

To understand when people are more likely to generate complex evaluations, the IR model proposes that two competing motivational drives influence the extent of evaluative processing. First, a drive to minimize the discrepancy between one's evaluation and the hedonic environment (i.e., to minimize error) increases reflective processing during evaluation. Second, a drive to minimize processing demands decreases in reflective processing during evaluation. These opposing drives create a dynamic tension that can help individuals to strike a delicate balance between an initial "gut" response and evaluations that are more nuanced but not computationally overwhelming. The influence of these competing motivations likely varies as a function of situational demands, current goals, and individual differences in processing style.

Thus, unlike models that propose a stark difference between automatic and controlled processes, the IR model suggests a continuum from relatively automatic to controlled evaluative processes that can operate on a set of representations. At the early stages of processing, the strongest weights associated with an attitude give rise to a specific pattern of activation, and result in quick and automatic evaluations. However, with more iterations and the potential for reflective processing in areas of the brain responsible for higher processing (e.g., prefrontal cortex), evaluations are shaped by a dynamic interaction of several bottom-up and top-down processes. This interaction allows for the foregrounding and backgrounding of particular patterns of activation in accordance with current contexts and goals. Evaluations based on additional iterations are generally more reflective. As individuals engage in reflective reprocessing, they are able to formulate more complex, nuanced representations of a stimulus (e.g., allowing a stimulus to be understood in terms of multiple conflicting dimensions of evaluation). This hierarchical approach, which views reflection as a matter

of degree, is consistent with contemporary characterizations of prefrontal cortical function (Bunge & Zelazo, 2006).

But although it is possible to draw parallels between standard dual-process models and the IR model, the nature of the relationship between what can be considered automatic and controlled differs in important ways. The IR model proposes that what is typically considered more controlled or reflective processing in dual-process models merely biases which representations remain active and the complexity of the evaluation that is possible given this set of active representations. Thus, the biasing of representations can come prior to processing any given stimulus, allowing for automatic regulation—the initial representation, categorization, and evaluation of an object can be modified at the earliest levels of processing. Indeed, attention to one category (race) or another (age) for stimuli that can be multiply categorized leads to greater affective priming to the focal category (Gawronski, Cunningham, LeBel, & Deutsch, 2010). That is, foregrounding one category or the other changed the initial evaluation as measured by affective priming.

Linking the Literatures on Attitudes, Evaluation, and Emotion Regulation

In this chapter, we suggest that the processes by which attitudes are transformed into evaluations may be more dynamic that previously considered. As such, the classical distinctions between automatic/implicit and controlled/explicit processes may need to be modified to articulate the dynamic and iterative nature of evaluative processes. In doing so, many parallels with the literature on affect and its regulation are noted. For example, to the extent that evaluations are constantly updated, and regulation (defined as biasing representations) exists throughout the process, this suggests that the ways that we shape affect will be important for the types of representations that are active, and how we use them to generate appropriate evaluations. For example, the processes of appraisal and reappraisal will be essential for determining the meaning of an object in a situation, and determining its value. To the extent that one is a successful dieter, by reappraising a donut as an efficient calorie delivery system rather than a tasty snack, the nature of active representations will be more negative, and a more negative evaluation will be constructed. This process is central to emotion regulation, because changing the value of the goal state or the experienced outcome is the most effective means to feel better (e.g., "I didn't actually want that donut, because it is bad for me"). Furthermore, attitudes and their transformation into information are essential for all stages of emotion regulation, from situation selection (one can use attitudes to determine whether a situation is likely to be good or bad) to attention deployment (attitudes guide attention; Roskos-Ewoldsen & Fazio, 1992). Attitudes provide the expectations for a stimulus, and this information is critical to guiding responses that lead to good outcomes and more positive emotions.

Conclusion

We hope the reader will find conceptual parallels within the models reviewed and emotion regulation. Many of the automatic influences of goals and standards on automatic evaluation, and the means and eliciting circumstances of effortful self-regulation of attitudes and evaluation, are likely relevant to the regulation of emotion. Unfortunately, these literatures have as yet seen little integration despite their potential to inform one another. For example, attitude theory has often neglected the role of arousal, which has been integral to the study of emotion. We end with a final note on the applicability of models of evaluation for emotion regulation. Although typically studied in isolation from one another, the processes involved in attitudes greatly overlap with those involved in emotion regulation. Indeed, as suggested in this chapter, the critical question concerning how one converts attitudes (stored representations about the world) into current evaluations (temporary affective experiences that can be used to drive thoughts and behavior) can be viewed fundamentally as a regulatory process in which an evoked affective response is shaped to reflect the exigencies of the moment.

This view goes beyond more traditional notions of regulation, which typically involve

the inhibition or alteration of an automatic response, dependent upon conscious will and ability. We argue that the translation of representation (attitude) to experience (evaluation) involves regulation, as active representations are shaped and reshaped across multiple levels of organization. At lower levels, the perceptual system helps to regulate what stimuli to attend to and what stimuli to ignore by biasing attention, while at higher levels, the more reflective regulatory systems make use of similar foregrounding and backgrounding processes. As such, the ability to regulate one's emotions effectively has been linked to the ways in which these lower- and higher-order processes interact (Lee, Heller, van Reekum, Nelson, & Davidson, 2012). Critically, the ability to reinterpret or reconstrue incoming information flexibly—or cognitive flexibility (Scott, 1962)—is an essential feature of these models and may be vital for emotion regulation. Although dynamic perspectives on attitudes are only beginning to receive attention, it is our hope that these perspectives will be influential in guiding future research beyond the scope of attitudes alone. After all, these models, at their core, are models of cognitive processing, and as such are relevant to and overlap with many domains, emotion regulation included.

Notes

1. This basic distinction between associative and propositional processes is shared with Strack and Deutsch's (2004) reflexive–impulsive model (RIM), which in turn drew from multiple earlier dual-process theories (see Smith & DeCoster, 2000, for a review). However, the APE model focuses on evaluation per se, whereas the RIM is explicitly a model of behavior.
2. It should be noted that some negations are familiar ("no way") and/or very easily reversed ("not good"), such that their appropriate meaning may be encoded even when cognitive resources are strained (e.g., Mayo, Schul, & Burnstein, 2004), rendering ironic effects unlikely in these cases.
3. However, its authors express skepticism that a strong dual-systems approach is consistent with emerging evidence from neuroscience.

References

Allport, G. W. (1935). Attitudes. In C. Murchison (Ed.), *Handbook of social psychology*. Worcester, MA: Clark University Press.

Barden, J., Maddux, W. W., Petty, R. E., & Brewer, M. B. (2004). Contextual moderation of racial bias: The impact of social roles on controlled and automatically activated attitudes. *Journal of Personality and Social Psychology, 87*, 5–22.

Bargh, J. A. (1994). The four horsemen of automaticity: Awareness, intention, efficiency, and control in social cognition. In R. S. Wyer, Jr. & T. K. Srull (Eds.), *Handbook of social cognition* (Vol. 1, 2nd ed., pp. 1–40). Hillsdale, NJ: Erlbaum.

Bargh, J. A. (1999). The cognitive monster: The case against controllability of automatic stereotyping effects. In S. Chaiken & Y. Trope (Eds.), *Dual process theories in social psychology* (pp. 361–382). New York: Guilford Press.

Bunge, S. A., & Zelazo, P. D. (2006). A brain-based account of the development of rule use in childhood. *Current Directions in Psychological Science, 15*, 118–121.

Cacioppo, J. T., Gardner, W. L., & Berntson, G. G. (1997). Beyond bipolar conceptualizations and measures: The case of attitudes and evaluative space. *Personality and Social Psychology Review, 1*, 3–25.

Carver, C. S., & Scheier, M. F. (1982). Control theory: A useful conceptual framework for personality—Social, clinical, and health psychology. *Psychological Bulletin, 92*, 111–135.

Conrey, F. R., & Smith, E. R. (2007). Attitude representation: Attitudes as patterns in a distributed, connectionist representational system. *Social Cognition, 25*, 718–735.

Crandall, C. S., Eshleman, A., & O'Brien, L. (2002). Social norms and the expression and suppression of prejudice: The struggle for internalization. *Journal of Personality and Social Psychology, 82*, 359–378.

Crone, E. A., & Van der Molen, M. W. (2004). Developmental changes in real-life decision-making: Performance on a gambling task previously shown to depend on the ventromedial prefrontal cortex. *Developmental Neuropsychology, 25*, 251–279.

Cunningham, W. A., & Johnson, M. K. (2007). Attitudes and evaluation: Toward a component process framework. In E. Harmon-Jones & P.

Winkielman (Eds.), *Social neuroscience: Integrating biological and psychological explanations of social behavior* (pp. 227–245). New York: Guilford Press.

Cunningham, W. A., Johnson, M. K., Raye, C. L., Gatenby, J. C., Gore, J. C., & Banaji, M. R. (2004). Separable neural components in the processing of Black and White faces. *Psychological Science, 15*, 806–813.

Cunningham, W. A., Raye, C. L., & Johnson, M. K. (2004). Implicit and explicit evaluation: fMRI correlates of valence, emotional intensity, and control in the processing of attitudes. *Journal of Cognitive Neuroscience, 16*, 1717–1729.

Cunningham, W. A., Van Bavel, J. J., Arbuckle, N. L., Packer, D. J., & Waggoner, A. S. (2012). Rapid social perception is flexible: Approach and avoidance motivational states shape P100 responses to other-race faces. *Frontiers in Human Neuroscience, 6*, 140.

Cunningham, W. A., & Zelazo, P. D. (2007). Attitudes and evaluations: A social cognitive neuroscience perspective. *Trends in Cognitive Sciences, 11*, 97–104.

Cunningham, W. A., Zelazo, P. D., Packer, D. J., & Van Bavel, J. J. (2007). The Iterative Reprocessing Model: A multilevel framework for attitudes and evaluation. *Social Cognition, 25*, 736–760.

De Houwer, J., & Moors, A. (2007). How to define and examine the implicitness of implicit measures. In B. Wittenbrink & N. Schwarz (Eds.), *Implicit measures of attitudes: Procedures and controversies* (pp. 179–194). New York: Guilford Press.

De Houwer, J., Thomas, S., & Baeyens, F. (2001). Associative learning of likes and dislikes: A review of 25 years of research on human evaluative conditioning. *Psychological Bulletin, 127*, 853–869.

Deutsch, R., Gawronski, B., & Strack, F. (2006). At the boundaries of automaticity: Negation as reflective operation. *Journal of Personality and Social Psychology, 91*, 385–405.

Devine, P. G. (1989). Stereotypes and prejudice: Their automatic and controlled components. *Journal of Personality and Social Psychology, 56*, 5–18.

Dovidio, J. F., Kawakami, K., Johnson, C., Johnson, B., & Howard, A. (1997). On the nature of prejudice: Automatic and controlled processes. *Journal of Experimental Social Psychology, 33*, 510–540.

Eagly, A. H., & Chaiken, S. (1993). *The psychology of attitudes*. Fort Worth, TX: Harcourt Brace Jovanovich.

Eiser, J. R., Fazio, R. H., Stafford, T., & Prescott, T. J. (2003). Connectionist simulation of attitude learning: Asymmetries in the acquisition of positive and negative evaluations. *Personality and Social Psychology Bulletin, 29*, 1221–1235.

Fazio, R. H. (1990). Multiple processes by which attitudes guide behavior: The MODE model as an integrative framework. In M. P. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 23, pp. 75–109). New York: Academic Press.

Fazio, R. H. (2001). On the automatic activation of associated evaluations: An overview. *Cognition and Emotion, 15*, 115–141.

Fazio, R. H. (2007). Attitudes as object-evaluation associations of varying strength. *Social Cognition, 25*, 603–637.

Fazio, R. H., Jackson, J. R., Dunton, B. C., & Williams, C. J. (1995). Variability in automatic activation as an unobtrusive measure of racial attitudes: A bona fide pipeline? *Journal of Personality and Social Psychology, 69*, 1013–1027.

Festinger, L. (1957). *A theory of cognitive dissonance*. Evanston, IL: Row Peterson.

Freeman, J. B., & Ambady, N. (2009). Motions of the hand expose the partial and parallel activation of stereotypes. *Psychological Science, 20*, 1183–1188.

Freeman, J. B., Ambady, N., Rule, N. O., & Johnson, K. L. (2008). Will a category cue attract you?: Motor output reveals dynamic competition across person construal. *Journal of Experimental Psychology: General, 137*, 673–690.

Fujita, K. (2011). On conceptualizing self-control as more than the effortful inhibition of impulses. *Personality and Social Psychology Review, 15*, 352–366.

Gawronski, B., & Bodenhausen, G. V. (2006). Associative and propositional processes in evaluation: An integrative review of implicit and explicit attitude change. *Psychological Bulletin, 132*, 692–731.

Gawronski, B., & Bodenhausen, G. V. (2011). The associative–propositional evaluation model: Theory, evidence, and open questions. *Advances in Experimental Social Psychology, 44*, 59–127.

Gawronski, B., Cunningham, W. A., Lebel, E. P., & Deutsch, R. (2010). Attentional influences on affective priming: Does categoriza-

tion influence spontaneous evaluations of multiply categorizable objects? *Cognition, 24*, 1008–1026.

Gawronski, B., Deutsch, R., Mbirkou, S., Seibt, B., & Strack, F. (2008). When "just say no" is not enough: Affirmation versus negation training and the reduction of automatic stereotype activation. *Journal of Experimental Social Psychology, 44*, 370–377.

Gawronski, B., & LeBel, E. P. (2008). Understanding patterns of attitude change: When implicit measures show change, but explicit measures do not. *Journal of Experimental Social Psychology, 44*, 1355–1361.

Gawronski, B., & Strack, F. (2004). On the propositional nature of cognitive consistency: Dissonance changes explicit, but not implicit attitudes. *Journal of Experimental Social Psychology, 40*, 535–542.

Gilbert, D. T. (1991). How mental systems believe. *American Psychologist, 46*, 107–119.

Greenwald, A. G., & Banaji, M. R. (1995). Implicit social cognition: Attitudes, self-esteem, and stereotypes. *Psychological Review, 102*, 4–27.

Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. (1998). Measuring individual differences in implicit cognition: The Implicit Association Test. *Journal of Personality and Social Psychology, 74*, 1464–1480.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*, 271–299.

Gross, J. J., & Barrett, L. F. (2011). Emotion generation and emotion regulation: One or two depends on your point of view. *Emotion Review, 3*, 8–16.

Herbert, C., Deutsch, R., Sütterlin, S., Kübler, A., & Pauli, P. (2011). Negation as a means for emotion regulation?: Startle reflex modulation during processing of negated emotional words. *Cognitive, Affective, and Behavioral Neuroscience, 11*, 199–206.

Hofmann, W., Gawronski, B., Gschwendner, T., Le, H., & Schmitt, M. (2005). A meta-analysis on the correlation between the Implicit Association Test and explicit self-report measures. *Personality and Social Psychology Bulletin, 31*, 1369–1385.

Hovland, C. I., Janis, I. L., & Kelley, H. H. (1953). *Communication and persuasion: Psychological studies of opinion change.* New Haven, CT: Yale University Press.

Jones, C. R., & Fazio, R. H. (2010). Person categorization and automatic racial stereotyping effects on weapon identification. *Personality and Social Psychology Bulletin, 36*, 1073–1085.

Jones, C. R., Olson, M. A., & Fazio, R. H. (2010). Evaluative conditioning: The "how" question. In M. P. Zanna & J. M. Olson (Eds.), *Advances in experimental social psychology* (Vol. 43, pp. 205–255). New York: Academic Press.

Karpinski, A., & Hilton, J. L. (2001). Attitudes and the Implicit Association Test. *Journal of Personality and Social Psychology, 81*, 774–788.

Lee, H., Heller, A. S., van Reekum, C. M., Nelson, B., & Davidson, R. J. (2012). Amygdala–prefrontal coupling underlies individual differences in emotion regulation. *NeuroImage, 62*, 1575–1581.

Lieberman, M. D. (2007). Social cognitive neuroscience: A review of core processes. *Annual Review of Psychology, 58*, 259–289.

Lowery, B. S., Hardin, C. D., & Sinclair, S. (2001). Social influence effects on automatic racial prejudice. *Journal of Personality and Social Psychology, 81*, 842–855.

Mauss, I. B., Evers, C., Wilhelm, F. H., & Gross, J. J. (2006). How to bite your tongue without blowing your top: Implicit evaluation of emotion regulation predicts affective responding to anger provocation. *Personality and Social Psychology Bulletin, 32*, 589–602.

Mayo, R., Schul, Y., & Burnstein, E. (2004). "I am not guilty" vs. "I am innocent": Successful negation may depend on the schema used for its encoding. *Journal of Experimental Social Psychology, 40*, 433–449.

McConnell, A. R., Dunn, E. W., Austin, S. N., & Rawn, C. D. (2011). Blind spots in the search for happiness: Implicit attitudes and nonverbal leakage predict affective forecasting errors. *Journal of Experimental Social Psychology, 47*, 628–634.

McConnell, A. R., & Rydell, R. J. (in press). The systems of evaluation model: A dual-systems approach to attitudes. In J. Sherman, B. Gawronski, & Y. Trope (Eds.), *Dual process theories of the social mind*. New York: Guilford Press.

Moskowitz, G. B., Gollwitzer, P. M., Wasel, W., & Schaal, B. (1999). Preconscious control of stereotype activation through chronic egalitarian goals. *Journal of Personality and Social Psychology, 77*, 167–184.

Olson, M. A., & Fazio, R. H. (2009). Implicit and explicit measures of attitudes: The perspective of the MODE model. In R. E. Petty,

R. H. Fazio, & P. Briñol (Eds.), *Insights from the new implicit measures* (pp. 19–63). New York: Psychology Press.

Olson, M. A., Fazio, R. H., & Hermann, A. D. (2007). Reporting tendencies underlie discrepancies between implicit and explicit measures of self-esteem. *Psychological Science, 18*, 287–291.

Payne, B. K., Burkley, M. A., & Stokes, M. B. (2008). Why do implicit and explicit attitude tests diverge?: The role of structural fit. *Journal of Personality and Social Psychology, 94*, 16–31.

Petty, R. E. (2006). A metacognitive model of attitudes. *Journal of Consumer Research, 33*, 22–24.

Petty, R. E., & Briñol, P. (2006). A metacognitive approach to "implicit" and "explicit" evaluations: Comment on Gawronski and Bodenhausen (2006). *Psychological Bulletin, 132*, 740–744.

Petty, R. E., Briñol, P., & DeMarree, K. G. (2007). The Meta-Cognitive Model (MCM) of attitudes: Implications for attitude measurement, change, and strength. *Social Cognition, 25*, 657–686.

Petty, R. E., Tormala, Z. L., Briñol, P., & Jarvis, W. B. G. (2006). Implicit ambivalence from attitude change: An exploration of the PAST model. *Journal of Personality and Social Psychology, 90*, 21–41.

Roskos-Ewoldsen, D. R., & Fazio, R. H. (1992). On the orienting value of attitudes: Attitude accessibility as a determinant of an object's attraction of visual attention. *Journal of Personality and Social Psychology, 63*, 198–211.

Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review, 110*, 145–172.

Rydell, R. J., & Durso, G. R. O. (2012). Can I borrow a feeling?: Spillover of negative arousal from inconsistent information during attitude formation diminishes perceptions of well-being. *Journal of Experimental Social Psychology, 48*, 575–578.

Rydell, R. J., & McConnell, A. R. (2006). Understanding implicit and explicit attitude change: A systems of reasoning analysis. *Journal of Personality and Social Psychology, 91*, 995–1008.

Rydell, R. J., McConnell, A. R., & Mackie, D. (2008). Consequences of discrepant explicit and implicit attitudes: Cognitive dissonance and increased information processing. *Journal of Experimental Social Psychology, 44*, 1526–1532.

Rydell, R. J., McConnell, A. R., Mackie, D. M., & Strain, L. M. (2006). Of two minds: Forming and changing valence-inconsistent implicit and explicit attitudes. *Psychological Science, 17*, 954–959.

Sassenberg, K., & Moskowitz, G. B. (2005). Don't stereotype, think different!: Overcoming automatic stereotype activation by mindset priming. *Journal of Experimental Social Psychology, 41*, 506–514.

Scott, W. A. (1962). Cognitive complexity and cognitive flexibility. *Sociometry, 25*, 405–414.

Schwarz, N., & Bohner, G. (2001). The construction of attitudes. In A. Tesser & N. Schwarz (Eds.), *Blackwell handbook of social psychology: Intraindividual processes* (pp. 436–457). Oxford, UK: Blackwell.

Sloman, S. A. (1996). The empirical case for two systems of reasoning. *Psychological Bulletin, 119*, 3–22.

Smith, E. R., & DeCoster, J. (2000). Dual process models in social and cognitive psychology: Conceptual integration and links to underlying memory systems. *Personality and Social Psychology Review, 4*, 108–131.

Spivey, M. J. (2007). *The continuity of mind*. Oxford, UK: Oxford University Press.

Strack, F., & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior. *Personality and Social Psychology Review, 8*, 220–247.

Todd, R. M., Cunningham, W. A., Anderson, A. K., & Thompson, E. (2012). Affect-biased attention as emotion regulation. *Trends in Cognitive Sciences, 16*, 365–372.

Watson, D., & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological Bulletin, 98*, 219–235.

Wilson, T. D., Lindsey, S., & Schooler, T. Y. (2000). A model of dual attitudes. *Psychological Review, 107*, 101–126.

Wojnowicz, M. T., Ferguson, M. J., Dale, R., & Spivey, M. J. (2009). The self-organization of explicit attitudes. *Psychological Science, 20*, 1428–1435.

Zanna, M. P., & Rempel, J. K. (1988). Attitudes: A new look at an old concept. In D. Bar-Tal & A. Kruglanski (Eds.), *The social psychology of knowledge* (pp. 315–334). New York: Cambridge University Press.

CHAPTER 17

Emotion Regulation in Couples

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Whereas research on emotion regulation in individuals has been extremely useful, there are numerous advantages to studying emotion regulation in couples. Couples afford high ecological validity, provide opportunities to view the rich panoply of emotion regulatory strategies, and are ideal for studying the dynamics of emotion regulation as partners engage in a rich choreography of emotional expression and regulation that unfolds in complex ways over time.

In this chapter, we consider several important aspects of emotion regulation in couples, discussing its social nature, defining qualities, consequences, development, and assessment. We end by considering the future of this research area, including unmet needs, unfilled gaps, and unanswered research questions. Our aim in this review of the relevant literatures is not intended to be exhaustive but rather to highlight key studies that illustrate important issues. We also draw anecdotally from our experiences with couples who seek therapy for troubled relationships (Levenson, Cowan, & Cowan, 2010). These couples almost always have problems with emotion regulation.

The Social Nature of Emotion Regulation

Research on emotion regulation in couples should be booming. After all, most human emotions occur in decidedly social situations (Campos, Walle, Dahl, & Main, 2011). Other people are deeply entwined in the fabric of our emotional lives. They are the proximal stimulus for most of our emotions and the recipients of most of the emotions we express. When others respond to our emotions with their own emotional reactions, they provide the fuel needed to sustain the chains of exchanged emotions that are so emblematic of our social lives.

With so much of human emotion being socially situated, it is reasonable to expect that most emotion regulation would be similarly social. In fact, it has been observed that up to 98% of emotion regulation episodes

may take place in social contexts (Gross, Richards, & John, 2006). Viewed from a functionalist perspective, emotion regulation is a critical element for promoting social cohesion. If we were to discharge our emotions upon others, full-bore, undiluted, and absent the moderating influence of emotion regulation, the resultant affective tsunami would have dire consequences for us and for our social groupings.

Given its profoundly social nature, we might expect that studies of emotion regulation would almost always be conducted in social situations. However, this could not be further from the truth. In a recent review of studies since 2001 (Campos et al., 2011), less than 12% of the studies assessed emotion regulation in the presence of another person (and this is an optimistic estimate that includes studies using both imagined and real others). Clearly, studies of emotion regulation in individuals have advantages over those conducted with couples. They are easier to administer and more amenable to tight experimental control. However, they are not optimal for studying the dynamic, interpersonal aspects of emotion regulation. Although most theorists would agree that emotion regulation is profoundly social in nature and is a fundamental area of concern in close relationships, the literature on emotion regulation in couples is still surprisingly immature, with many gaps and unanswered questions.

Before moving forward with our consideration of emotion regulation in couples in intimate relationships we should note that they are only one of many kinds of dyads for whom emotion regulation is critical (e.g., new acquaintances, friends, enemies). Moreover, emotion regulation in social contexts scales up from dyads through families, groups, communities, and nations. Emotion regulation in these larger social groupings is fascinating and instructive (e.g., communities and nations coping with fear, anger, and grief following collective losses, seen quite famously in the U.S. and world responses to the September 11, 2001, terrorist attacks). So, why focus on couples in intimate relationships? Dyads are the smallest social unit, thus providing an excellent starting point for building a science of socially embedded emotion regulation. Intimate relationships assume a central role in the lives of most individuals. For example, according to the 2009 census, 96% of all Americans over age 65 have been married at least once.

Defining Qualities

There are many ways to define emotion regulation. Because most theory and research in the field of emotion regulation have focused on individuals, popular definitions of emotion regulation reflect this bias. For example, Gross (1998b, p. 275) defines *emotion regulation* as "the processes by which *individuals* [emphasis added] influence which emotions they have, when they have them, and how they experience and express these emotions." Of course, a definition of this sort could be altered so that "individuals" becomes "individuals and couples." But does this cover all bases? The answer to this question depends on whether we think that emotion regulation in couples can be fully captured by summing the regulatory activities of the two individuals involved, or whether there are emergent qualities of emotion regulation that are found only in the couples context. Clearly there are aspects of emotion regulation that are common to both individuals and couples. For example, emotion regulation in both can be *explicit* (i.e., effortful) or *implicit* (i.e., automatic) (Gyurak, Gross, & Etkin, 2011), and successful or unsuccessful (Gross & Levenson, 1993). And in both contexts it can be difficult to determine where emotional reactivity ends and emotion regulation begins (Gross, Sheppes, & Urry, 2011). Nonetheless, as we hope the following sections illustrate, emotion regulation in couples has a number of characteristics that are quite different from those found in individuals.

Dynamic and Iterative

Consider two emotion regulatory scenarios. In the first, a recent PhD recipient is preparing to give an important job talk and is in the throes of a bout of stage fright. Fear is the dominant emotion welling up. He is concerned that the intensity of his fear, should it reach sufficiently high levels, will compromise the quality of the talk. The standard-issue emotion regulatory toolbox offers a number of strategies, including altering the

context, reappraising the situation, or willfully controlling aspects of the emotional response. The speaker chooses one (or more) of these strategies, applies them in the moment, successfully down-regulates the fear, gives an excellent talk, and averts the crisis.

Now, consider a second scenario involving emotion regulation in couples. A married couple is talking about the husband's pending surgery, and he is clearly experiencing high levels of fear. In that moment he wants calming support from his wife. But she is reacting with a great deal of sadness, talking about her concerns that he might die and that their children would go through life without a father. Getting from this starting point to a place where both husband and wife are feeling less distressed is not as straightforward as the situation in the first scenario, in large part because there are two actors involved. Both individuals have to react to their own *and* their partner's emotional state, the impact of each partner's regulatory attempts (some well-chosen, others misguided), and the unfolding sequence of action and reaction that will occur over time as the couple works toward achieving a state that is more emotionally optimal for both partners.

The second scenario illustrates some of the ways that emotion regulation in couples differs from emotion regulation in individuals. In regulation in couples there are always two actors and reactors, each with his or her own emotional motivations, goals, strengths, blind spots, and hot buttons. Couples often find themselves in a complex emotional landscape that changes continuously as partners express and regulate their own emotions, respond to each other's emotions and regulatory attempts, and try to regulate each other's emotions. This extremely fluid situation, replete with highly dynamic and iterative sequences of emotion, creates an extremely challenging, complex landscape for emotion regulation, one that is quite different from that faced by individuals.

Bidirectional

Although most discussions of emotion regulation in individuals allow for both up-regulation and down-regulation of emotion, the emphasis most often seems to be on *reducing* emotional responses. In the canonical examples, individuals seek to reduce their negative emotions to avoid harmful consequences for self and others. Consistent with this, our own early experimental studies of emotion regulation focused exclusively on the consequences of having subjects reduce their behavioral responses to emotion-inducing films (Gross & Levenson, 1993). It was only later that we began to consider up-regulation of emotion in these kinds of studies as well (Kunzmann, Kupperbusch, & Levenson, 2005). In recent years, especially as researchers have explored the neural bases of emotion regulation using patient and neuroimaging models, studies including both up-regulation and down-regulation have become more common (e.g., Gyurak, Goodkind, Kramer, Miller, & Levenson, 2012; Ochsner et al., 2004). However, it is still the case that when studying emotion regulation in individuals, the primary focus is on reducing emotion.

In the realm of emotion regulation in couples, this emphasis on down-regulation is less appropriate. There are many times when couples need to amplify the magnitude of emotion so that it emerges more clearly against the backdrop of other aspects of their interaction. This amplification takes many forms, ranging from the exaggerated tonality of emotional speech patterns used by mothers when they communicate with and soothe their infants (Fernald, 1991) to the stylized and exaggerated expressions of love and affection that are so important in courtship rituals. Interestingly, among those who seek couple therapy, it is extremely common for one partner (usually the woman in heterosexual couples) to desire the other partner (usually the man in these couples) to up-regulate emotion (i.e., expressing emotion more often and more clearly).

Bivalent

The prototypical examples of emotion regulation in individuals all focus on negative emotions. This is seen in common parlance (e.g., "Don't let them see you sweat" [fear], "Don't let it get to you" [anger], "Grown men don't cry" [sadness]); in many adolescent rituals in which a calm demeanor is maintained while viewing, touching, or ingesting extremely gross things (disgust);

and even in therapeutic contexts (where cathartic release is often sought for pent up anger, fear, and sadness). Consistent with this emphasis, most laboratory research on emotion regulation has focused on controlling negative emotions. Such work is facilitated by eliciting negative emotions such as disgust readily and powerfully, using static images (Lang, Greenwald, & Bradley, 1988) and films (Gross & Levenson, 1995). In addition, although studies comparing the regulation of negative and positive emotions have been rare, these comparisons have often not produced dramatic differences (e.g., Gross & Levenson, 1997).

In couples, however, the regulation of positive emotion is at least as important as the regulation of negative emotion. Upregulating positive emotion is critical for building and maintaining intimate relationships throughout the lifespan. This is seen in parent–infant and parent–child relationships, and continues with childhood friendships, early romantic relationships, mate selection, and long-term committed relationships (Carstensen, Graff, Levenson, & Gottman, 1996). Similarly, down-regulating positive emotion also plays an important role for couples. For example, failure to downregulate amusement in response to a partner's failures and insecurities can be critical. In these contexts, laughing, aggressive teasing, and unrelenting, unsupportive humor can be experienced as cruel and demeaning, thus serving to undermine relationship quality (see Martin, 2007).

Coregulatory

Most of us find managing our own emotions sufficiently challenging to occupy significant segments of our waking (and sleeping) hours. This challenge increases dramatically when we take on the additional responsibility of attempting to manage the emotions of another person. Whether the interaction partner is an infant, a friend, a romantic love interest, or a partner in a long-term relationship, moving the focus of emotion regulation from "my emotions" to "your and our emotions" takes us into new and complex realms. A recurrent theme in studies of optimal performance is that individuals function optimally when they are somewhat, but not overly, aroused (Yerkes & Dodson, 1908). We expect that the same is true of couples. Maintaining an optimal level of emotional arousal for couples, however, requires monitoring and regulating the emotional state of both partners. The dynamic and iterative nature of emotion exchanges in couples means that the level of emotional arousal is constantly changing. Thus, the maintenance of an optimal state requires continuous monitoring of arousal levels and continuous adjustment of regulatory efforts. This situation has caused us on multiple occasions to observe that a good marriage requires a good thermostat (a role most often assumed by wives in heterosexual couples; Gottman & Levenson, 1988).

Further complicating matters, in couples, one partner's regulatory efforts often become potent emotional stimuli for the other partner. Consider this example: A husband has been trying to soothe his wife's anger over a canceled vacation, hoping that when she is calmed down they will be able to discuss alternative plans. At this point in the interaction, the wife is overly aroused and the husband is relatively calm. The wife says, "I hate it when you try to manage me. It's insulting and belittling. You're just so incredibly selfish; if you really loved me, you wouldn't do this to me." Hearing this, the husband feels unjustly judged and injured. His anger starts welling up and he becomes quite defensive. Suddenly, they are *both* overly aroused and find themselves casting hurtful aspersions about each other's character defects. Any possibility of having a constructive discussion about vacation plans will soon be placed on indefinite hold.

Coregulation in couples would be challenging enough if both partners always had the same regulatory goals (e.g., both wanting to feel less aroused and more calm, or both wanting to intensify feelings of passionate love). However, the emotional stars are not always so well-aligned. The "demand–withdraw" pattern, commonly found in both opposite sex (Christensen, 1987) and same-sex (Holley, Sturm, & Levenson, 2010) couples, provides a good example. A couple wants to talk effectively about a significant relationship issue. However, one partner (typically the one who wants change) becomes quite aroused and engages energetically in complaining and criticizing the other partner. The criticized

partner (typically the one who wants to maintain the status quo) increasingly tunes out and withdraws emotionally from the interaction, thus achieving some level of calm. For this couple, the coregulatory challenge is to calm the overaroused, demanding partner and at the same time increase the emotional involvement of the underaroused, withdrawing partner. However, this must be done without causing either partner to overshoot the desired emotional endpoint (no easy feat). Coregulation of emotion in couples regularly introduces these kinds of complexities, which are simply not found in individual emotion regulation.

Consequences

It is easy to make a case for the importance of emotion regulation in the lives of individuals. Greater use of specific emotion regulation strategies, as measured by self- and other-reports, has been found to predict higher levels of well-being, mental health, physical health, relationship quality, and social functioning, and lower levels of problem behavior (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Caspi, Henry, McGee, Moffitt, & Silva, 1995; Gross & John, 2003; John & Gross, 2004; Lopes, Salovey, Côté, Beers, & Petty, 2005; Nelis et al., 2011). Although studies of these associations using laboratory assessments of emotion regulation (as opposed to self- and other-reports) are still rare, we recently found that greater ability to down-regulate and up-regulate emotional response (as assessed using well-established laboratory procedures) was associated with greater well-being and higher income (Côté, Gyurak, & Levenson, 2010). If emotion regulation is broadened to include delay of gratification, another laboratory-based paradigm, research strongly indicates that high levels of this ability early in preschool years are associated with a host of positive outcomes, including greater cognitive and academic competence and ability to cope with frustration later in life (Mischel et al., 2011).

Because of a relative dearth of studies that have directly measured emotion regulation in couples and its consequences, the case for the importance of emotion regulation in couples must be based on collateral literatures. One such literature, concerned with marriage and other committed relationships, has produced a number of findings that suggest greater ability to regulate emotion in couples is associated with positive outcomes. For example, questionnaire studies have found that couples who report less frequent use of "control or containment" (which is similar to suppression) of negative emotion have higher marital satisfaction (Feeney, 1999). In the literature on intimate partner violence, inability to regulate negative emotion has been associated with increased likelihood of partner abuse (McNulty & Hellmuth, 2008).

A paradigm for studying couples' interaction developed by Levenson and Gottman (1983) has been endorsed as an exemplar for how emotion regulation can be studied in social contexts (Campos et al., 2011). In this paradigm, couples (usually married heterosexual couples, but also same-sex and dating couples) come to the laboratory and engage in a series of unrehearsed 15-minute conversations on relationship topics (e.g., events of the day, a problem area, a pleasant topic). During these conversations, behavior is videotaped for subsequent coding of emotional behavior by trained observers, and in both partners a number of physiological measures relevant to emotional responding are monitored continuously. Later, partners view the videotapes of their conversations and use a rating dial to provide continuous ratings of the valence of their emotional experience during the interactions (Gottman & Levenson, 1985). These streams of continuous multimethod data (self-report, behavior, physiology) can be used to derive measures of emotion reactivity and emotion regulation in the individual partners and in the dyad.

A number of findings from these studies seem highly relevant when considering the consequences of emotion regulation for couples. In this regard, the discussions about marital problems (which can occasion intense negative emotions and heroic efforts at emotion regulation) have been particularly informative, with links found between measures of emotion regulation (in subjective experience, behavior, and physiology) and important consequences in several domains.

In terms of subjective emotional experience, low levels of both negative emotion

and negative emotion reciprocity (i.e., negative emotional experience by one partner followed by negative emotional experience by the other partner) have been associated with higher levels of marital satisfaction both concurrently and over time (Levenson & Gottman, 1983, 1985). In terms of emotional behavior, husbands' inability to deescalate negative emotion during marital conflict predicted less marital stability over time (Gottman, Coan, Carrere, & Swanson, 1998). In a similar vein, "regulated" couples, operationalized as those who produced an increasingly high ratio of positive to negative emotional behaviors over the course of a 15-minute conflictive interaction, had higher levels of marital satisfaction, lower risk for marital dissolution, and better health measured over a 4-year period (Gottman & Levenson, 1992). Finally, less escalation of negative emotional behavior was associated with higher marital satisfaction in couples in long-term marriages (Carstensen, Gottman, & Levenson, 1995).

The continuous measures of peripheral physiological activity have been particularly interesting, in part because autonomic nervous system responses are very difficult to control voluntarily (e.g., Levenson, 1976) and because they can provide a ready metric of changing levels of arousal in the couple. Using these measures, low levels of both physiological arousal and *physiological linkage* (synchrony between partners' physiology) were associated with higher marital satisfaction (Levenson & Gottman, 1983, 1985).

Couple Therapy

Not surprisingly, given its critical role in couple relationships and profound downstream consequences, issues with emotion regulation frequently assume center stage when couples seek treatment for relationship problems. Although the specifics differ from couple to couple, distressed couples almost always struggle with either down-regulating negative emotion (e.g., issues involving jealousy and disagreements over things such as household duties, relatives, child rearing, and finances) or up-regulating positive emotions (e.g., issues involving poor and infrequent communication, not doing things together, loss of sexual interest and intimacy, coldness and lack of empathy, and absence of joy) or both. Historically, couples therapies have focused more on nonemotional aspects of these problems (e.g., communication deficits, individual psychopathology, attachment histories, poor family-of-origin relationship models), but emotion and emotion regulation are increasingly becoming important foci in many forms of couple therapy (Gottman & Gottman, 2008; Johnson, 1996; Wile, 2002).

Development

Viewed from a developmental perspective, several key dyadic relationships assume prominence at different stages of the lifespan and serve as crucibles for the emergence and refinement of regulatory skills. Here we focus on three of these dyads: parents and infants, early romantic relationships, and late-life couples.

Parent–Infant Dyads

Parent–infant dyads, and mother–infant dyads in particular, invest significant efforts in emotion regulation. The initial focus is on reducing negative emotion (managing the infant's distress), but this quickly engenders efforts to increase positive emotion (engaging in activities that amuse, distract, and calm). In this stage of life, infants can become overwhelmed by their negative emotions and lack the skills to bring them under control themselves. Thus, infants rely on their caregivers to regulate their emotions (Thompson, 1991). The ontological origins of social emotion regulation clearly reside in these dyads. If all goes well, coregulation of emotion in the parent–infant dyad will lead to infants beginning to develop the ability to regulate their own emotions.

Attachment theory (Bowlby, 1988) provides the most influential account of the transition from coregulation to individual regulation in infancy, providing elegant descriptions about the kinds of parent–infant relationships that are likely to result in good versus poor emotion regulation in the infant. Attachment theory was given an enormous empirical boost with the development of observational methods for quantifying mother–infant attachment. For example,

the "Strange Situation" paradigm (Ainsworth, Blehar, Waters, & Wall, 1978) uses close observation of episodes of mother–infant separation and reunion to classify attachment styles in ways that have profound implications for emotion regulation initially in the dyad and ultimately in the infant (e.g., securely attached infants develop greater ability to regulate their own emotions than do insecurely attached infants).

The Strange Situation focuses on the down-regulation of negative emotions (primarily fear and sadness), but the mother–infant dyad also is involved in a great deal of coregulation of positive emotion. The social nature of positive emotion regulation in infancy is seen vividly in studies of the synchrony and reciprocity of positive emotion (Cole, Teti, & Zahn-Waxler, 2003; Tronick, 1989). In addition, parent–infant dyads often engage in elaborate behavioral rituals designed to increase positive emotions in the infant (e.g., tickling and peekaboo rituals). For parents, the infant's smile is a highly prized reinforcer; parents will go to great lengths to evoke smiles in their infants.

We include discussion of these early parent–infant dyads because we believe they have important implications for emotion regulation and other aspects of later intimate relationships. However, one enduring question about attachment styles (and associated emotion regulatory abilities) is how parent–infant interactions are related to attachment styles that characterize intimate relationships later in life (Mikulincer & Shaver, 2007). In this regard, some theorists have emphasized discontinuity (e.g., Kagan, 1984), while others have leaned more toward continuity (e.g., Bowlby, 1988). Conducting empirical research on these issues is quite challenging. One approach has been to utilize chain mediation models, in which experiences during one life stage predict experiences in the next, which in turn predict experiences in the next (Sroufe, Coffino, & Carlson, 2010). In one such study, which bridged infancy, adolescence, and adult relationships, secure attachment in infancy was found to predict more secure relationships with close friends in adolescence, which in turn predicted more positive daily emotional experiences in adult romantic relationships and less negative affect in conflict resolution (Simpson, Collins, Tran, & Haydon, 2007).

Early Romantic Dyads

Selecting a partner and building a romantic bond are critical developmental tasks. Although often viewed through a lens that emphasizes mate selection and family building (Havighurst, 1976), these relationships are also important vehicles for developing emotion regulatory skills. In contrast to parent–infant dyads, where the primary focus is on mastering down-regulation of negative emotions, in early romantic relationships the primary focus is clearly on up-regulating positive emotions. The emotions that are typically targets for this up-regulation include passionate love, affection, joy, excitement, and enthusiasm (Gable, Gonzaga, & Strachman, 2006).

Several lines of research highlight the importance of positive emotions in early romantic relationships. Romantic love is a phenomenon found across many cultures (Jankowiak & Fischer, 1998) and has been termed a "mammalian system for mate choice" (Fisher, Aron, & Brown, 2006). It is associated with a complex physiological, psychological, and behavioral profile that includes feelings of euphoria, focused attention, and obsessive thinking about a specific individual; craving for emotional connection with the other person; expanded sense of self; and greatly increased energy (A. Aron et al., 2005; E. N. Aron & Aron, 1996). There is good evidence linking the early stages of intense passionate love with subcortical reward and goal centers in the brain that are highly responsive to dopamine (A. Aron et al., 2005; Fisher, Aron, & Brown, 2006), which may help explain the almost addictive quality of passionate love, along with its attendant cravings (e.g., intensely missing the partner when absent) and powerful withdrawal reactions (e.g., the pain of lost love). From an emotion regulatory point of view, these high-intensity positive emotional states are highly desirable, and, not surprisingly, lovers engage in quite elaborate strategies to up-regulate their positive feelings to extremely high levels of intensity. There is little doubt that these intense feelings contribute significantly to mate selection and reproduction. In many ways, they are the perfect fuel for launching romantic dyads along the path to family formation.

Clearly, down-regulating negative emotion is an important item on the dyadic agenda at all stages of development. This often takes the form of managing jealousy (Shaver & Mikulincer, 2007) in early romantic relationships, in which real and imagined infidelities are a source of powerful negative emotions (fear, sadness, anger) that must be controlled if the relationship is to survive. Emotion regulation continues to play a critical role as romantic relations grow and develop. Looming large is the transition to parenthood, which is highly challenging for most couples (Cowan & Cowan, 1992; Doss, Rhoades, Stanley, & Markman, 2009). Successful navigation of this transition requires couples to deploy the full range of individual, dyadic, and, ultimately, triadic emotion regulatory skills.

Late-Life Dyads

In late life, as couples move beyond the prime reproductive period, life challenges and life goals change, and emotion regulatory needs change accordingly. During this developmental period, dealing with losses and finding meaning in life become particularly salient (Erikson, Erikson, & Kivnick, 1986). As individuals get older, they experience functional losses in domains such as cognition (Salthouse, 2004), physical abilities, and health (albeit with considerable individual differences, Rowe & Kahn, 1997). They also experience losses in their social networks as retirement from the workforce limits daily social contacts and friends are lost due to relocation, illness, and death (Charles & Carstensen, 2007; Wrzus, Hänel, Wagner, & Neyer, 2013). Lifespan developmental theory emphasizes the importance of finding new sources of meaning in late life, because earlier sources of meaning (e.g., partner selection, family building, career building) are no longer as relevant. These can include developing qualities of generativity (caring for future generations) and integrity (acceptance of one's life) (Erikson, 1950), and investing more deeply in close social relationships (Carstensen, Isaacowitz, & Charles, 1999).

In late-life couples, emotion regulation becomes very important and may contribute to older adults' relatively preserved levels of well-being even in the face of decline and loss (Mather, 2012). In survey studies, older individuals report believing that they improve in this ability (Gross et al., 1997). Laboratory studies (e.g., Shiota & Levenson, 2009) paint a more complex picture, with some regulatory strategies remaining stable with age (i.e., suppressing visible signs of emotional response), others declining with age (i.e., using detached appraisals to down-regulate emotion), and still others in fact improving (i.e., using positive appraisals to down-regulate emotion). In late life, up-regulating particular kinds of positive emotion is very important. For example, reminiscing with others about past accomplishments can be particularly rewarding (Erikson, 1982), and companionate love, which is characterized by low levels of passion but high levels of intimacy and commitment (Sternberg, 1986), often assumes the position of primary importance that was occupied earlier by romantic love. Also important is the ability to down-regulate particular negative emotions such as sadness (in response to interpersonal losses) and embarrassment (in response to losses in cognitive and physical abilities). Finally, complex emotions such as poignancy, become increasingly prevalent in late life (Ersner-Hershfield, Mikels, Sullivan, & Carstensen, 2008), leading to time spent reliving and savoring memories that have both positive and negative emotional qualities (e.g., children marrying and leaving home).

Importantly, the coregulation of emotion also becomes increasingly important as older adults spend more time with their spouses as opposed to friends and acquaintances (Charles & Carstensen, 2007). For those without close friends, down-regulating negative emotions (e.g., sadness, fear) associated with loneliness becomes critically important. The stakes may be especially high for failures of emotion regulation in late life; the negative consequences of loneliness, for example, on health, have been widely documented (Hawkley & Cacioppo, 2010).

Earlier we presented evidence that couples' ability to lower levels of physiological arousal is an important predictor of relationship quality and stability over time (Gottman & Levenson, 1992; Levenson & Gottman, 1985). These kinds of calming effects can be produced by touch (Coan,

Schaefer, & Davidson, 2006) and also by positive emotions, which can reduce levels of autonomic nervous system arousal produced by negative emotions in individuals (Fredrickson & Levenson, 1998) and in couples (Yuan, McCarthy, Holley, & Levenson, 2010). These soothing effects of positive emotions may contribute to positive emotions becoming increasingly important, desired, and salient in late life (Carstensen et al., 1999).

Assessment

Emotion regulation in couples can be measured using *self-report measures* that assess beliefs (one's own or those of others who know us) about emotion regulation. Alternatively, emotion regulation in couples can be measured using *performance measures* based on the observation of actual emotion regulation. In both self-report and performance measures, the focus can be on regulatory *abilities* (i.e., what the person or couple is capable of doing) or regulatory *practices* (i.e., what the person or couple typically does). As noted earlier, there are aspects (e.g., coregulation) and qualities (e.g., dynamic) of emotion regulation that are more prominent in couples than in individuals. Thus, the measures used for assessing emotion regulation in couples should be designed to capture these qualities, in addition to qualities that are also prominent in emotion regulation in individuals.

Unfortunately, the state of the art in measuring emotion regulation in couples is not as advanced as we would wish. As we discuss below, most existing self-report measures of emotion regulation clearly focus on individual regulation. The self-report measures that do have items relevant for assessing emotion regulation in couples were almost all designed for other purposes (e.g., measuring relationship satisfaction). Progress in developing methods for assessing actual regulatory performance in couples is also hindered by the lack of studies of emotion regulation that include an actual (or even an imagined) interaction partner (Campos et al., 2011). Moreover, when such studies have been conducted, they have often used unacquainted dyads (e.g., Butler et al., 2003). These stranger pairings, although clearly useful, are a far cry from the kinds of intimate dyads in which emotion regulation emerges and is refined during development (see earlier discussion).

Observational studies of mother–infant, mother–toddler, and mother–preschooler dyads (Cole et al., 2003; Denham, 1993; Dumas, LaFreniere, & Serketich, 1995; Tronick, 1989) provide one bright spot in this otherwise sparsely populated landscape. Although typically not designed to study emotion regulation per se, they do offer some valuable insights as to how emotion regulatory processes function in these early-life dyads.

Self-Report Measures

In the emotion regulation domain, most self-report measures focus on regulation in the individual, not in the couple. For example, the Emotion Regulation Questionnaire (Gross & John, 2003) assesses two styles of emotion regulation using a 10-item scale in which six items assess the dispositional tendency to use cognitive reappraisal strategies and four items assess the dispositional tendency to use suppression strategies. Another individual-focused inventory, the Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004), comprises 36 items that assess six dimensions of emotion regulation: (1) lack of awareness of emotional responses, (2) lack of clarity of emotional responses, (3) nonacceptance of emotional responses, (4) limited access to emotion regulation strategies, (5) difficulties controlling impulses when experiencing negative emotions, and (6) difficulties engaging in goal-directed behavior when experiencing negative emotions.

It is certainly possible to alter an individual self-report measure of emotion regulation so that items refer to a particular couple relationship. However, this would essentially constitute a new instrument, and its reliability and validity would need to be established. Moreover, questionnaires that were originally developed with a focus on the individual are unlikely to assess aspects of emotion regulation that are particularly relevant to couples (e.g., reciprocity of emotion, reactions to each other's regulatory styles).

The literature on close relationships has produced a number of self-report inventories

that were developed to assess relationship functioning and satisfaction. A subset of these inventories focuses on couples' conflict resolution and communication skills, and includes items that are relevant to assessing couples' emotion regulation. For example, in the 78-item Revised Conflict Tactics Scales (Straus, Hamby, Boney-McCoy, & Sugarman, 1996), respondents are asked about how they deal with disagreement, with questions asking how often they "shouted at or yelled at my partner," or how often conflict escalated into several more violent acts. In the 109-item Managing Affect and Differences Scale (Arellano & Markman, 1995), subscales that are directly relevant to emotion regulation include Negative Escalation (e.g., "Unable to get out of heated arguments"), Stop Actions (e.g., "When conflicts get out of hand, agree to stop and talk at a later time"), and Withdrawal (e.g., "When discussing issues, my partner usually withdraws for fear of conflict"). In a brief, 10-item screening inventory for relationship discord (Whisman, Snyder, & Beach, 2009), respondents are asked, "Whenever you are feeling sad, does your partner make you feel loved and happy again?" and "Do minor disagreements with your partner often end up in big arguments?"

There are a few self-report measures that do focus on individuals' emotion regulation vis-à-vis a specific partner (e.g., a romantic partner or a parent). Arguably, the best known of these are primarily designed to assess attachment styles, but they do have items of relevance to emotion regulation. The Experiences in Close Relationships Scale (Brennan, Clark, & Shaver, 1998) is a 36-item measure that assesses avoidance and anxiety in close relationships, with items that focus on the dyadic context (e.g., "I prefer not to show a partner how I feel deep down"). The Adult Attachment Interview (Main & Goldwyn, 1984) is based on 20 questions, some of which focus on important aspects of emotion regulation (e.g., experiences with parents involving distress, separation, rejection, or loss). It analyzes responses to these questions, focusing on not only what is said but also how it is said (e.g., the coherence of the narrative).

All of the measures reviewed thus far yield scores that are considered to represent trait-like qualities. Such approaches are ideal for capturing enduring dispositional qualities of couples' regulatory styles but are not well-suited for capturing the dynamics of emotion regulation. Emotion self-reports can be obtained in ways that yield more dynamic information using affect rating dial (Ruef & Levenson, 2007) and experience sampling (Hektner, Schmidt, & Csikszentmihalyi, 2007) methodologies. There have also been attempts to use self-report measures of emotion regulation in new ways to capture dynamic dyadic processes. For example, in one study (Butner, Diamond, & Hicks, 2007), couples were asked to provide daily ratings of positive and negative affect for 3 weeks, operationalizing coregulation as covariation in partners' daily levels of affect and coupling of the rates of change of partners' affective cycles. In a similar vein, Ferrer and Nesselroade (2003) assessed emotional experience of partners in one married dyad, who recorded their experience of 20 emotions over 182 consecutive days.

None of these approaches provides an off-the-shelf solution for measuring emotion regulation in couples, but they do provide items and scales (mostly focused on the management of negative emotion) that could be useful starting points for building more comprehensive couple assessment instruments. Nonetheless, our enthusiasm for self-report measures of emotion regulation is tempered by the problems that beset all self-report measures (e.g., social desirability and other self-presentation biases, vulnerability to wording and context). In the realm of emotional functioning, these problems are compounded by individuals often not being very accurate observers and reporters of the nuances of their own emotional functioning. Evidence of this comes from findings of low correlations between self-report and behavioral measures of emotional functioning (e.g., low correlations between well-established self-report measures of empathy and a performance-based measure of empathic accuracy; Levenson & Ruef, 1992) and variations among individuals in the coherence between self-report, behavioral, and physiological measures of emotion (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005; Sze, Gyurak, Yuan, & Levenson, 2010). Whether these kinds of problems do in fact beset particular questionnaires designed to measure emotion reg-

ulation in couples, and the ultimate utility of such questionnaires for particular purposes, are best settled on the basis of actual data.

Performance Measures

Whereas self-report measures of emotion regulation assess the rater's beliefs about emotion regulation in self or others, performance-based measures assess emotion regulation *in vivo*, evaluating emotion regulation as it actually occurs. These are often multimethod performance measures that include assessment of subjective experience, emotional behavior, and physiological activation.

As noted earlier, performance measures (and self-report measures) can focus on either regulatory abilities or regulatory practices. Measures of regulatory abilities typically provide participants with specific instructions to alter some aspect of emotional responding (e.g., "Try not to let your emotions show"), then assess how well they do (e.g., measuring the amount of visible emotional expression using a sensitive behavioral coding system). Measures of regulatory practices, in contrast, typically place participants in a situation in which emotion regulation would be expected to occur (e.g., discussing an area of conflict with a relationship partner), then assess how well participants regulate their emotions (e.g., measuring positive and negative emotional behaviors and physiological arousal).

Each of these approaches has advantages and disadvantages. For example, measures of abilities tell us a lot about what people are capable of doing in the emotion regulatory domain but not necessarily what they actually do in their day-to-day lives (e.g., a person might be able to suppress all visible signs of emotion when watching a sad movie but be very volatile in interactions with her romantic partner, or vice versa). With measures of practices, the lack of instructions reduces experimental control, and inferential leaps must be made between what is measured and its relationship to emotion regulation (e.g., slowed heart rate might indicate emotional calming, but it can also have many different psychological and physiological causes). We should also note that recent evidence from neurological patients indicates that ability and performance measures of emotion regulation are likely subserved by different neural circuits (Goodkind, Gyurak, McCarthy, Miller, & Levenson, 2010). For all of these reasons, measures of abilities and performance should not be used interchangeably.

Measures of regulatory ability that have been widely used in studies of emotion regulation in individuals instruct participants to suppress and amplify behavioral responses (e.g., Gross & Levenson, 1993, 1997; Hagemann, Levenson, & Gross, 2006; Roberts, Levenson, & Gross, 2008) and use various appraisal strategies (e.g., Gross, 1998a; Shiota & Levenson, 2009). However, these measures have not been used as much with couples. One exception is a study by Richards, Butler, and Gross (2003), in which dating couples were instructed to reappraise or suppress their emotions as they discussed a relationship conflict to determine the effects on memory for conversation utterances (which were increased by reappraisal and decreased by suppression) and emotional memories (which were increased by suppression).

Measures of regulatory practices have been utilized in studies of emotion regulation in adult dyads, with emotion regulation operationalized in a number of different ways, including (1) the amount of negative or positive emotional experience (e.g., feeling positive regard; Murray, 2005); (2) the amount or ratio of negative and positive emotional behavior (Gottman, 1993; Gottman & Levenson, 1992); (3) autonomic nervous system activation (Levenson & Gottman, 1985); and (4) central nervous system activation (Coan et al., 2006).

Measures of regulatory practices have also been utilized in mother–infant dyads, most famously in research using the Strange Situation (Ainsworth, Blehar, Waters, & Wall, 1978). In this test, children's behavior is measured after separation from the attachment figure, a prototypical situation for eliciting distress and fear in infants. Another measure of emotion regulation in infant–caregiver dyads is the Still-Face Paradigm (Tronick, Als, Adamson, Wise, & Brazelton, 1978). Here, the primary caregiver becomes unresponsive and maintains a neutral facial expression. In response, infants typically show increased gaze aversion, less smiling, and heightened negative affect.

Many of the aforementioned approaches to measuring emotion regulation in couples result in scores that represent regulation averaged over some period of time. However, it is also possible for performance-based measures to be dynamic, quantifying patterns of emotional reactivity and regulation that unfold over time. These more dynamic approaches have been utilized in studies that track changes in emotional experience (e.g., Levenson & Gottman, 1983), emotional behavior (see review in Gottman & Levenson, 1988), autonomic nervous system physiology (Yuan et al., 2010), hormonal responses (Laurent & Powers, 2007), and central nervous system activation (Coan et al., 2006). Particularly promising are those techniques that use the responses of both members of the dyad to characterize qualities of emotional coregulation over time (Butler, 2011). These include measures of (1) *emotional reciprocity*—the exchange of emotions between partners in continuous self-ratings of emotional experience (Levenson & Gottman, 1983) or observational coding of emotional behavior (Carstensen et al., 1995; Gottman et al., 1998; Gottman & Levenson, 1992; Julien, Brault, Chartrand, & Bégin, 2000; Tronick et al., 1978); (2) *emotional linkage*—the extent to which physiological, hormonal, or mood responses of partners become "synchronized" or can be predicted from each other (Levenson & Gottman, 1983; Saxbe & Repetti, 2010); or (3) *physiological soothing*—the transition from high to low levels of autonomic activation in a couple (Yuan et al., 2010). Recently, Levenson (2013) described a new statistical approach that characterizes cycles of emotion (transitions between high and low arousal, and between negative and positive emotion) that incorporates continuous measurement of behavior, physiology, and subjective experience.

Agenda for Future Research

Research on emotion regulation in couples is poised for growth and discovery. Studies of close social relationships are dramatically increasing in many areas of psychology. Clearly, we have only scratched the surface in terms of both understanding the role that emotion regulation plays in the functioning of couples' relationships and using couples' relationships as a test bed for increasing our understanding of what emotion regulation is, how it operates, and its sources and consequences. For this research area to move forward and realize its potential, there are several pressing needs:

1. Needed are sound self-report and performance measures of couples' emotion regulation that have been carefully constructed and have well-established psychometric qualities of reliability and validity. Measures are needed that (a) assess both emotion regulatory abilities (what people *can* do) and practices (what people *do* do); (b) move beyond a primary focus on the down-regulation of negative emotion to include the up-regulation and down-regulation of both positive and negative emotion; (c) assess multiple aspects of emotion regulation (subjective experience, behavior, physiology); and (d) allow assessment of emotion regulation in ways that capture its dynamic, iterative, co-regulatory nature.

2. Needed are the development and refinement of experimental paradigms that are appropriate for studying emotion regulatory abilities and practices in couples. We believe that observational studies of couple and parent–child interactions provide good bases for moving forward, but hope that new paradigms will also be developed that stimulate new research in this area.

3. Relationships between self-report and performance measures of emotion regulation in couples need to be studied and established. The existing self-report measures of emotion regulation in individuals have generally not been studied in this way; thus, there is not a firm basis for assuming equivalencies across methods.

4. Issues concerning the distinctions between emotion reactivity and emotion regulation need to be addressed in emotion regulation in couples, in much the same way as they are being addressed in research on individual emotion regulation (e.g., Ochsner et al., 2009). These are challenging issues with deep theoretical and practical implications. They should benefit greatly from expanding knowledge about how emotional functioning is organized in the central and peripheral nervous systems.

5. Research is needed on the antecedents of emotion regulation in couples. We expect that additional progress can be made in exploring biological (e.g., genetic, temperamental) and psychological (e.g., personality, attachment history) factors that predispose couples to develop particular regulatory styles. Of great interest are the ways that couples' emotion regulatory styles develop over the course of the relationship, when they are malleable, and when they solidify.

6. More research is needed on the *consequences* of emotion regulation, with special attention given to mapping different kinds of emotion regulation in relation to outcomes in multiple domains (including physical and mental heath, well-being, and relationship quality). This kind of research would benefit greatly from longitudinal designs; we expect that many effects of particular regulatory styles only emerge over fairly long periods of time.

7. Research is needed on the similarities and differences that emerge when socially situated emotion regulation is scaled up from dyads to larger groups (e.g., community responses to traumatic events). We need more comparisons of the nature of emotion regulation in different kinds of dyads (e.g., romantic, friendship, coworker dyads). We also need more research on how communities deal with tragic and triumphant events, with a particular focus on the emotion regulatory challenges that must be faced in their aftermath.

8. Research is needed on ways to improve emotion regulation abilities in individuals, dyads, and groups. In the realm of adult relationships, there are a number of therapeutic approaches that afford particular attention to emotional functioning (Gottman & Gottman, 2008; Johnson, 1996; Wile, 2002). Unfortunately, research on the efficacy and effectiveness of these treatments tends to compare outcomes for one extremely complex, multifaceted treatment package to a non-treatment condition (or sometimes to another highly complex treatment package). More research is needed using designs that enable honing in on the "active ingredients" responsible for specific areas of change and improvement. For parent–child relationships, despite a huge cottage industry devoted to proffering parenting advice, the research needs are similar.

Although the state of the science in emotion regulation in couples is still relatively immature, the potential is clearly enormous. With the development of new measurement tools and sound, ecologically valid experimental paradigms that enable us to study regulatory dynamics in interpersonal contexts, we expect great progress to be made in many important areas related to emotion regulation in couples.

References

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the Strange Situation.* Oxford, UK: Erlbaum.

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*, 217–237.

Arellano, C. M., & Markman, H. J. (1995). The Managing Affect and Differences Scale (MADS): A self-report measure assessing conflict management in couples. *Journal of Family Psychology, 9*, 319–334.

Aron, A., Fisher, H., Mashek, D. J., Strong, G., Li, H., & Brown, L. L. (2005). Reward, motivation, and emotion systems associated with early-stage intense romantic love. *Journal of Neurophysiology, 94*, 327–337.

Aron, E. N., & Aron, A. (1996). Love and the expansion of the self: The state of the model. *Personal Relationships, 3*, 45–58.

Bowlby, J. (1988). *A secure base: Parent–child attachment and healthy human development.* New York: Basic Books.

Brennan, K. A., Clark, C. L., & Shaver, P. R. (1998). Self-report measurement of adult attachment: An integrative overview. In J. A. Simpson & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 46–76). New York: Guilford Press.

Butler, E. A. (2011). Temporal interpersonal emotion systems: The "TIES" that form relationships. *Personality and Social Psychology Review, 15*, 367–393.

Butler, E. A., Egloff, B., Wilhelm, F. H., Smith, N. C., Erickson, E. A., & Gross, J. J. (2003). The social consequences of expressive suppression. *Emotion, 3*, 48–67.

Butner, J., Diamond, L. M., & Hicks, A. M. (2007). Attachment style and two forms of affect coregulation between romantic partners. *Personal Relationships, 14*, 431–455.

Campos, J. J., Walle, E. A., Dahl, A., & Main, A. (2011). Reconceptualizing emotion regulation. *Emotion Review, 3*, 26–35.

Carstensen, L. L., Gottman, J. M., & Levenson, R. W. (1995). Emotional behavior in long-term marriage. *Psychology and Aging, 10*, 140–149.

Carstensen, L. L., Graff, J., Levenson, R. W., & Gottman, J. M. (1996). Affect in intimate relationships: The developmental course of marriage. In C. Magai & S. H. McFadden (Eds.), *Handbook of emotion, adult development, and aging* (pp. 227–247). San Diego, CA: Academic Press.

Carstensen, L. L., Isaacowitz, D. M., & Charles, S. T. (1999). Taking time seriously: A theory of socioemotional selectivity. *American Psychologist, 54*, 165–181.

Caspi, A., Henry, B., McGee, R. O., Moffitt, T. E., & Silva, P. A. (1995). Temperamental origins of child and adolescent behavior problems: From age three to fifteen. *Child Development, 66*, 55–68.

Charles, S. T., & Carstensen, L. L. (2007). Emotion regulation and aging. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 307–327). New York: Guilford Press.

Christensen, A. (1987). Detection of conflict patterns in couples. In K. Hahlweg & M. J. Goldstein (Eds.), *Understanding major mental disorder: The contribution of family interaction research* (pp. 250–265). New York: Family Process Press.

Coan, J. A., Schaefer, H. S., & Davidson, R. J. (2006). Lending a hand: Social regulation of the neural response to threat. *Psychological Science, 17*, 1032–1039.

Cole, P. M., Teti, L. O., & Zahn-Waxler, C. (2003). Mutual emotion regulation and the stability of conduct problems between preschool and early school age. *Development and Psychopathology, 15*, 1–18.

Côté, S., Gyurak, A., & Levenson, R. W. (2010). The ability to regulate emotion is associated with greater well-being, income, and socioeconomic status. *Emotion, 10*, 923–933.

Cowan, C. P., & Cowan, P. A. (1992). *When partners become parents*. New York: Basic Books.

Denham, S. A. (1993). Maternal emotional responsiveness and toddlers' social-emotional competence. *Journal of Child Psychology and Psychiatry, 34*, 715–728.

Doss, B. D., Rhoades, G. K., Stanley, S. M., & Markman, H. J. (2009). The effect of the transition to parenthood on relationship quality: An 8-year prospective study. *Journal of Personality and Social Psychology, 96*, 601–619.

Dumas, J. E., LaFreniere, P. J., & Serketich, W. J. (1995). "Balance of power": A transactional analysis of control in mother–child dyads involving socially competent, aggressive, and anxious children. *Journal of Abnormal Psychology, 104*, 104–113.

Erikson, E. H. (1950). *Childhood and society*. New York: Norton.

Erikson, E. H. (1982). *The life cycle completed: A review*. New York: Norton.

Erikson, E. H., Erikson, J., & Kivnick, H. (1986). *Vital involvement in old age*. New York: Norton.

Ersner-Hershfield, H., Mikels, J. A., Sullivan, S. J., & Carstensen, L. L. (2008). Poignancy: Mixed emotional experience in the face of meaningful endings. *Journal of Personality and Social Psychology, 94*, 158–167.

Feeney, J. A. (1999). Adult attachment, emotional control, and marital satisfaction. *Personal Relationships, 6*, 169–185.

Fernald, A. (1991). Prosody in speech to children: Prelinguistic and linguistic functions. *Annals of Child Development, 8*, 43–80.

Ferrer, E., & Nesselroade, J. R. (2003). Modeling affective processes in dyadic relations via dynamic factor analysis. *Emotion, 3*, 344–360.

Fisher, H. E., Aron, A., & Brown, L. L. (2006). Romantic love: A mammalian brain system for mate choice. *Philosophical Transactions of the Royal Society B: Biological Sciences, 361*, 2173–2186.

Fredrickson, B. L., & Levenson, R. W. (1998). Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. *Cognition and Emotion, 12*, 191–220.

Gable, S. L., Gonzaga, G. C., & Strachman, A. (2006). Will you be there for me when things go right?: Supportive responses to positive event disclosures. *Journal of Personality and Social Psychology, 91*, 904–917.

Goodkind, M. S., Gyurak, A., McCarthy, M., Miller, B. L., & Levenson, R. W. (2010). Emotion regulation deficits in frontotemporal lobar degeneration and Alzheimer's disease. *Psychology and Aging, 25*, 30–37.

Gottman, J. M. (1993). A theory of marital dis-

solution and stability. *Journal of Family Psychology, 7*, 57–75.

Gottman, J. M., Coan, J., Carrere, S., & Swanson, C. (1998). Predicting marital happiness and stability from newlywed interactions. *Journal of Marriage and the Family, 60*, 5–22.

Gottman, J. M., & Gottman, J. S. (2008). Gottman method couple therapy. In A. S. Gurman (Ed.), *Clinical handbook of couple therapy* (pp. 138–164). New York: Guilford Press.

Gottman, J. M., & Levenson, R. W. (1985). A valid procedure for obtaining self-report of affect in marital interaction. *Journal of Consulting and Clinical Psychology, 53*, 151–160.

Gottman, J. M., & Levenson, R. W. (1988). The social psychophysiology of marriage. In P. Noller & M. A. Fitzpatrick (Eds.), *Perspectives on marital interaction: Monographs in social psychology of language, No. 1.* (pp. 182–200). Clevedon, UK: Multilingual Matters.

Gottman, J. M., & Levenson, R. W. (1992). Marital processes predictive of later dissolution: Behavior, physiology, and health. *Journal of Personality and Social Psychology, 63*, 221–233.

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the Difficulties in Emotion Regulation Scale. *Journal of Psychopathology and Behavioral Assessment, 26*, 41–54.

Gross, J. J. (1998a). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224–237.

Gross, J. J. (1998b). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*, 271–299.

Gross, J. J., Carstensen, L. L., Pasupathi, M., Tsai, J., Skorpen, C. G., & Hsu, A. Y. C. (1997). Emotion and aging: Experience, expression, and control. *Psychology and Aging, 12*, 590–599.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*, 348–362.

Gross, J. J., & Levenson, R.W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64*, 970–986.

Gross, J. J., & Levenson, R. W. (1995). Emotion elicitation using films. *Cognition and Emotion, 9*, 87–108.

Gross, J. J., & Levenson, R. W. (1997). Hiding feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology, 106*, 95–103.

Gross, J. J., Richards, J. M., & John, O. P. (2006). Emotion regulation in everyday life. In D. K. Snyder, J. Simpson, & J. N. Hughes (Eds.), *Emotion regulation in couples and families: Pathways to dysfunction and health* (pp. 13–35). Washington, DC: American Psychological Association.

Gross, J. J., Sheppes, G., & Urry, H. L. (2011). Taking one's lumps while doing the splits: A big tent perspective on emotion generation and emotion regulation. *Cognition and Emotion, 25*, 789–793.

Gyurak, A., Goodkind, M. S., Kramer, J. H., Miller, B. L., & Levenson, R. W. (2012). Executive functions and the down-regulation and up-regulation of emotion. *Cognition and Emotion, 26*(1), 103–118.

Gyurak, A., Gross, J. J., & Etkin, A. (2011). Explicit and implicit emotion regulation: A dual-process framework. *Cognition and Emotion, 25*, 400–412.

Hagemann, T., Levenson, R. W., & Gross, J. J. (2006). Expressive suppression during an acoustic startle. *Psychophysiology, 43*, 104–112.

Havighurst, R. J. (1976). *Developmental tasks and education*. New York: David McKay.

Hawkley, L. C., & Cacioppo, J. T. (2010). Loneliness matters: A theoretical and empirical review of consequences and mechanisms. *Annals of Behavioral Medicine, 40*, 218–227.

Hektner, J. M., Schmidt, J. A., & Csikszentmihalyi, M. (2007). *Experience sampling method: Measuring the quality of everyday life*. Thousand Oaks, CA: Sage.

Holley, S. R., Sturm, V. E., & Levenson, R. W. (2010). Exploring the basis for gender differences in the demand–withdraw pattern. *Journal of Homosexuality, 57*, 666–684.

Jankowiak, W. R., & Fischer, E. F. (1998). A cross-cultural perspective on romantic love. In J. M. Jenkins, K. Oatley, & N. L. Stein (Eds.), *Human emotions: A reader* (pp. 55–62). Malden, MA: Blackwell.

John, O. P., & Gross, J. J. (2004). Healthy and unhealthy emotion regulation: Personality processes, individual differences, and life span development. *Journal of Personality, 72*, 1301–1333.

Johnson, S. M. (1996). *The practice of emotionally focused marital therapy: Creating connection*. Philadelphia: Brunner/Mazel.

Julien, D., Brault, M., Chartrand, É., & Bégin, J. (2000). Immediacy behaviours and synchrony in satisfied and dissatisfied couples. *Canadian Journal of Behavioural Science, 32*, 84–90.

Kagan, J. (1984). *The nature of the child*. New York: Basic Books.

Kunzmann, U., Kupperbusch, C. S., & Levenson, R. W. (2005). Behavioral inhibition and amplification during emotional arousal: A comparison of two age groups. *Psychology and Aging, 20*, 144–158.

Lang, P. J., Greenwald, M. K., & Bradley, M. M. (1988). *The International Affective Picture System (IAPS) standardization procedure and initial group results for affective judgments*. Gainesville: Center for the Study of Emotion and Attention, University of Florida.

Laurent, H., & Powers, S. (2007). Emotion regulation in emerging adult couples: Temperament, attachment, and HPA response to conflict. *Biological Psychology, 76*, 61–71.

Levenson, R. W. (1976). Feedback effects and respiratory involvement in voluntary control of heart rate. *Psychophysiology, 13*, 108–114.

Levenson, R. W. (2013). Emotion and emotion regulation. In D. Hermans, B. Rimé, & B. Mesquita (Eds.), *Changing emotions* (pp. 105–112). New York: Psychology Press.

Levenson, R. W., Cowan, C. P., & Cowan, P. A. (2010). A specialty clinic model for clinical science training: Translating couples research into practice in the Berkeley Couples Clinic. In M. C. Schulz, M. K. Pruett, P. K. Kerig, & D. Ross (Eds.), *Strengthening couple relationships for optimal child development: Lessons from research and intervention* (pp. 197–209). Washington, DC: American Psychological Association.

Levenson, R. W., & Gottman, J. M. (1983). Marital interaction: Physiological linkage and affective exchange. *Journal of Personality and Social Psychology, 45*, 587–597.

Levenson, R. W., & Gottman, J. M. (1985). Physiological and affective predictors of change in relationship satisfaction. *Journal of Personality and Social Psychology, 49*, 85–94.

Levenson, R. W., & Ruef, A. M. (1992). Empathy: A physiological substrate. *Journal of Personality and Social Psychology, 63*, 234–246.

Lopes, P. N., Salovey, P., Côté, S., Beers, M., & Petty, R. E. (2005). Emotion regulation abilities and the quality of social interaction. *Emotion, 5*, 113–118.

Main, M., & Goldwyn, R. (1984). *Adult attachment scoring and classification system*. Unpublished manuscript, University of California, Berkeley.

Martin, R. A. (2007). *The psychology of humor: An integrative approach*. Amsterdam, The Netherlands: Elsevier.

Mather, M. (2012). The emotion paradox in the aging brain. *Annals of the New York Academy of Sciences, 1251*, 33–49.

Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. H., & Gross, J. J. (2005). The tie that binds?: Coherence among emotion experience, behavior, and physiology. *Emotion, 5*, 175–190.

McNulty, J. K., & Hellmuth, J. C. (2008). Emotion regulation and intimate partner violence in newlyweds. *Journal of Family Psychology, 22*, 794–797.

Mikulincer, M., & Shaver, P. R. (2007). *Attachment in adulthood: Structure, dynamics, and change*. New York: Guilford Press.

Mischel, W., Ayduk, O., Berman, M. G., Casey, B. J., Gotlib, I. H., Jonides, J., et al. (2011). "Willpower" over the life span: Decomposing self-regulation. *Social Cognitive and Affective Neuroscience, 6*, 252–256.

Murray, S. L. (2005). Regulating the risks of closeness: A relationship-specific sense of felt security. *Current Directions in Psychological Science, 14*, 74–78.

Nelis, D., Kotsou, I., Quoidbach, J., Hansenne, M., Weytens, F., Dupuis, P., et al. (2011). Increasing emotional competence improves psychological and physical well-being, social relationships, and employability. *Emotion, 11*, 354–366.

Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D., et al. (2004). For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *NeuroImage, 23*, 483–499.

Ochsner, K. N., Ray, R. R., Hughes, B., McRae, K., Cooper, J. C., Weber, J., et al. (2009). Bottom-up and top-down processes in emotion generation: Common and distinct neural mechanisms. *Psychological Science, 20*, 1322–1331.

Richards, J. M., Butler, E. A., & Gross, J. J. (2003). Emotion regulation in romantic relationships: The cognitive consequences of con-

cealing feelings. *Journal of Social and Personal Relationships, 20*, 599–620.

Roberts, N. A., Levenson, R. W., & Gross, J. J. (2008). Cardiovascular costs of emotion suppression cross ethnic lines. *International Journal of Psychophysiology, 70*, 82–87.

Rowe, J. W., & Kahn, R. L. (1997). Successful aging. *The Gerontologist, 37*, 433–440.

Ruef, A. M., & Levenson, R. W. (2007). Continuous measurement of emotion. In J. A. Coan & J. J. B. Allen (Eds.), *The handbook of emotion elicitation and assessment* (pp. 286–297). New York: Oxford University Press.

Salthouse, T. A. (2004). What and when of cognitive aging. *Current Directions in Psychological Science, 13*, 140–144.

Saxbe, D., & Repetti, R. L. (2010). For better or worse?: Coregulation of couples' cortisol levels and mood states. *Journal of Personality and Social Psychology, 98*, 92–103.

Shaver, P. R., & Mikulincer, M. (2007). Adult attachment strategies and the regulation of emotion. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 446–465). New York: Guilford Press.

Shiota, M. N., & Levenson, R. W. (2009). Effects of aging on experimentally instructed detached reappraisal, positive reappraisal, and emotional behavior suppression. *Psychology and Aging, 24*, 890–900.

Simpson, J. A., Collins, W. A., Tran, S., & Haydon, K. C. (2007). Attachment and the experience and expression of emotions in romantic relationships: A developmental perspective. *Journal of Personality and Social Psychology, 92*, 355–367.

Sternberg, R. J. (1986). A triangular theory of love. *Psychological Review, 93*, 119–135.

Straus, M. A., Hamby, S. H., Boney-McCoy, S., & Sugarman, D. B. (1996). The Revised Conflict Tactics Scales (CTS2). *Journal of Family Issues, 17*, 283–316.

Sroufe, L. A., Coffino, B., & Carlson, E. A. (2010). Conceptualizing the role of early experience: Lessons from the Minnesota longitudinal study. *Developmental Review, 30*, 36–51.

Sze, J. A., Gyurak, A., Yuan, J. W., & Levenson, R. W. (2010). Coherence between emotional experience and physiology: Does body awareness training have an impact? *Emotion, 10*, 803–814.

Thompson, R. A. (1991). Emotional regulation and emotional development. *Educational Psychology Review, 3*, 269–307.

Tronick, E., Als, H., Adamson, L., Wise, S., & Brazelton, T. B. (1978). The infant's response to entrapment between contradictory messages in face-to-face interaction. *Journal of the American Academy of Child and Adolescent Psychiatry, 17*, 1–13.

Tronick, E. Z. (1989). Emotions and emotional communication in infants. *American Psychologist, 44*, 112–119.

Whisman, M. A., Snyder, D. K., & Beach, S. R. H. (2009). Screening for marital and relationship discord. *Journal of Family Psychology, 23*, 247–254.

Wile, D. B. (2002). Collaborative couple therapy. In A. S. Gurman & N. S. Jacobson (Eds.), *Clinical handbook of couple therapy* (3rd ed., pp. 281–307). New York: Guilford Press.

Wrzus, C., Hänel, M., Wagner, J., & Neyer, F. J. (2013). Social network changes and life events across the life span: A meta-analysis. *Psychological Bulletin, 139*, 53–80.

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit formation. *Journal of Comparative Neurology and Psychology, 18*, 459–482.

Yuan, J. W., McCarthy, M., Holley, S. R., & Levenson, R. W. (2010). Physiological downregulation and positive emotion in marital interaction. *Emotion, 10*, 467–474.

CHAPTER 18

The Cultural Regulation of Emotions

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Emotion regulation promotes an individual's social adjustment. Having an emotion means to take a stance, to have a particular relationship with the world (Solomon, 2004), and to have a specific intention to act (Frijda, 1986). To take anger as an example, the experience of anger implies an attitude of nonacceptance, an assessment that one has a relatively high level of control over (others in) the situation (Frijda, Kuipers, & Terschure, 1989), and a readiness to act in such a way that these other people accommodate to your wishes, goals, and values (Stein, Trabasso, & Liwag, 1993). Emotions are thus relationship engagements (Mesquita, Marinetti, & Delvaux, 2012). *Emotion regulation* refers to all those processes involved in fashioning emotions to be most adaptive within the relationship.

Cultural differences in emotion regulation are to be expected, because the common and most valued relationships differ across sociocultural contexts (e.g., Markus & Kitayama, 1991). Therefore, the emotions that are most adaptive within those relationships—the endpoints of emotion regulation—are likely to differ cross-culturally as well. For example, feelings of cheerfulness and happiness are conceived as "good" and "desirable" in European American culture (D'Andrade, 1984; Wierzbicka, 1994), because they signal that a person has successfully managed the central cultural tasks of standing out and accomplishing personal and material goals (e.g., Hochschild, 1995). Happiness communicates to other Americans a "good inner self" and psychological well-being (Markus & Kitayama, 1994). Yet, as natural as the desire to be happy may seem to most American readers, this emotion norm is far from universal. The anthropologist Catherine Lutz (1987), herself a European American, was reprimanded for smiling at a girl who acted happy during her stay with the Ifaluk (on a Pacific atoll). The Ifaluk condemn happiness, because they believe it leads a person to neglect his or her social duties. Whereas European Americans seek to yield and to maximize happiness, Ifaluk life is geared toward minimizing this emotion. An emotion's fit with the cultural models of self and relationships will thus determine whether it is up- or down-regulated.

In this chapter we propose, first, that the endpoints of emotion regulation in each culture are those that match the culture's valued ideas and norms of how to be a good person and how to relate to other people—the so-called cultural models of self and relating (Bruner, 1990; D'Andrade, 1984; Markus & Kitayama, 1991). Second, we propose that cultural regulation of emotions may be initiated by an individual, but often is an effect of the way the social environment is

organized. Third, we will show that culture (defined both at the level of the individual and at the level of the social environment) plays an important role in all stages of emotion regulation.

Cultural Models

To provide a foundation for the discussion on emotion regulation, we first briefly review the idea of the cultural model itself. Dominant cultural models of self and relating are manifest in two distinct ways that may be described as "culture in the world" and "culture in the head" (Adams & Markus, 2004) On the one hand, "culture in the world" refers to a culture's daily routines and organizational structures, to the reward structures that are in place, to social expectancies, and to the common types of social interaction. "Culture in the world" thus stands for the affordances and constraints that implicitly (but powerfully) shape individual experience. On the other hand, "culture in the head" refers to internalized goals, values, meanings, representations, and behavioral repertoires, and thus translates into experiential and behavioral tendencies. Both manifestations of cultural models—in the world and in the head—appear to be involved in emotion regulation.

This idea can be illustrated by contrasting the most widely studied cultural models: the European American and East Asian cultural models. According to middle-class European American models of self and relating, the individual should be independent and free from others, as well as stand out among them (e.g., Kim & Markus, 1999). On the one hand, everyday social arrangements foster a self that is independent and free. Examples are sleeping arrangements: In contrast to infants in many areas of the world, European American infants sleep by themselves very early on (Morelli, Rogoff, Oppenheim, & Goldsmith, 1992; Shweder, 1991). On the other hand, culture is internalized as the psychological tendency of self-enhancement and the value attached to choice in European American contexts (Heine, Lehman, Markus, & Kitayama, 1999; Hochschild, 1995).

In contrast, the dominant goal of the self in most East Asian cultural contexts is to be like others and to enhance the fit between what one is doing and what is expected (e.g., Heine et al., 1999; Lebra, 1992). The culture values a person's self-improvement, with the aim of meeting these relational expectations, fulfilling role-based obligations, and demonstrating one's loyalty to significant social ingroups (Heine et al., 1999; Rothbaum Pott, Azuma, Miyake, & Weisz, 2000). Culture in the world is organized toward these goals: Politeness rituals and strictly prescriptive role behavior are cases in point. "Culture in the head" can be seen from the East Asian tendency to take the perspective of the generalized other and thus focus on meeting expectations (Cohen, Hoshino-Brown, & Leung, 2007; Masuda et al., 2008).

It should be noted that the theoretical framework of cultural models does not assume or argue that people who live in the same cultural context engage in exactly the same way. First, people engage in many models at the same time, such as models of gender, socioeconomic status, cohort, ethnicity, religion, and professional status. These models all structure individuals' reality, as do family dynamics, close relationship characteristics, and parenthood. Second, and perhaps relatedly, people do not internalize the dominant cultural models in the same way and to the same extent. Yet even if people have not completely internalized the dominant models, they still have to contend with cultural models in the world (Shweder, 1991).

The "Right" Emotions Match the Cultural Models

What feels "right" differs substantially across cultures, yet we propose that everywhere the endpoints of emotion regulation match the cultural models of self and relationships (Eid & Diener 2001; Kitayama, Mesquita, & Karasawa, 2006; Mesquita & Leu, 2007; Tsai, Knutson, & Fung, 2006). The "right" emotions may be those consistent with injunctive norms (*ought*-emotions, [dis]approved by most others in the culture), ideal emotions (emotions *wanted* by most others in the culture), or descriptive norms (emotions *actually* experienced by most others in the culture). Cultural differences in the explicit feeling rules and ideal emotions

illustrate the different endpoints of regulation; cultural differences in the commonly experienced emotions are taken as evidence for culture-specific emotion norms, if they match the respective models of self and relating.

Feeling Rules and Ideal Emotions Match the Cultural Models

A culture's feeling rules—the most desirable and valued emotional states—are endpoints of emotion regulation. Differences in feeling rules can be understood from differences in the dominant cultural models. For instance, Eid and Diener (2001) conducted a large cross-cultural study in which participants from both independent (European American and Australian) and interdependent (China and Taiwan) cultural contexts rated the desirability of several emotions, both positive and negative. The largest cultural differences in desirability were found for "pride" and "guilt." Feelings of pride were more positively valued in independent than in interdependent cultures, whereas the opposite was true for feelings of guilt. This may be the case, because pride signals a person's autonomy and uniqueness, which are valued in independent cultures but considered "dangerous" in interdependent cultures that recognize the potential of pride to disrupt social harmony. Conversely, guilt may be desirable from the viewpoint of East Asian cultural models, because it signals an individual's concern for relational harmony (and the readiness to take full responsibility for a violation of this harmony), but undesirable in Western cultures because it suggests a less than positive performance of the individual.

There are also systematic cultural differences in the emotions people "ideally would like to feel" (Tsai et al., 2006). Tsai and her colleagues asked people from different cultures to rate their "ideal feelings" on emotion scales that represented the four quadrants of the affective circumplex; The *affective circumplex* is defined by the dimensions of pleasantness (unpleasant–pleasant) and activation (low–high). They found consistent differences between European Americans, who "ideally" wanted to feel more high-activation positive states such as excitement and elation, and East Asians, who preferred low-activation positive states such as peaceful and serene feelings. Further research showed that ideal emotions prepare individuals best for the tasks that are culturally central (Tsai, Miao, Seppala, Fung, & Yeung, 2007). High-activation positive emotions, promoted in North American contexts, prepare individuals for influencing others. In contrast, low-activation positive emotions, promoted in East Asian contexts, facilitate social adjustment.

Patterns of Emotional Experience Match the Cultural Models

That the endpoints of regulation differ may also be inferred from cultural differences in the prevalent emotional patterns (i.e., the patterns of frequencies and intensities), which can be understood from the pertinent cultural models of self and relating. Adopting a variety of methods, Kitayama and his colleagues (2006; Kitayama & Markus, 2000) found that European American participants reported more *socially disengaging* emotions—such as feeling pride, anger, or irritation—and Japanese participants reported more *socially engaging* emotions—such as feeling close, ashamed or indebted. The dimension of social engagement was empirically derived, and emotions on the disengaging end of the dimension underline an individual's independence, whereas emotions on the engaging end foreground the connectedness between people. The studies made use of the self-reported frequency of emotions in the past month, the self-reported emotional experience in response to a predefined set of situation types, and daily dairy studies. Cultural differences in emotions, as they appear from these studies, correspond with other reports of emotions for the same cultures (see the high frequency and prevalence of shame in Japanese cultural contexts as reported by Benedict, 1946; Heine et al., 1999). Culturally prevalent patterns of emotions thus appeared to match the cultural models of self and relating.

That the endpoints of regulation are culturally defined is also suggested by research on emotional *acculturation*: changes in the patterns of emotional experience after people move to a different culture. In a series of studies, we asked immigrant and majority groups to rate the patterns of emotions experienced in different types of situations that

were defined according to the valence (positive, negative) and the social engagement (engaged, disengaged) of the emotion they tended to elicit. For each type of situation, we compared immigrants' pattern of emotion ratings with the average majority member's pattern. The more an immigrant had been exposed to the new culture, the more his or her pattern of emotions resembles that of the average majority member. This finding held true for two different minority groups in two different majority cultures: Turkish immigrants in Belgium, and Korean immigrants in the United States (De Leersnyder, Mesquita, & Kim, 2011). In both immigrant groups, the number of years spent in the new culture, as well as the degree of contact with members of the majority culture, predicted emotional similarity to the majority culture. A separate study compared, for the same type of situations, emotional patterns of Belgian Turks and Belgian natives to those of Turks living in Turkey (De Leersnyder, Mesquita, & Kim, 2013). Emotional patterns of Belgian Turks were more similar to the Belgian norm than were emotional patterns of Turks in Turkey, even though the Turks in Turkey were on average better educated (and in that respect more similar to the Belgians) than Belgian Turks. The combined findings suggest that exposure to a new culture sparks changes in the prevalent emotional patterns that supposedly match the new cultural models more closely.

That cross-culturally different endpoints of regulation are rewarding can be inferred from the link with well-being. The combined evidence suggests that having the "right" emotions is associated with higher well-being. In the diary study reviewed earlier (Kitayama et al., 2006), emotions that were consistent with the descriptive norm best predicted well-being: disengaging emotions in the European American sample, and engaging emotions in the Japanese sample. Similarly, our own research with U.S., Korean, and Belgian samples showed that emotional similarity to the cultural average predicted higher satisfaction with social relationships (De Leersnyder, Mesquita, Kim, Eom, & Choi, 2013).Thus, having the "right" emotions appears to be rewarding, even if the exact processes involved are as yet unknown.

Conversely, emotions that are *discrepant* with the culturally ideal emotions have negative consequences for well-being. Tsai and her colleagues (2006) found that the discrepancy between a person's actual and ideal emotions predicted that person's level of depressive symptoms, but only for the domain of emotions that was culturally most focal: The discrepancy between actual and ideal high-activation positive emotions predicted depression in European Americans, whereas the discrepancy between actual and ideal low-activation positive emotions predicted depression in East Asians. Therefore, *not* having the "right" emotions appears to have negative personal consequences.

In summary, emotions appear to be regulated in ways that improve their match with the prevalent cultural models of self and relating. This is suggested by findings that (1) actual emotions fit the culture's models of self and relating, (2) immigrants' actual emotions become more consistent with the new cultural models, and (3) emotions that are consistent with either the descriptive norms or the ideal emotions in a culture positively predict well-being. The rest of this chapter is devoted to the question of *how* the match between emotions and cultural models is achieved: This is the domain of emotion regulation proper.

Cultural Regulation of Emotions

Emotion regulation has been defined broadly as "the processes that influence which emotions we have, when we have them, and how we experience or express these emotions" (Gross, 1998; Gross, Sheppes, & Urry, 2011, p. 767). In this chapter, we subscribe to this definition, and we note that it is agnostic to (1) the source of regulation, and (2) whether the regulation is effortful or conscious. We propose that culturally driven emotion regulation may be initiated by either individuals themselves or their social environments, and they may be either conscious and effortful or automatic and effortless. We speak of cultural regulation when there is evidence that emotional processes are fashioned to match the cultural models of self and relationship.

Most of the literature on emotion regulation has focused on *individual emotion regulation*—for example, a person who avoids seeing a friend who makes her feel miserable, who seeks distraction after a fight with

her boss, or who tries to see the situation in a different light. However, a full understanding of the role of culture in emotion regulation requires that we move beyond individual emotion regulation to include what we call *social emotion regulation*. We speak of social emotion regulation when the social environment is the agent of emotion regulation. Culture "in the world" is clearly important to this type of regulation. Examples abound: Sex segregation in some cultures is a means to avoid situations of embarrassment (Abu-Lughod, 1986); head hunting rituals in another culture serve to channel individuals' ingroup anger (Rosaldo, 1980). Closer to home, social sharing with friends can help to restore a person's self-esteem after a painful breakup, thereby alleviating distress. Thus, culture plays an important role in both individual and social emotion regulation.

It should be noted that our approach differs from accounts that assimilate emotion regulation to the effortful redirection of "the spontaneous flow" of emotions (Koole, 2009, p. 6). First, in light of cultural differences in the most prevalent emotions, we challenge the notion of spontaneous flow. Second, we propose that emotion regulation is often automatic (cf. Bargh & Williams, 2007; Gyurak, Gross, & Etkin, 2011; see Mauss, Bunge, & Gross, 2008, for a cultural perspective on automatic emotion regulation).

It is not always possible (and perhaps not meaningful either; see Mesquita & Frijda, 2011) to draw a distinction between cultural emotion regulation and culturally influenced emotion generation. Regulatory processes are clearly distinct when individual and social emotion regulation changes the course of an ongoing emotion in culture-specific ways, but in many cases, emotion regulation is inferred from the fact that emotional outcomes cross-culturally vary in nonrandom ways. For example, the types of situations that individuals encounter may be responsible for aligning emotions with the endpoints of regulation (e.g., politeness reduces anger, and may thus help to achieve relational harmony). In this case, the distinction between emotion generation and emotion regulation becomes futile (Mesquita & Frijda, 2011). However, we speak of emotion regulation in these cases because the social organization indisputably plays a role in "what emotions we have" and "when we have them" (Gross et al., 2011, p. 767). To the extent that either individual or social processes seem to increase the match of emotions to norms, we consider them forms of emotion regulation.

We assume that a culture's individual and social emotion regulation strategies go hand in hand, and supplement each other, in an attempt to realize the cultural norms (Kitayama, Karasawa, & Mesquita, 2004; Mauss et al., 2008). Below we discuss evidence for the role of culture in multiple strategies of emotion regulation; for each strategy we consider evidence for both individual and social emotion regulation.

Situation Selection

One major path of emotion regulation has been termed *situation selection*, described as "approaching or avoiding certain people, places, or objects in order to regulate emotions" (Gross, 1998, p. 283). Cultural differences in situation selection are suggested with regard to individual and social emotion regulation. Differences in individuals' motivational focus are taken as evidence for the role of culture in individual emotion regulation; differences in the social realities that individuals encounter—the a priori selection of situations—are considered a form of social emotion regulation.

Individual Regulation of Situation Selection

There is some evidence that different cultural models affect individuals' situation selection. A number of studies found that the relative focus of individuals on either avoiding negative or approaching positive situations differs across cultures (e.g., Elliott, Chirkov, Kim, & Sheldon, 2001; Lee, Aaker, & Gardner, 2000). Whereas the dominant focus in American contexts appears to be on the *accomplishment* of positive outcomes (i.e., a promotion focus), East Asians and Russians have been found to be more concerned with *avoiding* failures to meet social expectations (i.e., a prevention focus). These cultural differences in motivational focus can be understood from the respective models of self and relating: An independent American model emphasizes

self-enhancement and the achievement of positive outcomes, whereas the more interdependent East Asian and Russian models underline the importance of avoiding social violations, and may thus be more driven by avoidance than approach.

That differences in situation selection may affect emotional experience was suggested by a cross-cultural vignette study by Lee et al. (2000, Studies 3–5). The researchers examined differences in emotional reactions to success and failure between European Americans (promotion focus) and East Asian Hong Kong Chinese (prevention focus). They hypothesized that a promotion focus would foster happiness under conditions of success, and sadness under conditions of failure, whereas a prevention focus would lead to reports of relaxation or relief upon success, and anxiety upon failure. Consistent with the differences in motivational focus, the European American participants reported higher intensities of happiness–depressed emotions than relief–anxiety emotions, whereas the Chinese participants reported higher intensities of relief–anxiety than happiness–depressed emotions. This is some of the first evidence that cultural differences in situation selection may be related to differences in the prevalent types of emotions. In summary, cultural models of self and relating motivate individual-level selection of situations, which itself can be understood as a form of emotion regulation.

Social Regulation of Situation Selection

Social situation selection may render certain emotions either more frequent or more rare. For example, European American social life is characterized by practices that serve to make individuals feel special and unique, and that promote happiness and feeling good about themselves (D'Andrade, 1984). Contemporary American schools promote the happiness and pride of their students with practices that make the children feel important and special. As early as preschool, children are the focus of attention during "show-and-tell" meetings, and throughout elementary school, children's accomplishments are marked by smiley faces, stickers, and gift-box rewards for every achievement, however small. The importance of self-esteem is nicely illustrated by anecdotal evidence. In his book *The Geography of Thought*, the American psychologist Richard Nisbett (2003) describes how the school board in his hometown even "debated whether the chief goal of the schools should be to impart knowledge or inculcate self-esteem" (p. 55).

In contrast, many of the practices in Japanese cultural contexts promote anticipatory fear or shame, consistent with a cultural model emphasizing the continuing obligation to accommodate others, fulfill one's roles, and perfect one's contributions in order to approach others' expectations or cultural ideals in general (Heine et al., 1999). For example, at the end of each day Japanese schoolchildren are encouraged to engage in self-reflection or self-criticism (i.e., *hansei*), so that they can look for ways to improve their shortcomings or weaknesses in order to meet the group's standards (e.g., Lewis, 1995). The constant awareness of one's shortcomings is conducive to the experience of emotions such as anticipated fear and shame.

Culturally comparative research provides systematic support for the idea that situations eliciting culturally condoned emotions, such as shame in Japan, are relatively more frequent than culturally condemned emotions, such as anger in Japan (e.g., Boiger, Mesquita, Uchida, & Barrett, 2013). In a recent study, we compared the frequency of shame and anger situations in Japan and the United States, utilizing carefully selected, representative elicitors of shame and anger that had been reported in extensive pilot studies by either Japanese or U.S. college students. Shame was chosen because it was expected to be consistent with an interdependent, but not with an independent, model of self and relating; anger was selected because it was thought to have the opposite connotations. New samples of Japanese and U.S. students read standardized vignettes describing these eliciting situations, and rated (1) their likelihood of occurrence and (2) the degree to which the situations would elicit the intended emotion (anger or shame). In the United States, where shame violates the cultural model of independence, we expected people to avoid shame, but in Japan, where shame is an expression of the cultural model of interdependence, we

expected them to promote it. In contrast, we expected anger to be avoided in Japan (because it interferes with social harmony), and promoted in the United States (since it underlines independence). The findings provided support for culturally different patterns of situation selection. Situations that elicited higher levels of shame were rated as more likely to occur in Japan, and as less likely to occur in the United States. In contrast, situations that were thought to elicit more anger were rated as more frequent in the United States, and less frequent in Japan. Thus, the "social" selection of situations may contribute to the previously reported differences in the frequencies with which people in American and Japanese cultural contexts experience anger (disengaging) and shame (engaging) emotions (see Kitayama et al., 2006). One way of understanding these findings is that Americans simply encounter more anger-provoking situations, and Japanese more shame-provoking situations in their everyday lives. Social conventions may help to avoid situations of interpersonal frustration in Japan, and situations of interpersonal criticism in the United States, respectively. In other words, social interactions may be structured in culture-specific ways that affect emotional experience.

Child-rearing practices provide another example of social situation selection affecting emotional experience. Several studies suggest that Japanese mothers structure their children's environment in ways that encourage relative stability and moderation of emotion, whereas American mothers provide greater situational variability, thus increasing the probability that their children will experience a range of both positive and negative emotions (Rothbaum et al., 2000). Japanese mothers lull and comfort their infants more by soothing and maintaining close proximity to their children. In doing so, they create a safe and stable environment, thus decreasing the likelihood that their children experience strong negative emotions. In contrast, American mothers allow their children more exploratory activity and use more distal proximity strategies (e.g., eye contact) than do Japanese mothers (Rothbaum et al., 2000). They offer their children the opportunity for exploration, and in doing so create more excitement but also greater potential for negative emotions.

In summary, cultural differences in the common forms of social interaction account for differences in emotion experience. We speak of *social situation selection* if these differences in emotional experience match the respective cultural models of self and relating. Several studies suggest the existence of cultural differences in social situation selection.

Focus of Attention

Culture may also be instrumental in regulating the focus of attention, or what in the emotion regulation literature has been called *attentional deployment* (Gross, 1998): the channeling of attention in ways that are conducive to the desired emotional outcome. There is some evidence that attention may be channeled in ways that are consistent with the cultural models.

Individual Regulation of the Focus of Attention

Cultural models may affect individuals' focus of attention on relatively different aspects of emotional situations. In a comparative study on emotion perception, Japanese and Americans rated the emotions of a central person expressing anger, sadness, or happiness, who was surrounded by four other people (Masuda et al., 2008). The facial expressions of the other people varied, independent of the expression of the central person. Consistent with an independent model, Americans exclusively focused attention on the central person's emotions, disregarding the emotions of the surrounding people. Consistent with an interdependent model, however, the Japanese focused attention on the other people in the picture as well. For example, Japanese rated the sadness of the central person to be higher if the other people in the situation were sad as well, relative to situations in which the other people were not sad. Eye tracking confirmed that, compared to American participants, Japanese participants spent more time scanning the periphery of the picture for the facial expressions of the other people. Therefore, consistent with interdependent models of self and relating, Japanese focused their attention on everybody involved in the social situation.

Culture might focus individuals' attention on not only different types of *social* information but also differently *valenced* information. A recent study by Grossmann, Ellsworth, and Hong (2012) compared Russian and European American participants' focus on positive and negative emotional stimuli, respectively. This was based on ethnographic and empirical work that characterized Russian people as "brooders" who place greater value than European Americans on immersing themselves in negative feelings. The researchers hypothesized that Russians' attention would be drawn more to negative stimuli. In support of this hypothesis, Russians, but not European Americans, spent significantly more time looking at negative than at positive stimuli. In a follow-up study with biculturals, Russian culture was compared to a European culture (Latvia). Making use of a technique used to study cultural frame switching in biculturals, the authors primed bicultural Russian Latvians' with either Russian or Latvian culture, by presenting them with Russian or Latvian cultural symbols. Priming biculturals with their Russian heritage led to a faster recognition of negative words and a slower recognition of positive words as compared to their baseline reaction time to these words; also consistent with the authors' expectations, Russian Latvians primed with Latvian cultural symbols failed to focus more attention on negative information. Interestingly, the effect of priming Russian culture was more pronounced for biculturals who reported more affinity with their Russian heritage; it is possible that the priming activated a more elaborate Russian emotional repertoire in these individuals.

Social Regulation of the Focus of Attention

Other people are often involved in funneling an individual's attention. We refer to this as *social regulation* when the changed focus of attention aligns the individual's emotional experiences with the dominant cultural model of self and relating. Research on caregiver–child interactions in two Nepalese groups illustrates this idea nicely (Cole, Tamang, & Shrestha, 2006). Tamang and Chhetri-Brahmin mothers were asked what they would do if their 4- or 5-year-old child was angry at them. Both groups of mothers channeled their children's attention in ways that were consistent with the cultural models. The majority of Tamang mothers reported that they would give the children food and cajole them in order to calm them down; this distraction (of attention) strategy is consistent with Tamang Buddhist models of social harmony that focus on egalitarianism, compassion, and tolerance. In contrast, the majority of Chhetri-Brahmin mothers reported they would respond by telling their children to behave, to study, and to go to school. The Chhetri-Brahmin mothers thus focused their children's attention on the values of social order and disciplined action central to their Hindu cultural models, supposedly inducing more conflictual emotions. In both cases, the refocusing of attention had an effect on emotional experience that was consistent with the cultural model.

Further evidence for the role of others in focusing attention comes from an observational study with European American and Taiwanese mothers of 3-year-olds. European American mothers drew the child's attention to his or her independent achievements, thereby fostering the child's happiness and pride. In contrast, Taiwanese mothers shamed their children by focusing their attention on their transgressions, and the negative effects these transgressions had had on the mothers (Miller, Fung, & Mintz, 1996). In both cases, parental regulation of the child's attention served to increase the fit between the child's emotional experience and the cultural model.

Although more obvious in child research (for a review, see Diamond & Aspinwall, 2003), emotion regulation by others does not cease in childhood or adolescence; even after children are mature enough to internalize effective emotion regulation strategies of their own, significant social partners continue to contribute regulatory influence across the lifespan. This can be observed in not only adult interpersonal practices such as the provision of comfort and support, the expression of empathy, the redirection of attention, or suggestions for cognitive reframing of emotionally salient information (Diamond & Aspinwall, 2003), but also other common practices of social sharing of emotions in adults (e.g., support groups). In summary, other people may focus a person's

attention in ways that render emotional experience consistent with cultural models of self and relating. Individuals' social environments may be especially likely to help and channel attention when individuals focus on aspects of the situation that render their emotional experience inconsistent with the cultural model.

Regulation of Appraisal

Another major path of emotion regulation is through fashioning the individual's appraisals of the situation. Appraisal always occurs against the backdrop of the cultural models that constitute reality. These cultural models may be internalized, in which case they play a role in individual emotion regulation, or they may be instantiated by the social context, in which case they may activate in the individual certain appraisals; the latter may be seen as a form of social appraisal regulation.

Individual Regulation of Appraisal

Cultural models form the background against which situations are appraised, and emotional experience is contingent on an individual's interpretation of the situation (e.g, Ellsworth, 1994; Mesquita & Ellsworth, 2001). One obvious way in which cultural models of self and relating are essential to emotion regulation is by fashioning the appraisal processes to create certain emotions.

Individual appraisal is fashioned, in the first place, by the concerns and schemas against which situations are appraised. Cultures differ with respect to these concerns. For instance, what it means to a woman that a man—not her intimate—looks at her may differ depending on whether her culture construes such attention as an honor violation, a sign that she is attractive, or a marker that she is being objectified in a sexist world. Receiving male attention has different emotional consequences in each of these cases: A woman would feel an emotion of shame or embarrassment if she lived in an honor culture, an emotion of pride or excitement if she experienced the male attention as a sign of her attractiveness, and an emotion of indignation if she considered it an act of sexism. Depending on the "availability" or "salience" of certain interpretations, the woman would feel a different emotion. We refer to these cultural differences in appraisal as forms of cultural emotion regulation, even if they may not involve attempts at effortful regulation on the part of the individual. The salience of concerns that fit the cultural models is "culture in the head." It is this culture in the head that renders the emotion normative (i.e., "regulates" the emotion).

Differences in daily emotional experiences can often be understood from cultural differences in individuals' common world views or daily preoccupations (also "culture in the head"). For example, the emotion of anger is characterized by appraisals of an event as unpleasant, going against personal goals, but relatively controllable. In trying to understand the near absence of frustration and anger among Tahitians, the American anthropologist Robert Levy (1978, p. 226) pointed to "a shared common sense that individuals have very limited control over nature and over the behavior of others." Thus, as Levy notes, a universe so defined is "*cognitively* less frustrating than those cultures which define realities in which almost anything is possible to individuals" (p. 226). The general expectation that the world is minimally rewarding, and that there is no way to force rewards, leads to a lower prevalence of anger. The Tahitian perspective on life contrasts with middle-class American models that emphasize and value control and predictability (Mesquita & Ellsworth, 2001). In middle-class American contexts, the prevailing view is that the world is malleable. Changing or influencing the environment rather than adjusting to it tends to be a dominant response, reflecting the assumption that circumstances can be made to fit one's personal goals (Boiger, Mesquita, Tsai, & Markus, 2012; Morling, Kitayama, & Miyamoto, 2002; Weisz, Rothbaum, & Blackburn, 1984). Hence, there is a relative emphasis on control and agency in middle-class American appraisals of emotional situations. As such, people more readily appraise events in terms of inconsistency with their goals, the responsibility of other people, and the possibility of forcing others to accommodate to their wishes; hence, the emotional experiences of frustration and anger are more likely (Frijda, 1986). Cultural models thus provide the backdrop against

which events are meaningful. A sense that the world is a place in which agents make a difference also renders the experience of a *lack* of agency more unpleasant. This is illustrated by a study by Roseman, Dhawan, Rettek, Naidu, and Thapa (1995), in which Indian and American college students reported instances of anger, sadness, and fear, and rated their experiences on several appraisal dimensions. Cultural models for Indian college students are less likely to make salient the possibility of successful control of one's circumstances than are cultural models for American students (Savani, Markus, Naidu, Kumar, & Berlia, 2010). Consistent with this understanding, Roseman and colleagues (1995) found that Indian college students rated self-reported emotional events to be less "incongruent with their motives" than did their American counterparts. As expected, Indian students also reported lower overall intensities of both sadness and anger; moreover, these cultural differences in emotion intensity were fully mediated by the perception that the event was less discrepant with goals. Although the link between the cultural emphasis on controllability and the perception of goal relevance was not measured, we suggest that cultural representations of agency affected the perception of a discrepancy between emotional events and personal goals, and in turn influenced the intensity of felt sadness and anger.

A more direct test of the relationship between concerns and emotional experiences was provided by two recent studies of Belgian students (De Leersnyder & Mesquita, 2013), who were asked to describe a recently experienced emotional situation and to indicate *if* and *to what extent* the situation had been either consistent or inconsistent with a number of different concerns, some of which were other-focused (e.g., being loyal, helping others), and others which were self-focused (personal success, ambition). The frequency with which these values were judged as relevant to the emotional situations exactly mirrored young Belgians' value hierarchy (i.e., most important values as "guiding principles in people's life"), as obtained from a national representative sample by the European Social Survey (ESS, Round 5; Norwegian Social Science Data Services, 2012). Moreover, the types of emotions depended on the types of values considered relevant. The combined results thus suggest that (1) culturally salient concerns are more readily available as standards of evaluation for emotional situations, and (2) the different types of concerns translate into different patterns of emotional experience. As such, emotional experiences are culturally regulated to be *about* the most important cultural themes and concerns.

In summary, cultural models appear to provide the backdrop of expectations and point(s) of view with which individuals meet the world. As such, culture regulates the meaning and thus the emotional significance of the world to the individual.

Social Regulation of Appraisal

Other people appear to influence an individual's appraisals in two ways. One is by example: Other people's emotions and appraisals inform an individual's appraisals in a given situation. There is developmental work showing that children look at their caregivers' facial expressions in trying to appraise a situation as, for example, dangerous or safe (e.g., Campos & Stenberg, 1981). Adults also use other people's appraisals as input for the meaning of the situation (Parkinson & Simons, 2009). We expect that social appraisal regulation plays a large role in cultural differences, because the prevalent emotions in each culture differ. When other people's emotions are used as a compass, this compass will point in different directions for different cultural environments. There are some illustrations of this phenomenon in the parenting literature: Parents' own reactions to their children's emotions may sometimes model their children's appraisal. For instance, Japanese parents rarely express direct disagreement with their angry children; instead, they go through cycles of mutual perspective taking (Trommsdorff & Kornadt, 2003) or express negativity indirectly (e.g., by silence). Parents thus model the interpretation of the situation as one that requires perspective taking. In contrast, German parents in the same study (Trommsdorff & Kornadt, 2003) were much more confrontational, and in that way, modeled the appraisal as one of conflict.

A second social appraisal situation consists of more explicit approval or disapproval of certain emotions or interpretations. There

is again some cross-cultural work on social appraisal regulation in the caregiver–child literature (Boiger & Mesquita, 2012; De Leersnyder, Boiger, & Mesquita, 2013). For example, parents may pay attention to some of their children's emotions, thus validating the appraisal of the situation, and ignore other emotions of the child, thus failing to endorse the child's interpretation of the event. German mothers who witnessed their children's mishaps focused on the children's distress, thereby confirming that the children had a good reason for their negative emotions. By contrast, Japanese and Indian mothers ignored their child's negative emotions, thus challenging their interpretation of the situation as one of distress (Trommsdorff & Friedlmeier, 2010). Japanese caregivers also helped their children adopt the outside-in perspective that is also common for Japanese adults: They drew their children's attention to how their anger makes others feel (e.g., Conroy, Hess, Azuma, & Kashiwagi, 1980). Reappraising angering situations in this way may explain the lower levels of anger in Japan (Zahn-Waxler, Radke-Yarrow, & King, 1979). European American parents, on the other hand, expected their children to self-assert and to stand up for themselves (Conroy et al., 1980). When dealing with an angry child, they tended to encourage appraisals of frustration in the child, leading to high levels of anger.

To our knowledge, there is no systematic empirical evidence that adults help each other in reappraising situations in ways that are consistent with their cultural values. However, there is some anecdotal evidence for social appraisal regulation beyond childhood. For instance, Kitayama and Masuda (1995) describe how American friends help each other when one is feeling shameful and down "by reorienting the person's attention . . . to external objects or events the person can reasonably blame for the impeding problem" (p. 220). This social regulation may explain why shame is frequently transformed into anger in the American cultural context (Kitayama & Masuda, 1995). By reappraising the shameful event as being caused by others rather than by oneself, the focus of appraisal shifts from one's own painful shortcomings to the more empowering experience of self-integrity, and others' blameworthiness. Maintaining high self-esteem and avoiding self-critical information constitute central goals for the American independent self; anger can thus be seen as a more desirable endpoint of emotion regulation than shame.

Behavior Regulation

Cross-cultural differences have traditionally been explained as differences in deliberate and effortful suppression of emotional expressions that did not match the cultural ideals (e.g., Matsumoto, 1990). The idea has been that people across different cultural contexts adhere to the prevalent "display rules" (i.e., the "cultural norms that dictate the management and modification of emotional displays depending on social circumstances; Ekman & Friesen, 1969)," and that this type of post hoc regulation is the most important route to cultural differences in emotion (Matsumoto et al., 2008, p. 58). While it is clear that cultural differences in emotion regulation are not due to differences in response suppression only, there is evidence that suggests cultural differences in this strategy, too. We discuss this evidence below, but we also suggest that cultural differences in the regulation of emotional behavior are not limited to differences in individual emotion suppression.

Individual Regulation of Behavior

There is evidence of cultural differences in display rules. Several studies have linked the use of display rules to the culture's level on the individualism dimension; a dimension that is an important aspect of cultural models of self and relationship. Cultures with interdependent models are thought to be low on individualism (and high on collectivism); while independent cultures are high on individualism. Pertinent studies have found that there is a greater desire to express emotions, especially positive, high-activation emotions in cultures higher on individualism. For instance, Matsumoto and colleagues (2008) asked participants from 32 different countries what they *should do* if they felt each of seven emotions (anger, contempt, disgust, fear, happiness, sadness, and surprise) on a scale ranging from emotion amplification (i.e., *express more*), to deamplification (i.e.,

express less) or masking (i.e., *hide emotion with smile*). The authors found that the country's scores on individualism (as indexed by Hofstede, 2001) predicted the country's mean level of emotional expressivity. However, several qualifications of these results are in order.

First, much of the relationship between individualism and expression was "carried by the relationship between individualism with happiness and surprise" (Matsumoto et al., 2008, p. 66). Thus, whereas people in the United States, Canada, and Australia expressed a desire to up-regulate happiness and surprise, people in East Asian countries indicated that they would actively seek to avoid these emotional states (see also Tsai et al., 2006). Second, nearly all of the emotions included in the current study except sadness were high-activation (disengaging) emotions. Since the expression of high-activation disengaged emotions would be more *in*consistent with the prevalent cultural models, there may indeed be a higher *need* to regulate these types of emotions in East Asian than in Western cultural contexts. Consequently, the association between emotion expressivity and individualism may not have been found (or even be in the opposite direction) if only *low*-activation *engaging* emotional experiences had been included—an idea that is supported by the negative (partial) correlation between individualism and emotional expressivity of sadness (Matsumoto et al., 2008). Therefore, rather than concluding that individuals in collectivist cultures suppress their emotions more than individuals in individualist cultures, a better conclusion might be that people across cultural contexts engage in the suppression of *those* emotions that are inconsistent with the pertinent cultural models.

There are also cultural differences in both the effectiveness and costs of emotion suppression (Cheung & Park, 2010; Mauss & Butler, 2010; Soto, Perez, Kim, Lee, & Minnick, 2011) Convergent results from experimental and questionnaire studies show that suppression of emotional responses is effortful in European American samples, and although effective in modifying emotional *behavior*, it does not change the emotional experience. Furthermore, in European American samples, suppression is associated with increases in physiological arousal and with detrimental effects on memory, felt affect (less positive and no less negative emotion), and social relationships (e.g., Gross, 1998; Richards & Gross, 2000). Finally, habitual suppression in European Americans was related to both high levels of depressive symptoms and low levels of life satisfaction (Cheung & Park, 2010; Soto et al., 2011).

In Western cultures, emotional suppression has therefore been found to be maladaptive. However, there are many indications that this is not the case in Asian American or East Asian samples. First, emotion suppression may be more effective in these samples. For example, in a study by Mauss and Butler (2010), Asian Americans who valued emotional control were not only coded as expressing less anger but they also reported *feeling* less angry than before. Moreover, they showed a "challenge pattern" of cardiovascular responding, suggesting that the suppression was not that effortful, because their resources met or exceeded the demands of the situation. Second, habitually engaging in emotionally suppressive behavior was unrelated to psychological well-being in Hong Kong Chinese participants (Soto et al., 2011) and much less associated with depression in Asian American compared to European American respondents (Cheung & Park, 2010).

The different meanings of suppression can be understood from the respective cultural models (see Mesquita & Delvaux, 2012). Whereas the *expression* of one's feeling in independent cultural contexts defines identity, it does not do so in interdependent contexts. These latter contexts emphasize situational adjustment, and may thus encourage suppression of emotions whenever the situation requires it.

Culturally comparative work has focused on up- and down-regulation of emotions but has not paid much attention to the quality of emotional behavior: What do people usually do when they feel a certain emotion? Descriptive, ethnographic work has suggested that individuals in some cultures have specific behavioral scripts associated with certain emotions (Mesquita & Frijda, 1992). These behavioral scripts may be considered the outcome of regulation to the extent that they serve the prevalent cultural models of self and relationships. A good illustration

is the Balinese reaction to socially threatening events, falling asleep (Bateson & Mead, 1942), which can be understood as an expression of the emotion of fear that avoids disrupting the cultural model. Falling asleep satisfies, at least subjectively, the goal of reducing one's exposure to threat, while avoiding the emotional disruption that other fear responses are felt to cause. Therefore, falling asleep can be considered a culturally effective form of individual emotion regulation.

In summary, cultural models implicate display rules for emotion regulation: Individuals tend to suppress emotions that are inconsistent with the cultural models. Emotional suppression is an individual regulation strategy that requires different efforts, that differs in effectiveness and is associated with different psychological costs across different cultural contexts, in ways that can be understood from the cultural models of self and relationship. Furthermore, cultures may model specific behaviors that meet the cultural models. When individuals engage in behavior that is culturally modeled, we call this regulation. We do not think that individuals necessarily suppress another (more natural) response before engaging in culture-specific behavior. Rather, the fact that they choose a culturally valued, rather than a less valued response, makes this a meaningful case of cultural regulation.

Social Regulation of Behavior

Across cultures, the preferred contexts and practices for behavioral expression differ as well. Certain social contexts provide opportunity for emotional expression, whereas other cultural contexts inhibit the display of emotions, or of certain emotions. Rituals are an example: "Rituals consist of prescribed behavior modes that remove the need to expose one's individual feelings. Yet at the same time, they form opportunities to vent one's emotions in a socially acceptable way" (Mesquita & Frijda, 1992, p. 197). A good example of a ritual that appears to provide an institutionalized means for expressing emotional behaviors that would otherwise conflict with the prevailing cultural model was described for the Philippine Ilongots by the anthropologist Michelle Rosaldo. In this small community, in-group harmony was essential for survival of the group. Ilongots, therefore, saw it as a communal responsibility to regulate emotions that could threaten the harmony of the group. *Liget*—an emotion denoting energy, passion, and anger simultaneously—was one of those threatening emotions (Rosaldo, 1980). A ritual of headhunting was in place, should feelings of *liget* arise. When one or more Ilongot men experienced the heavy feeling of *liget*, a group of them would go out to kill an outsider. After the beheading, the Ilongot men came home purged of violence, and the community celebrated the overcoming of *liget* by singing together. The ritual thus directed the behavioral intentions elicited by *liget* toward an out-group target, channeling a potentially intraculturally disruptive behavior into one that reinforced the solidarity of the group. Rituals may thus be understood as prototypes for the mechanisms by which sociocultural practices afford opportunities to express (or act on) emotions in culturally supported ways.

In summary, there are cultural differences in both individual and social behavioral regulation. Some of these are differences in suppression versus expressivity. However, the connotations of suppression are not culturally the same. Other differences are better seen as differences in the selection of behavior, often in the function of the models and practical opportunities for behavior that the culture provides.

Cultural Differences in the Preference for Different Strategies of Emotion Regulation

In a number of studies that have compared cultural preferences for particular types of emotion regulation, the suppression of emotional responses was not contrasted with expression, but rather with reappraisal, a different type of emotion regulation. In different studies, participants from independent cultural contexts expressed a stronger preference for reappraisal over suppression than participants in interdependent cultural contexts, for whom this preference was sometimes even absent (e.g., Novin, Banjeree, Dadhkah, & Rieffe, 2009; Soto et al., 2011). These differential preferences may correspond to different cultural models. In independent cultural contexts, the expres-

sion of one's feeling is identity defining. A change in emotional expression achieved by reappraisal respects the link between feeling and expression. On the other hand, suppression of emotions seems fine in interdependent cultural contexts, where the emphasis is on situational adjustment; suppression and reappraisal would be equivalent means to reaching this goal. Among respondents from independent cultural contexts, the use of suppression is inversely related to the use of reappraisal, suggesting that they more readily use the one *or* the other strategy. In contrast, there is a positive correlation between the use of suppression and reappraisal among respondents from interdependent cultural contexts, suggesting that they use *both* strategies when they attempt to regulate their emotions (Matsumoto et al., 2008).

Conclusion

There is substantial evidence of the cultural regulation of emotions, conceived here as the combined mechanisms that align an individual's emotional experience to the pertinent cultural model. Cultural regulation takes place not only at the level of culture, by way of the habitual social practices and interpersonal interactions that constitute an individual's lived environment, but also at the level of individual schemas, goals, and modes of attention. Moreover, regulation takes place at all the different stages of the emotion: Both cultural and individual processes are instrumental in creating and modifying emotional events, channeling an individual's attention, and shaping an individual's appraisals of the situation in ways that promote culturally desirable emotions; moreover, both cultural and individual processes shape emotion expression into congruence with cultural norms. Cultural practices and psychological tendencies thus co-constitute emotional experiences and expressions that are aligned with prevailing cultural models of self and relating.

At the level of the individual, emotion regulation is often implicit and automatic, although it *can* also be conscious and effortful. When it is automatic, it can be conceived in terms of psychological tendencies: the tendency to engage in certain types of interactions, to focus attention on certain aspects of the situation, and to appraise a situation in a certain way. These psychological tendencies may originate from culture-level regulation. Thus, individuals' tendencies to experience certain emotions may be a consequence of repeated exposure to a cultural set of daily situations (see Kitayama & Imada, 2010). For instance, a habitual focus on controllability may chronically increase the propensity to appraise a situation as one of high control, and thus of experiencing anger. In the same way, caregivers' repeated emphasis on the child's inability to meet social standards may bias the child's focus toward noticing failures, thus enhancing the tendency to feel shame. The resulting regulation is *implicit* and *antecedent-focused*.

The large cultural differences observed in everyday emotional experience suggest that emotion regulation is widespread. However, given the functional role of emotions in guiding adaptive behavior, we contend that effortful emotion regulation must be very limited; it is "culture's last resort" for shaping emotions in a culturally normative fashion, used only when all other means have failed. Most regulatory processes must be automatic, as in the culturally different psychological tendencies that align an individual's experience and expression with the cultural models.

The cultural perspective contributes several insights to emotion regulation generally. First, it suggests that while the goal of emotion regulation may universally be proper self-presentation and relationship maintenance, specific cultural models define the norms for self-presentation, as well as the ways to maintain a relationship. Cultural models thus set specific goals for emotion regulation. In this way, emotion regulation ties a person to the most important cultural goals and values, just as it ties a person to his or her personality (e.g., Tamir, 2005).

Second, the cultural perspective suggests that emotion regulation is not merely an *intrapersonal* process. Rather, emotions are importantly regulated by the ways in which our worlds are structured and our lives are organized. We have illustrated how emotion is regulated at the level of cultural practices through the structuring of social situations and the dynamics of social interactions; close others' attempts to modify the indi-

vidual's focus of attention within a situation; and the opportunities afforded for emotional behavior. The structure of everyday life can be seen as the primary way in which emotions are culturally regulated, although at this level the distinction between emotion generation and emotion regulation is mute (see also Kappas, 2011; Mesquita & Frijda, 2011).

Taken together, the results suggest that there is "redundancy" in the cultural regulation of emotions (Levy, 1978), such that regulatory processes at many different loci of the emotion, and as initiated by many different sources, converge into a pattern of emotional experience and expression that fits the cultural model (more than not).

References

Abu-Lughod, L. (1986). *Veiled sentiments*. Berkeley: University of California Press.

Adams, G., & Markus, H. R. (2004). Toward a conception of culture suitable for a social psychology of culture. In M. Schaller & C. S. Crandall (Eds.), *Psychological foundations of culture* (pp. 335–360). Mahwah, NJ: Erlbaum.

Bargh, J. A., & Williams, L. E. (2007). The nonconscious regulation of emotion. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 429–445). New York: Guilford Press.

Bateson, G., & Mead, M. (1942). *Balinese character: A photographic analysis*. New York: New York Academy of Sciences.

Benedict, R. (1946). *The chrysanthemum and the sword. Patterns of Japanese culture*. Boston: Houghton Mifflin.

Boiger, M., & Mesquita, B. (2012). The construction of emotion in interactions, relationships, and cultures. *Emotion Review, 4*, 221–229.

Boiger, M., Mesquita, B., Tsai, A., & Markus, H. (2012). Influencing and adjusting in daily emotional situations: A comparison of European and Asian American action styles. *Cognition and Emotion, 26*, 332–340.

Boiger, M., Mesquita, B., Uchida, Y., & Barrett, L. F. (2013). Condoned or condemned: The situational affordance of anger and shame in Japan and the US. *Personality and Social Psychology Bulletin, 39*(4), 540–553.

Bruner, J. (1990). *Acts of meaning*. Cambridge, MA: Harvard University Press.

Campos, J. J., & Stenberg, C. (1981). Perception, appraisal, and emotion: The onset of social referencing. In M. E. Lamb & L. R. Sherrod (Eds.), *Infant social cognition: Empirical and theoretical considerations* (pp. 273–314). Hillsdale, NJ: Erlbaum.

Cheung, R. Y. M., & Park, I. J. K. (2010). Anger suppression, interdependent self-construal, and depression among Asian American and European American college students. *Cultural Diversity and Ethnic Minority, 16*(4), 517–525.

Cohen, D., Hoshino-Browne, E., & Leung, A. K.-Y. (2007). Culture and the structure of personal experience: Insider and outsider phenomenologies of the self and social world. In M. P. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 39, pp. 1–67). San Diego, CA: Academic Press.

Cole, P. M., Tamang, B. L., & Shrestha, S. (2006). Cultural variations in the socialization of young children's anger and shame. *Child Development, 77*, 1237–1251.

Conroy, M., Hess, R. D., Azuma, H., & Kashiwagi, K. (1980). Maternal strategies for regulating children's behavior: Japanese and American families. *Journal of Cross-Cultural Psychology, 11*, 153–172.

D'Andrade, R. G. (1984). Culture meaning systems. In R. A. Shweder & R. A. Levine (Eds.), *Culture theory: Essays on mind, self, and emotion* (pp. 88–119). Cambridge, UK: Cambridge University Press.

De Leersnyder, J., Boiger, M., & Mesquita, B. (2013). Cultural regulation of emotion: Individual, relational, and structural sources. *Frontiers in Psychology, 4*(55). [E-publication prior to print]

De Leersnyder, J., & Mesquita, B. (2013). *What are emotions about?: The role of cultural values in emotional experience*. Manuscript in preparation.

De Leersnyder, J., Mesquita, B., & Kim, H. (2011). Where do my emotions belong?: A study of immigrants' emotional acculturation. *Personality and Social Psychology Bulletin, 37*, 451–463.

De Leersnyder, J., Mesquita, B., & Kim, H., (2013). Emotional acculturation. In D. Hermans, B. Mesquita, & B. Rime (Eds.), *Changing emotions* (pp. 127–133). Hove, UK: Psychology Press.

De Leersnyder, J., Mesquita, B., & Kim, H. (2013). *Emotional fit with culture: A prediction of individual differences in relational well-being*. Manuscript under review.

Diamond, L. M., & Aspinwall, L. G. (2003).

Emotion regulation across the life span: An integrative perspective emphasizing self-regulation, positive affect, and dyadic processes. *Motivation and Emotion, 27*, 125–156.

Eid, M., & Diener, E. (2001). Norms for experiencing emotions in different cultures: Inter- and intranational differences. *Journal of Personality and Social Psychology, 81*(5), 869–885.

Ekman, P., & Friesen, W. V. (1969). The repertoire of nonverbal behavior: Categories, origins, usage, and coding. *Semiotica, 1*, 49–98.

Elliott, A., Chirkov, V., Kim, Y., & Sheldon, K. (2001). A cross-cultural analysis of avoidance (relative to approach) personal goals. *Psychological Science, 12*, 505–510.

Ellsworth, P. C. (1994). Sense, culture, and sensibility. In S. Kitayama & H. R. Markus (Eds.), *Emotion and culture: Empirical studies of mutual influence* (pp. 23–50). Washington, DC: American Psychological Association.

ESS Round 5: European Social Survey Round 5 Data. (2012). *Data file edition 3.0*. Norway: Norwegian Social Science Data Services, data archive and distributor of ESS data.

Frijda, N. H. (1986). *The emotions*. Cambridge, UK: Cambridge University Press.

Frijda, N. H., Kuipers, P., & Terschure, E. (1989). Relations between emotion, appraisal and emotional action readiness. *Journal of Personality and Social Psychology, 57*, 212–228.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*, 271–299.

Gross, J. J., Sheppes, G., & Urry, H. L. (2011). Emotion generation and emotion regulation: A distinction we should make (carefully). *Cognition and Emotion, 25*, 765–781.

Grossmann, I., Ellsworth, P. C., & Hong, Y. (2012). Culture, attention, and emotion. *Journal of Experimental Psychology: General, 14*(1), 31–36.

Gyurak, A., Gross, J. J., & Etkin, A. (2011). Explicit and implicit emotion regulation: A dual-process framework. *Cognition and Emotion, 25*(3), 400–412.

Heine, S. J., Lehman, D. R., Markus, H. R., & Kitayama, S. (1999). Is there a universal need for positive self-regard? *Psychological Review, 106*(4), 766–794.

Hochschild, J. L. (1995). What is the American dream? In J. L. Hochschild (Ed.), *Facing up to the American dream: Race, class and the soul of the nation* (pp. 15–38). Princeton, NJ: Princeton University Press.

Hofstede, G. H. (2001). *Culture's consequences: Comparing values, behaviors, institutions and organizations across nations* (2nd ed.). Thousand Oaks, CA: Sage.

Kappas, A. (2011). Emotion and regulation are one! *Emotion Review, 3*, 17–25.

Kim, H., & Markus, H. R. (1999). Deviance or uniqueness, harmony or conformity?: A cultural analysis. *Journal of Personality and Social Psychology, 77*(4), 785–800.

Kitayama, S., & Imada, T. (2010). Implicit independence and interdependence: A cultural task analysis. In B. Mesquita, L. F. Barrett, & E. R. Smith (Eds.), *The mind in context* (pp. 174–200). New York: Guilford Press.

Kitayama, S., Karasawa, M., & Mesquita, B. (2004). Collective and personal processes in regulating emotions: Emotion and self in Japan and the US. In P. Philippot & R. S. Feldman (Eds.), *The regulation of emotion* (pp. 251–273). Hillsdale, NJ: Erlbaum.

Kitayama, S., & Markus, H. R. (2000). The pursuit of happiness and the realization of sympathy: Cultural patterns of self, social relations, and well-being. In E. Diener & E. Suh (Eds.), *Subjective well-being across cultures* (pp. 113–161). Cambridge, MA: MIT Press.

Kitayama, S., & Masuda, T. (1995). Reappraising cognitive appraisal from a cultural perspective. *Psychological Inquiry, 6*, 217–223.

Kitayama, S., Mesquita, B., & Karasawa, M. (2006). The emotional basis of independent and interdependent selves: Socially disengaging and engaging emotions in the US and Japan. *Journal of Personality and Social Psychology, 91*(5), 890–903.

Koole, S. L. (2009). The psychology of emotion regulation: An integrative review. *Cognition and Emotion, 23*(1), 4–41.

Lebra, T. S. (1992). Self in Japanese culture. In N. E. Rosenberger (Ed.), *Japanese sense of self*. New York: Oxford University Press.

Lee, A. Y., Aaker, J. L., & Gardner, W. L. (2000). The pleasures and pains of distinct self-construals: The role of interdependence in regulatory focus. *Journal of Personality and Social Psychology, 78*(6), 1122–1134.

Levy, R. I. (1978). Tahitian gentleness and redundant controls. In A. Montagu (Ed.), *Learning non-aggression: The experience of non-literate societies* (pp. 222–235). New York: Oxford University Press.

Lewis, C. C. (1995). *Educating hearts and minds*. New York: Cambridge University Press.

Lutz, C. (1987). Goals, events, and understand-

ing in Ifaluk emotion theory. In N. Quinn & D. Holland (Eds.), *Cultural models in language and thought* (pp. 290–312). New York: Cambridge University Press.

Markus, H. R., & Kitayama, S. (1991). Culture and self: Implications for cognition, emotion, and motivation. *Psychological Review, 98*(2), 224–253.

Markus, H. R., & Kitayama, S. (1994). The cultural construction of self and emotion: Implications for social behavior. In S. Kitayama & H. R. Markus (Eds.), *Emotion and culture: Empirical studies of mutual influence* (pp. 89–130). Washington, DC: American Psychological Association.

Masuda, T., Ellsworth, P. C., Mesquita, B., Leu, J., Tanida, S., & Veerdonk, E. (2008). Placing the face in context: Cultural differences in the perception of facial emotion. *Journal of Personality and Social Psychology, 94*(3), 365–381.

Matsumoto, D. (1990). Cultural similarities and differences in display rules. *Motivation and Emotion, 14*(3), 195–214.

Matsumoto, D., Yoo, S. H., Fontaine, J., Anguas-Wong, A. M., Arriola, M., & Ataca, B. (2008). Mapping expressive differences around the world: The relationship between emotional display rules and individualism versus collectivism. *Journal of Cross-Cultural Psychology, 39*(1), 55–74.

Mauss, I. B., Bunge, S. A., & Gross, J. J. (2008). Culture and automatic emotion regulation. In S. Ismer, S. Jung, S. Kronast, C. van Scheve, & M. Vanderkerckhove (Eds.), *Regulating emotions: Culture, social necessity and biological inheritance* (pp. 39–60). London: Blackwell.

Mauss, I. B., & Butler, E. A. (2010). Cultural context moderates the relationship between emotion control values and cardiovascular challenge versus threat responses. *Biological Psychology, 84*, 521–530.

Mesquita, B., & Delvaux, E. (2012). A cultural perspective on emotion labor. In A. Grandey, J. Diefendorff, & D. Rupp (Eds.), *Emotional labor in the 21st century: Diverse perspectives on emotion regulation at work* (pp. 251–272). New York: Psychology Press/Routledge.

Mesquita, B., & Ellsworth, P. C. (2001). The role of culture in appraisal. In K. R. Scherer & A. Schorr (Eds.), *Appraisal processes in emotion: Theory, methods, research* (pp. 233–248). New York: Oxford University Press.

Mesquita, B., & Frijda, N. H. (1992). Cultural variations in emotions: A review. *Psychological Bulletin, 112*(2), 179–204.

Mesquita, B., & Frijda, N. H. (2011). An emotion perspective on emotion regulation. *Cognition and Emotion, 25*, 782–784.

Mesquita, B., & Leu, J. (2007) The cultural psychology of emotions. In S. Kitayama & D. Cohen (Eds.), *Handbook for cultural psychology* (pp. 734–759). New York: Guilford Press.

Mesquita, B., Marinetti, C., & Delvaux, E. (2012). The social psychology of emotions. In S. Fiske & N. Macrae (Eds.), *Sage handbook of social cognition* (pp. 290–310). Thousand Oaks, CA: Sage.

Miller, P. J., Fung, H., & Mintz, J. (1996). Self-construction through narrative practices: A Chinese and American comparison of early socialization. *Ethos, 24*(2), 237–280.

Morelli, G. A., Rogoff, B., Oppenheim, D., & Goldsmith, D. (1992). Cultural variation in infants' sleeping arrangements: Questions of independence. *Developmental Psychology, 28*(4), 604–613.

Morling, B., Kitayama, S., & Miyamoto, Y. (2002). Cultural practices emphasize influence in the United States and adjustment in Japan. *Personality and Social Psychology Bulletin, 28*(3), 311–323.

Nisbett, R. E. (2003). *The geography of thought. How Asians and Westerners think differently . . . and why.* New York: Free Press.

Novin, S., Banjeree, R., Dadhkah, A., & Rieffe, C. (2009). Self-reported use of display use in the Netherlands and Iran: Evidence for sociocultural influence. *Social Development, 18*(2), 397–411.

Parkinson, B., & Simons, G. (2009). Affecting others: Social appraisal and emotion contagion in everyday decision making. *Personality and Social Psychology Bulletin, 35*, 1071–1084.

Richards, J. M., & Gross, J. J. (2000). Emotion regulation and memory: The cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology, 79*, 410–424.

Rosaldo, M. Z. (1980). *Knowledge and passion: Ilongot notions of self and social life.* New York: Cambridge University Press.

Roseman, I., Dhawan, N., Rettek, S., Naidu, R. K., & Thapa, K. (1995). Cultural differences and cross-cultural similarities in appraisals and emotional responses. *Journal of Cross-Cultural Psychology, 26*(1), 23–48.

Rothbaum, F., Pott, M., Azuma, H., Miyake, K., & Weisz, J. (2000). The development of close relationships in Japan and the United

States: Paths of symbiotic harmony and generative tension. *Child Development, 71*(5), 1121–1142.

Savani, K., Markus, H. R., Naidu, N. V. R., Kumar, S., & Berlia, N. (2010). What counts as a choice?: U.S. Americans are more likely than Indians to construe actions as choices. *Psychological Science, 21*, 391–398.

Shweder, R. A. (1991). *Thinking through cultures*. Cambridge, MA: Harvard University Press.

Solomon, R. C. (2004). *Thinking about feeling: Contemporary philosophers on emotions*. New York: Oxford University Press.

Soto, J. A., Perez, C. R., Kim, Y.-H., Lee, E. A., & Minnick, M. R. (2011). Is expressive suppression always associated with poorer psychological functioning?: A cross-cultural comparison between European Americans and Hong Kong Chinese. *Emotion, 11*(6), 1450–1455.

Stein, N. L., Trabasso, T., & Liwag, M. (1993). The representation and organization of emotional experience: Unfolding the emotion episode. In M. Lewis & J. M. Haviland (Eds.), *Handbook of emotions* (pp. 279–300). New York: Guilford Press.

Tamir, M. (2005). Don't worry, be happy?: Neuroticism, trait-consistent affect regulation, and performance. *Journal of Personality and Social Psychology, 89*, 449–461.

Trommsdorff, G., & Friedlmeier, W. (2010). Preschool girls' distress and mothers' sensitivity in Japan and Germany. *European Journal of Developmental Psychology, 7*, 350–370.

Trommsdorff, G., & Kornadt, H. (2003). Parent–child relations in cross-cultural perspective. In L. Kuczynski (Ed.), *Handbook of dynamics in parent–child relations* (pp. 271–305). Thousand Oaks, CA: Sage.

Tsai, J. L., Knutson, B., & Fung, H. H. (2006). Cultural variation in affect valuation. *Journal of Personality and Social Psychology, 90*, 288–307.

Tsai, J. L., Miao, F., Seppala, E., Fung, H. H., & Yeung, D. (2007). Influence and adjustment goals: Mediators of cultural differences in ideal affect. *Journal of Personality and Social Psychology, 92*(6), 1102–1117.

Weisz, J. R., Rothbaum, F. M., & Blackburn, T. C. (1984). Standing out and standing in: The psychology of control in America and Japan. *American Psychologist, 39*(9), 955–969.

Wierzbicka, A. (1994). Emotion, language, and cultural scripts. In S. Kitayama & H. R. Markus (Eds.), *Emotion and culture: Empirical studies of mutual influence* (pp. 133–196). Washington, DC: American Psychological Association.

Zahn-Waxler, C., Radke-Yarrow, M., & King, R. A. (1979). Child rearing and children's prosocial initiations toward victims of distress. *Child Development, 50*, 319–330.

PART VI

PERSONALITY PROCESSES AND INDIVIDUAL DIFFERENCES

CHAPTER 19

Temperament and Emotion Regulation

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In recent years, advances in research on temperament have contributed to many other areas of psychology, including socioemotional and personality development, psychopathology, emotion, and emotion regulation (Zentner & Shiner, 2012). *Temperament* refers to constitutionally based individual differences in reactivity and self-regulation, and historically, has been linked to the biology of the person (Rothbart, 2011). Recent temperament research has employed increasingly sophisticated psychometric methods, developing a basic taxonomy of temperament that extends from infancy to adulthood and links temperament to neural anatomy and function.

In our research, broad factors of Fear, Frustration, Negative Affectivity, Extraversion/Surgency, Affiliativeness, Orienting/Perceptual Sensitivity, and Effortful Control have been extracted from questionnaire data (Putnam, Ellis, & Rothbart, 2001; Rothbart, 2011). These dimensions show strong similarities to those found in other laboratories, and reactive and self-regulative aspects of temperament have been observed in both the laboratory and in parent and self-report data (Zentner & Shiner, 2012).

In this chapter we describe concepts of emotion and emotion regulation within a temperament framework. We first put forward definitions of temperament, personality, emotion, and emotion regulation. We then consider how defense and approach emotion systems regulate each other, followed by a review of links between emotion regulation and individual differences in orienting. We then examine in detail the relation of emotion regulation to temperamental effortful control (EC) and describe the underpinnings of EC in the brain's executive attention network. Because temperament develops, and because EC provides a general mechanism for the control of emotion that is not well-developed until early childhood (Rueda, 2012), we describe emotion regulation in a developmental context. Finally, we raise empirical issues about the study of emotional reactivity and regulation, and describe future directions for research.

Definitions of Key Terms

Rothbart and Bates (2006) define *temperament* as "constitutionally based individual differences in reactivity and self-regulation, in the domains of affect, activity, and attention." This chapter focuses on the temperament domains of affect and attention, and the term *affect* is used synonymously with *emotion*. The term *constitutional* indicates that temperament is biologically based and influenced over time by heredity, matura-

tion, and experience. *Reactivity* and *self-regulation* are terms originally used broadly by Rothbart and Derryberry (1981) to organize the temperament domain. *Reactivity* refers to responses to change in the external and internal environment, including reactions that can be conceptualized broadly or narrowly (e.g., fear, motor activity, orienting, negative affectivity, cardiac reactivity). Reactivity is measured in terms of the latency, duration, and intensity of affective, motor, and orienting reactions (Rothbart & Derryberry, 1981). *Self-regulation* involves effortful attention that serves to modulate reactivity and organize change.

Individual differences in temperament fall within the more general rubric of personality. We define *personality* as individual differences in dispositional traits, coping strategies, and cognitions of self and others that influence the person's adjustments to the environment. *Dispositional traits* are patterns of thoughts, emotions, and behavior that show consistency across situations and stability over time (Allport, 1937), and *temperament traits* constitute a subset of personality traits (Rothbart, 2011). Temperament traits include consistent patterns of reactivity of the emotions and the self-regulation of thoughts, emotions, and action through attention. We note that this definition relates temperament to personality and at the same time differentiates temperament from other personality domains.

Personality goes beyond temperament to include attitudes, cognitive coping strategies, concepts of self and other, values, morals, and beliefs, but there is also evidence that the broad personality traits of Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Openness may be organized around the basic temperament traits that can be observed early in life (McCrae et al., 2000; Rothbart, 2011; Shiner & Caspi, 2012).

Emotions can be seen as broadly integrative systems that order feelings, thoughts, and actions (LeDoux, 1989, 2000). They represent the output of neural information-processing networks that assess the meaning or affective significance of events for the individual. Whereas object recognition systems and spatial processing systems address the questions "What is it?" and "Where is it?," emotion-processing networks address the questions "Is it good for me?"; "Is it bad for me?"; and "What shall I do about it?" (action tendencies). These networks have been evolutionarily conserved to allow the organism to deal with environmental and internally generated threat and opportunity. When we discuss temperamental reactivity, we include individual differences in emotion-processing networks. Because the emotions include both action tendencies and physiological support for these tendencies, they have regulatory aspects, and one emotion can regulate another. Thus, fear potentiates withdrawal, attack, or inhibition, and positive affectivity potentiates the speed and energy of approach.

By *emotion regulation*, we mean the modulation of a given emotional reaction, including its inhibition, activation, or graded modulation. *Down-* and *up-regulation* refer to general reductions or increases in the activation of emotion-related neural networks. Emotion regulation includes attentional strategies employed through effortful control (EC), as well as the modulating effects of other emotions and orienting. Orienting early in life is chiefly reactive, and when distractors are presented to infants, orienting modulates the expression of the infants' emotions (Harman, Rothbart, & Posner, 1997). Later, orienting comes more under the control of executive attention. A purer form of *self-regulation* is seen in the executive attentional processes underlying EC that regulate reactivity (Posner & Rothbart, 2007a, 2007b).

Thus when we consider individual differences in temperament, we are dealing with a dynamic balance between emotional tendencies, and between emotions and attention. Again, it is important to recognize that emotion regulation strategies go far beyond temperament to reflect cognitive and experience-dependent behavioral strategies, although temperamental characteristics are likely to be involved in their development (Rueda, 2012).

Temperament and Emotion–Motivation

Emotion, cognition, and behavior are organized around the goals of the organism (Campos, Caplovitz, Lamb, Goldsmith, &

Stenberg, 1983; Derryberry & Rothbart, 1997). These goals have been evolutionarily conserved in the nervous system but are further programmed by the person's specific experiences, plans, and effortful actions. Affective–motivational systems of emotion evaluate goal-relevant situations and organize goal-appropriate behavior. Based on a review of the literature, Derryberry and Rothbart (1997) described temperament systems that include emotional components. These include systems that support defense and fear, approach and appetitive behaviors, and nurturance/affiliation. Here, we concentrate on the first two of these, the defense and approach systems.

Defense and Approach Systems

The term *defense* describes a system of brain networks that serve to avoid harm by promoting organized responses to immediate and long-term threats (Derryberry & Rothbart, 1997). Portions of the lateral and central amygdala appear to be key neural structures in the functioning of this network. Fear, anxiety, and defensive anger are emotions that reflect activation of the defense system. These emotions are related to behavioral tendencies to inhibition or withdrawal from active threats and avoidance of potentially threatening situations in a flight response. When withdrawal from severe threats is blocked, however, the defense system also serves to promote defensive aggression and a fight response. Systems analogous to the defense system are common to many psychobiological approaches to emotion, including LeDoux's (1989) fear system and Gray's behavioral inhibition system (Gray & McNaughton, 1996; see review by Zuckerman, 2012).

Fear and anxiety are aversive states commonly associated with various forms of dysfunction and pathology in the clinical literature (Rothbart & Posner, 2006). Consequently, it might be expected that individuals exhibiting high levels of fear would be more prone to pathology than those exhibiting lower levels, and there is some evidence that this is the case (Rothbart & Bates, 2006). A psychobiological approach, however, notes that fear and anxiety are evolutionarily conserved to promote potentially adaptive behavior when signals of danger are detected, so very low levels of fear and anxiety can also be problematic. There is a need for some level of fear and anxiety to promote adaptive patterns of responding, while under- or overactivation of the system may promote maladaptive responding. Although all negative emotions contribute to general negative affectivity or stress proneness, we have also found it useful to differentiate anger from fear (Rothbart, 2011; Rothbart & Bates, 2006). Anger is positively related to the approach (Extraversion/Surgency) system described next, and it is particularly linked to the development of externalizing problems (Rothbart, 2011; Rothbart & Bates, 2006).

An *approach system* serves the goal of resource acquisition by promoting organized responses to potential rewards (Derryberry & Rothbart, 1997). Dopaminergic neurons appear to form the core of this network, which also extends to the basolateral amgydala, ventral tegmental area, and the nucleus accumbens. Positive emotional states, such as joy and elation, as well as feelings of eager anticipation, are thought to reflect the broad alterations in neurological and physiological responding that occur in the activation of approach. These serve to bias responding toward positive emotion and reward acquisition, and can be seen in behavioral tendencies to seek out and approach rewards and attentional sensitivity to rewards (Derryberry & Reed, 1994). When goals are blocked, anger and possibly aggression may result (see review by Deater-Deckard & Wang, 2012).

Co-Occuring Approach and Defense Reactions

When situations present potential reward along with risk, both approach and defense systems can become active and may either cooperate or compete to influence processing. In some networks, there appears to be localized competition affecting specific cortical and subcortical processes in a winner-take-all process, in which the "stronger" system dominates (Norman & Shallice, 1986). However, over time, activation strength may wax and wane, leading to an alternation of opposing behaviors or to the appearance of disorganized or inconsistent responding. In our laboratory, we have seen such opposing behaviors in infants, who show abrupt

alternation between approach-related and withdrawal-related behaviors, such as smiling directly followed by distress in response to potentially threatening stimuli.

Inhibitory influences of defense and approach systems may not be symmetrical. Instead, defense systems appear to have preferential status. Observation of a normative "negativity bias" in humans and other animal species (e.g., Ito & Cacioppo, 2005) suggests that defense tends to dominate approach in situations that present both risk and reward. While the balance may be usually skewed toward defense, this balance may not characterize every individual. Instead, individuals will differ in the degree to which they exhibit a negativity bias based on temperament and life experience (Ito & Cacioppo, 2005).

Aspects of the approach system, including sociability, smiling and laughter, and approaching novel objects, can be observed by 3 months of age, and show normative increases over the first year of life (Rothbart, Derryberry, & Hershey, 2000). Late in the first year, some infants also begin to demonstrate fear and related behavioral inhibition of approach and positive emotion in response to unfamiliar and intense stimuli (Rothbart, 1988), and fearful inhibition shows considerable longitudinal stability across childhood and into adolescence (Kagan & Fox, 2006). In our longitudinal research, infant fear was assessed by inhibited approach and negative emotion in the laboratory, and it predicted childhood fear, sadness, and shyness at 7 years of age (Rothbart et al., 2000). Fear also predicted lower approach, impulsivity, and aggression. These findings suggest that fear is involved in the regulation of both approach and aggressive tendencies (as argued by Gray & McNaughton, 1996).

More fearful infants also showed greater empathy, guilt, and shame in childhood (Rothbart, Ahadi, & Hershey, 1994). These findings suggested that fear might be involved in the early development of moral motivation, and Kochanska (1997) has found that temperamental fearfulness predicts conscience development in preschool-age children. Extreme fear, on the other hand, may lead to problems through children's rigid control of their own behavior, as in Block's (2002) description of overcontrolled responses that can limit children's positive experiences. Thus, although fear and its component inhibition allows the first major control system over approach, it is a reactive one that can lack flexibility.

Other temperament emotion systems also regulate each other. For example, affiliativeness is related to lower anger/aggression, and affiliativeness is also related to higher internalizing negative affect, including sadness (Rothbart, 2011). In this chapter, however, we focus on individual differences in developing networks of attention and emotion regulation. We begin with individual differences in orienting.

Attention and Emotion Regulation

Orienting

Much of the early life of the infant is concerned with the regulation of state, including regulation of distress (Rothbart, 2011). Orienting, that is, the selection of information from sensory input (Posner, 2012), is a major mechanism for this regulation (Posner, Rothbart, Sheese, & Voelker, 2012). Caregivers report how they use attention to regulate the state of the infant, distracting their infants by bringing their attention to other stimuli. As infants orient, they are often quieted, and their distress appears to diminish.

We have studied orienting and soothing in 3- to 6-month-old infants (Harman, Rothbart, & Posner, 1997). Infants were first shown a sound and light display that led to distress in about 50% of the infants. These infants strongly oriented to interesting visual and auditory soothing events when they were presented, and during their orienting, facial and vocal signs of distress disappeared. As soon as the orienting stopped (e.g., when the object was removed), however, infant distress returned to almost exactly the level shown prior to presentation of the distractor. An internal system, which we termed the "distress keeper," appeared to hold a computation of the initial level of distress, so that it returned if the infant's orientation to the novel event was lost. In later studies, we found that infants could be quieted by distraction for as long as 1 minute, without changing the eventual level of distress reached once the orienting had ended (Harman et al., 1997).

For young infants, the control of orienting is at first largely in the hands of caregiver presentations. By 4 months, however, infants have gained considerable control over disengaging their gaze from one visual location and moving it to another, and greater orienting skill in the laboratory has been associated with lower parent-reported negative emotion and greater soothability (Johnson, Posner, & Rothbart, 1991). Sustained attention, as indexed by engagement with low-intensity toys (blocks) at 9 months, is also predictive of observed effortful control at 22 months (Kochanska, Murray, & Harlan, 2000). Difficulty with sustaining attention at 9 months predicts behavioral inhibition in toddlers and social difficulties in adolescents (Pérez-Edgar et al., 2010), and Crockenberg, Leerkes, & Barrig (2008) found that infants who focused more on frustrating stimuli at 6 months of age were more aggressive as toddlers.

We observed a number of changes in emotion regulation in longitudinal observations of infants between 3 and 13 months (Rothbart, Ziaie, & O'Boyle, 1992). Older infants increasingly looked to their mothers during presentation of arousing stimuli such as masks and unpredictable mechanical toys. Infants' disengagement of attention from arousing stimuli was also related to lower levels of negative affect in the laboratory at 13 months. Stability from 10 to 13 months was found in infants' use of attentional disengagement, mouthing, hand to mouth (e.g., thumb sucking), approach, and withdrawing the hand, suggesting that some of the infants' regulatory strategies were becoming habitual. Over the period from 3 to 13 months, passive self-soothing decreased and more active approach, attack on the object, and body self-stimulation increased. Infants who showed the greatest distress at 3 months, however, tended to persist in an early form of regulation, self-soothing. Once a mechanism for emotion regulation develops, it may persist because it has brought relief, even though more sophisticated emotion regulation mechanisms are now available, an important consideration in developmental approaches to social relationships and clinical problems (Rothbart, 2011).

Other studies have found direct links between infants' disengagement of attention and decreases in their concurrent negative emotion. In one study of 7-month-olds, parent reports of infants' orienting in daily life were related to higher levels of positive emotionality and lower levels of negative emotionality (Sheese, Voelker, Posner, & Rothbart, 2009). Early mechanisms for coping with negative emotion may also later be transferred to the control of cognition and behavior (Posner & Rothbart, 1998). Correlations have been found, for example, between infants' use of regulatory strategies in anger-inducing situations and their preschool ability to delay responses (Calkins & Williford, 2003). In research by Mischel and his colleagues (Sethi, Mischel, Aber, Shoda, & Rodriguez, 2000), toddlers' use of distraction strategies in an arousing situation was positively related to their later delay of gratification at age 5.

Activation of defense or approach may also alter orienting and the processing of sensory information (LeDoux, 2000). Selection and attention to emotion-related stimuli may be critically important in situations related to the health and well-being of the individual. This selection process can be viewed as involving multiple potential targets in competition for attention. Orienting of attention can produce a bias toward one or more of these targets, and emotion is related to attentional bias (see review by Kanske & Kotz, 2012). For example, more threatening stimuli are associated with greater vigilance in a probe detection task (Beaver, Mogg, & Bradley, 2005), and presentation of aversive stimuli is associated with increased interference during mathematical problem-solving and line detection tasks (Schimmack, 2005).

The effect of emotions on perceptual processes varies as temperament systems develop and differs across individuals, reflecting each person's pattern of temperamental reactivity. In adults, negative emotionality, neuroticism, and trait anxiety have been related to biases in patterns of looking to various kinds of threatening stimuli (see review by Derryberry & Reed, 2002), as well as to patterns of attention toward detecting errors (Paulus, Feinsten, Simmons, & Stein, 2004). Neuroticism has also been linked to difficulty in disengaging attention from sources of threat, and extraversion is related to similar difficulties in disengaging from rewarding stimuli (Derryberry & Reed, 2002).

Initial orienting to threatening or rewarding stimuli is often accompanied by behavioral quieting and muted physiological responding, including cardiac deceleration, but orienting may be quickly followed by an action phase involving behaviors such as approach, fight, or flight that require dramatic changes in metabolic functioning. The modulation of autonomic arousal in preparation for behavioral responding is another mechanism through which defense and approach systems can influence responding. Relevant here are changes in skin conductance, heart and respiration rate, blood pressure and patterns of blood circulation, potentiation of the startle response, and secretion of cortisol and catecholamines (Bradley, Codispoti, Cuthbert, & Lang, 2001).

We now turn to attention processes underlying the development of EC, a flexible and attention-based form of self-regulation of thought, action, and emotion. We focus particularly on this topic because of its importance to behavior and emotion regulation, its susceptibility to training, and the many recent research advances in this area.

EC and Executive Attention

EC is defined as the ability to inhibit a dominant response so as to activate a subdominant response (Rothbart & Bates, 2006). EC initially emerged from psychometric temperament research as a higher-order factor that included lower-order scales of attentional focusing, inhibitory control, perceptual sensitivity, and low-intensity pleasure in childhood (Rothbart, 2011; Rothbart, Ahadi, Hershey, & Fisher, 2001), and attentional control, inhibitory control, and activational control in adulthood (Evans & Rothbart, 2007). We have proposed that EC describes individual differences in self-regulatory capacities linked to the functioning of the executive attention network (Posner & Rothbart, 1998, 2007a, 2007b).

EC predicts a broad array of outcomes, including the development of higher prosocial and lower antisocial behavior (Lengua & Wachs, 2012; Rueda, 2012). The direct regulation of negative emotional reactions, such as anger, anxiety, or fear, represents one avenue through which EC can influence behavior. It has been argued that individuals with higher EC are better able to regulate the affective responses that promote antisocial behavior (e.g., Kochanska et al., 2000). EC has also been positively linked to empathy and prosocial behavior (Rothbart & Bates, 2006), and it interacts with contextual variables to predict behaviors linked to emotion regulation. For example, early temperament interacts with parenting practices to predict conscience development (Kochanska, 1997) and psychopathology (see review in Rothbart, 2011).

Kochanska et al. (2000) have characterized the construct of EC as "situated at the intersection of the temperament and behavioral regulation literatures" (p. 220). What does EC mean for theories of temperament and personality? It means that contrary to earlier theories that emphasized how behavior is driven by positive and negative emotions or one's level of arousal, we are not always at the mercy of emotion and the affective–motivational systems (Rothbart, 2011). Using EC, we can more flexibly approach the situations we fear and inhibit the actions we desire. The efficiency of EC, however, may depend on the strength of the emotional processes against which effort is exerted. For example, when a child must delay an approach to an appealing toy, the child with a stronger disposition to approach is likely to require greater EC to succeed.

EC is also involved in the inhibition of immediate approach, with the goal of attaining a larger reward later, as in delay of gratification research (Shoda, Mischel, & Peak, 1990), and in Block's (2002) hedonism of the future. EC also allows activation of behavior that would otherwise not be performed, letting one act "on principle" when the principle opposes one's otherwise dominant response. EC is not itself a basic motivation; rather, it provides the means to effectively satisfy desired ends. It resembles the attentional capacities that underlie Block's (2002) construct of ego resiliency, that is, the ability to shift one's level of control flexibly depending on requirements of the situation.

There is substantial evidence that during childhood EC, as measured bytemperament questionnaires, is correlated with the ability to resolve conflict in many cognitive tasks

(Rothbart, 2011; Rueda, 2012). This provides an opportunity to examine the brain systems that are related to both conflict resolution and EC.

The Executive Attention Network

Posner and colleagues have identified three brain networks that serve different functions of attention and have different neural anatomies and neuromodulators (Posner, 2012). The first two networks involve *alerting* and *orienting*, respectively. The third, the *executive attention network*, which functions to monitor and resolve conflict, involves the anterior cingulate cortex (ACC), anterior insula, and basal ganglia. Dorsal areas of the cingulate have been implicated in the regulation of cognitive processing, while more ventral areas have been implicated in emotional processing (Bush, Luu, & Posner, 2000). In more recent research, individual differences in the functioning of the executive attention network have been theoretically and empirically linked to the temperament construct of EC (Chang & Burns, 2005; Posner, 2012; Rothbart, 2011).

According to one theory, executive attention, and the ACC in particular, is involved primarily in the monitoring of conflict between potentially competing systems (Botvinick, Braver, Barch, Carter, & Cohen, 2001) whereas the prefrontal cortex, particularly on the right side, is involved in inhibition of competing systems (see review by Posner, 2012). This division of labor between the ACC and the prefrontal cortex may not be the full story, but these ideas suggest complementary associations between the ACC and prefrontal cortex in self-regulation.

The ACC, one of the main nodes of the executive attention network, has been linked to specific functions related to self-regulation (see reviews by Posner, 2012; Rueda, 2012). These include the monitoring of conflict, control of working memory, regulation of emotion, and response to error. In emotion studies, the cingulate is often seen as part of a network involving the orbitofrontal cortex and amygdala that regulates our emotional response to input. Activation of the ACC is observed when people are asked to control their reactions to strong positive (Beauregard, Levesque, & Bourgouin, 2001) and negative emotions (Ochsner, Bunge, Gross, & Gabrieli, 2002).

As noted previously, dorsal areas of the cingulate have been implicated in regulating cognitive processing and more ventral areas in emotional processing (Bush et al., 2000). The portion of the ACC related to emotion regulation has a very high level of tonic activity, even at rest. This activity, together with the widespread connectivity of the cingulate to other brain areas, may be related to the need to maintain emotional control even when one is not performing any specific task. There is also some evidence of an inhibitory interaction between the more ventral emotion regulation part of the ACC and the more dorsal areas related to cognitive control (Drevets & Raichle, 1998).

ACC activation in response to perceived errors has been reported in infants as young as 7 months of age (Berger, Tzur, & Posner, 2006). Adults show ACC activation and usually slow down following an error. However, in a study using a Simple Simon task, we found that slowing down following an error did not arise until children were between 39 and 41 months of age (Jones, Rothbart, & Posner, 2003). We believe that the difference between infants who detect the error and children who modify action based on the error involves the change in connectivity of the ACC to other brain structures that takes place during childhood (Fair et al., 2009; Gao et al., 2009). The connectivity hypothesis may also have implications for other issues in development, for example, the observation that fear is shown in distress reactions before it is linked to behavioral inhibition (Rothbart et al., 2000). Further research is needed to relate connectivity to behavioral changes such as slowing.

One assessment of executive attention in early childhood is the spatial conflict task, in which object identity and spatial location of a stimulus are placed in conflict (Gerardi-Caulton, 2000). Here the child has two response keys, labeled with pictures of stimuli. Stimuli are presented on a computer screen, and the child is instructed to press the key corresponding to the picture presented. Conflict trials are those in which the stimulus appears on the side opposite the correct key.

Between 30 and 36 months of age, children learn to perform this task, which requires inhibiting the dominant response toward a spatial location in order to make a response based on matching identity (Gerardi-Caulton, 2000; Rothbart, Ellis, Rueda, & Posner, 2003). At 24 months, children are only able to carry out this task when the stimulus is on the same side of the computer screen as the matching response key (the congruent condition), but by 30 months, most children can handle incongruent trials in which the matching target is on the opposite side of the stimulus, although they are greatly slowed in conflict trials (adults are also slowed in this condition).

Children with higher levels of performance on spatial conflict have also been reported by their parents as having higher levels of EC and lower levels of negative emotionality on the Children's Behavior Questionnaire (CBQ; Rothbart et al., 2001, 2003). Two-year-old children who could not complete the spatial conflict task were also described by their parents as having lower EC and higher negative emotionality. These findings are consistent with the idea that the capacity to engage in rule-based action in conflict situations can support responding to social rules and regulation of emotion in daily life.

Another important conflict task is the Attention Networks Task (ANT), which measures efficiency of the alerting, orienting, and executive attention networks. In the ANT flanker task, the response to a target is in conflict with surrounding flanker stimuli (Rueda et al., 2004). Both spatial conflict and flanker tasks have been linked in imaging studies to the functioning of the brain's executive attention network (Fan, Flombaum, McCandliss, Thomas, & Posner, 2003) and have been used as model tasks for assessing the functioning of this network (Posner, 2012). Using the Child ANT (Rueda et al., 2004), significant improvement in conflict resolution has been found up until age 7, but a remarkable similarity in both reaction time and accuracy was found from the age of 7 to adulthood.

Throughout childhood, ANT performance is related to EC performance (Rothbart, 2011; Rueda, 2012), and adults high in EC show lower conflict scores and lower negative emotion (Rothbart, 2011). In adolescents and adults, performance on the ANT has also been linked to a number of psychopathologies (see review by Posner, 2012). These studies illustrate the close relation between attention, EC, and emotion regulation throughout life.

EC in Childhood

Evidence for stability of EC has been found in research by Mischel and his colleagues (Shoda et al., 1990). Preschoolers were tested on their ability to wait for a delayed treat that was larger than a readily accessible one. Children better able to delay gratification were found to have better self-control and greater ability to regulate reactions to stress and frustration. Their delay of gratification in seconds also predicted parent-reported attentiveness, concentration, emotion regulation, and intelligence during adolescence. In follow-up studies when the participants were in their 30s, preschool delay predicted goal-setting and self-regulatory abilities (Ayduk et al., 2000), suggesting remarkable continuity in self-regulatory skills.

Some of the complexity of this continuity in self-regulation is reflected in longitudinal studies of genetic contributions to early development. There is substantial evidence in adult studies that genetic alleles in the dopamine system are related to executive attention (Posner, 2012). One of these genes, the dopamine 4 receptor gene, was found to influence sensation seeking behavior as early as 18–20 months in interaction with the quality of parenting (Sheese, Voelker, Rothbart, & Posner, 2007). When the 7-repeat allele was present, relatively low-quality parenting produced higher sensation-seeking ratings, but when the 7-repeat was absent, sensation seeking was moderate or low, regardless of parenting quality. The remarkable susceptibility of children with the 7-repeat allele to parental and other environmental influences has been replicated multiple times (e.g., Belsky & Pluess, 2009). We did not see any relation of this gene to EC at 18–20 months (Sheese et al., 2007), but by 4 years there was a gene × parenting quality interaction in which EC ratings of children with the 7-repeat were more positively influenced by parenting quality than children without

the 7-repeat (Sheese, Voelker, Rothbart & Posner, 2012). Adult studies have also indicated that people with the 7-repeat allele are more influenced by their environment than those without it (Larsen et al., 2010). Thus, there appears to be an underlying continuity of genetic influence even though neural networks may be changing in their connectivity.

EC plays an important role in the development of conscience, with greater internalized conscience in children high in EC (Kochanska et al., 2000). Both the reactive temperamental control system of fearful inhibition and the attention-based system of EC appear to regulate the development of socialized thought and behavior, with the influence of fear found earlier in development. We have found that 6- to 7-year-old children who were high in EC were also high in empathy and guilt/shame, and low in aggressiveness (Rothbart et al., 1994). EC may support empathy by allowing children to attend to another child's condition instead of focusing only on their own sympathetic distress. Eisenberg, Fabes, Nyman, Bernzweig, and Pinulas (1994) found that 4- to 6-year-old boys with good attentional control tended to deal with anger by using nonhostile verbal methods rather than overt aggression.

During the toddler and preschool years, development of the executive attention network underlying EC allows children greater control of stimulation and response, including the ability to select responses in a conflict situation. Aksan and Kochanska (2004) found that children who were more fearful and inhibited at 33 months showed more volitional inhibitory control at 45 months. They suggest that more fearful and inhibited children have a greater opportunity to foster their own self-control during their periods of slow approach to novel situations.

Attention and Emotion Regulation

We now consider contributions of executive attention to emotion regulation via distraction, suppression, and reappraisal. In the section on orienting we described how defense and approach systems monitor for goal-relevant stimuli and, once activated, bias orienting toward some targets. Levels of emotional activation are thus maintained or increased until something occurs to interrupt the cycle. One way to interrupt the cycle is to perform actions that remove the stimulus or make it less relevant. For example, if a person is afraid of heights, visual cues indicating precipitous drops may initiate a cycle of anxiety and fear. One way to interrupt this cycle is to back away from the ledge. If such an action is not available or is undesirable, a second approach is to "not look down." Information reception can thus be controlled to reduce the activation of the system. This kind of regulation can also be applied to the approach system. Just as one can look away from things that are feared, one can also look away from things that are desired. Consistent with this hypothesis, children who are most able to resist temptation are those most likely to orient away from desired stimuli while waiting for them (e.g., Sethi et al., 2000).

Regulation of orienting may not always be sufficient to manage emotional reactions. Not looking at a cookie may reduce the probability we will eat it, but even when we are not looking at it, we still know it is there. Conceptual processing and memory allow us to maintain an internal representation of the stimulus over time. Internal representations may be just as effective, or even more so, in triggering and maintaining the activation of affective systems. Consequently, regulating internal representations becomes an important avenue for the regulation of emotional responding. Executive attention has been related to regulation of inappropriate cognitions, as in the Stroop effect (Posner, 2012), and similar mechanisms are likely involved in emotional regulation.

The same general network involved in control of emotions is active during the manipulation of internal representations, such as generating word associations (Posner & Raichle, 1994). Because working memory is finite, focusing attention on other representations may lead to the exclusion of "unwanted" representations. The executive attention system serves to monitor and resolve conflict among brain networks and may facilitate the manipulation of internal representations by allowing for the selection of one representation over another. We can thus attempt to control emotional responding by literally thinking about other things,

a process that has been referred to as *distraction*. However, as an emotion regulation strategy, distraction may have limited utility and ultimately adverse long-term consequences (Gross, this volume).

Research on sensory systems has shown that attention can work by increasing *activation* in an attended sensory system, as well as by reducing or suppressing activation of other systems (Posner & Raichle, 1994). To date, the upregulation of emotions has not been commonly addressed in either theoretical treatments or empirical studies of emotion regulation, although Kieras, Tobin, Graziano, and Rothbart (2005) have found that children higher in EC skills are more likely to be able to smile at the presentation of a disappointing gift, and laboratory research has linked high executive attention on the Child ANT to smiling in this paradigm (Rothbart et al., 2003). It seems likely that, as in orienting to sensory systems, the ability to smile in the face of disappointment involves both activation of smiling and the *suppression* of the emotion related to the disappointing gift.

Links have also been found between the executive attention network and another strategy for altering representations to promote emotion regulation, *reappraisal*, which involves reinterpreting the meaning or value of a representation (Gross, 2002). Ochsner et al. (2002) found that reappraisal led to reduced activity in the amgydala, suggesting that reappraisal changes emotion-related processing. Subsequent research has indicated that prefrontal and anterior cingulate regions are involved in the modulation of emotion processing through reappraisal (Ochsner et al., 2004). In some studies the orbitofrontal area that lies adjacent to the ACC seems to account for the major difference in brain activity found between distraction and reappraisal (Kanske, Heissler, Schönfelder, Bongers, & Wessa, 2011). Reappraisal may thus be another mechanism through which the executive attention system regulates affective systems, in that it involves a competition among alternative internal representations. The executive attention system facilitates the selection of a secondary representation over the prepotent representation (Posner, 2012). Recent studies of depressed patients have found that emotion regulation through reappraisal does not result in the same reduction in amygdala activity in patients as in normal participants. However, distraction worked similarly in both groups (Kanske, Heissler, Schönfelder, & Wessa, 2012).

Controlling attentional orienting, excluding prepotent representations, and reinterpretation are strategies for reducing activation of reactive systems by altering overt or internal input into those systems. A different avenue for regulation concerns altering output of the reactive systems. Bidirectional links between the affective–motivational systems and areas of output, such as the autonomic system, allow for feedback loops that can maintain emotional states (Tang & Posner, 2009). The executive attention system may enable efforts to modulate different aspects of physiological arousal, including both up-regulation and down-regulation. Direct manipulation of aspects of physiological arousal that may be under conscious control, such as changes in rates of respiration and body muscle tension, is one possibility. Evidence from imaging studies and animal models indicates that the ACC plays a substantial role in the modulation of autonomic reactivity in response to situational demands (e.g., Critchley et al., 2003).

So far, we have primarily considered emotion regulation in terms of reduction of activation. The defense and approach systems motivate adaptive behavior by inducing emotional states, while the executive attention system allows for the suppression of these reactive systems. However, it is important to note that the same mechanisms through which the executive attention system produces reductions in activation of reactive systems can also be used to produce increases in activation. If we want to induce an affective state, we can consciously shift attention to the appropriate affect-inducing stimuli, recall affect-inducing representations, or reinterpret neutral representations to promote affective responses. The executive attention system may also allow us to elicit emotional responses by increasing physiological arousal or by producing behaviors that are consistent with the desired emotional state.

Since the executive attention network is important in emotional control, an obvious question is whether intervention during preschool or later can improve the functioning

of this network and thus alter the ability to regulate emotion. We now describe efforts to improve executive attention through training.

Training Executive Attention

There may be two quite different ways to train attention (Tang & Posner, 2009). One involves training by exercising a particular attention network, and the other, a change in brain state through meditation or related procedures. We have examined training of the executive attention system in children ages 4–6 years (Rueda, Rothbart, McCandliss, Saccomanno, & Posner, 2005). The programs we have used were adapted from those used to train monkeys for space flight (Rumbaugh & Washburn, 1995). They involve first training the child to use a joystick to place a cat on grass rather than on mud. As training proceeds, the grass shrinks and control becomes more difficult; these skills are further built on to train anticipation, improve memory, and exercise the ability to resolve conflict (Rueda et al., 2005).

Both 4- and 6-year-olds showed greater improvements in the brain networks related to executive attention after 5 days of computer training than a control group that interacted with videos (Rueda et al., 2005). The group taking attention training also showed improved IQ scores. A replication and extension of this study to 10 days of training was carried out in a Spanish preschool (see review in Rueda, 2012). In addition to replicating previous work, attention training in this study improved children's emotion regulation and self-control, including their performance on delay of reward and children's gambling tasks. A number of other methods used in classroom studies have also been effective in enhancing the development of executive attention; they are helpful to children with and without problems in attention (see Diamond & Lee, 2011, for a summary).

Studies of mindfulness meditation have also reported improvements of adults on the conflict measure of the ANT and more positive mood after only 5 days of training in comparison with a control group given relaxation training (Tang et al., 2007). After 5 days of training, stress levels, as measured by cortisol secretion, were reduced following a cognitive challenge of mental arithmetic. After 30 days of training, cortisol levels were lower even at baseline, suggesting that the meditation group members experienced reduced stress in their daily life to a greater degree than those without this training (Tang et al., 2007). The mechanism of these changes appears to be in alterations of the activation and connectivity of the ACC to other brain areas (Tang, Lu, Fan, Yang, & Posner, 2012).

Issues in Temperament Assessment

In temperament studies involving emotion regulation it has been common to consider only individual differences in components of reactive temperament, without considering either regulatory aspects of temperament, such as EC, or the interactions between these components. Studies focusing on only single traits or looking only at main effects may be missing important opportunities for understanding the dynamic contribution of temperament to outcomes. Research supports the importance of considering interactions among dimensions of temperament in predicting external criteria. For example, Gunnar, Sebanc, Tout, Donzella, and van Dulmen (2003) found that high levels of surgency (approach) combined with low levels of EC were associated with higher cortisol levels and higher peer rejection in childhood (see reviews by Rothbart, 2011; Rothbart & Bates, 2006).

It is also important to note that depending on context, the systems may either act relatively independently, in direct competition, or complement one another. Without considering the person's goals, it is difficult to predict his or her particular reactions a priori. For example, considering fearfulness in terms of the defense system emphasizes that the network serves not only to inhibit behavior in some circumstances but also to motivate avoidance or attack. In contrast, EC, considered as an index of individual differences in the functioning of the executive attention system, serves to inhibit and facilitate processing in diverse cognitive and affective networks, with the goal of modulating prepotent responses. Sometimes, the goals of the defense and executive attention systems may agree. In these instances

we would expect both temperamental fearfulness and EC to contribute to predicting an outcome (e.g., following directions during a fire drill). In other circumstances the goals of the systems may be at odds, and we would expect behavioral outcomes to reflect an interaction between the two systems (e.g., jumping out of an airplane).

The approach adopted here also suggests that it is important to consider how each system contributes to a given temperament assessment. When we bring a child into the laboratory and attempt to assess EC using a variety of game-like tasks, task performance reflects dynamic interactions among several systems, not just EC. The approach system may respond to engaging activities and rewards; the defense system may respond to the novelty of the laboratory, the procedures, and the experimenters. Finally, the executive attention system contributes to task performance, and also to keeping emotional responses in line with the parent's, experimenter's, and personal expectations. We can try to get direct assessments of one aspect of temperament, but outcomes almost always reflect a mix of reactive and regulatory processes. For example, Laptook, Klein, Olino, Dyson, and Carlson (2010) showed that children displaying the same low approach/engagement could be differentiated into children with low positive affect and children with high behavioral inhibition.

Inhibitory control tasks, commonly employed in behavioral assessments of EC, present an interesting example of how even basic assessments may potentially tap into multiple temperament systems. Assessments such as the day–night Stroop task focus on assessing the inhibition of prepotent responses. However, to perform this task successfully, children must also sit still, follow instructions, attend to the stimuli, and activate nonprepotent responses; motivation to perform the task is also important (Huizenga, van der Molen, Bexkens, Bos, & van den Wildenbeg, 2012).

Even in the absence of overt feedback or reward from the experimenter, we might expect participants' reactive responses to vary in the performance of these tasks. Consistent with this idea, Wolfe and Bell (2004) found that while parent-reported inhibitory control was a significant predictor of performance on inhibitory control laboratory tasks ($r = .36$), parent-reported approach/anticipation was a better predictor ($r = -.56$). Thus, while inhibitory control may be properly considered a facet of EC, inhibitory control measures may reflect an interaction between the approach and the executive attention systems.

Dynamic interaction between brain areas is also found in imaging studies when increases in activation of the ACC are accompanied by reduced activity in the amygdala during control of negative emotion (Etkin, Egner, Peraza, Kandel, & Hirsch, 2006). In patient populations such as those suffering from borderline personality disorder (Goldstein et al., 2007) or depression (Kanske & Kotz, 2012), this interaction is disrupted.

This dynamic view of interacting systems suggests that single-method assessments of temperament may be problematic. When possible, researchers should employ batteries of differentiated assessments in which the common feature is the temperament characteristic of interest. Multimethod assessment procedures are further complemented when multiple temperament traits are assessed (e.g., in questionnaires), particularly when reactive and regulatory components of temperament are being examined. A multitrait approach allows us to better assess individual differences in regulation by controlling for individual differences in reactivity, and vice versa.

Future Directions

We suggest that our understanding of emotion regulation will be advanced if we conceptualize temperament constructs in terms of affective–motivational and attentional systems, study these systems at multiple levels, including the level of neural networks, and consider dynamic interactions among these systems. Considering emotional reactivity and emotion regulation as core components of temperament represents a first step toward a more dynamic, process-oriented view. The second step leads to considering reactive and regulative components of temperament together rather than in isolation. Linking temperament to the study of emotion and emotion regulation also provides temperament researchers with a rich empirical base for generating models of tempera-

ment development, such as models of the development of social interaction and the development of coping strategies (Rothbart, 2011). Advances in understanding the basic processes in emotional reactivity and emotion regulation should inform and enrich research on temperament. At the same time, research on temperament offers a unique perspective for examining emotion and emotion regulation.

References

Aksan, N., & Kochanska, G. (2004). Links between systems of inhibition from infancy to preschool years. *Child Development, 75*, 1477–1490.

Allport, G. W. (1937). *Personality: A psychological interpretation*. Oxford, UK: Holt.

Ayduk, O., Mendoza-Denton, R., Mischel, W., Downey, G., Peake, P., & Rodriguez, M. (2000). Regulating the interpersonal self: Strategic self-regulation for coping with rejection sensitivity. *Journal of Personality and Social Psychology, 79*, 776–792.

Beauregard, M., Levesque, J., & Bourgouin, P. (2001). Neural correlates of conscious self-regulation of emotion. *Journal of Neuroscience, 21*(RC165), 1–6.

Beaver, J. D., Mogg, K., & Bradley, B. P. (2005). Emotional conditioning to masked stimuli and modulation of visuospatial attention. *Emotion, 5*, 67–79.

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental stress. *Psychological Bulletin, 135*, 895–908.

Berger, A., Tzur, G., & Posner, M. I. (2006). Infant brains detect arithmetic error. *Proceeding of the National Academy of Sciences USA, 103*, 12649–12653.

Block, J. (2002). *Personality as an affect-processing system: Toward an integrative theory*. Mahwah, NJ: Erlbaum.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*, 624–652.

Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and motivation I: Defense and appetitive reactions in picture processing. *Emotion, 1*, 276–299.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4*, 215–222.

Calkins, S. D., & Williford, A. P. (2003, April). *Anger regulation in infancy: Consequences and correlates*. Paper presented at the meeting of the Society for Research in Child Development, Tampa, FL.

Campos, J. J., Caplovitz, B. K., Lamb, M. E., Goldsmith, H. H., & Stenberg, C. (1983). Socioemotional development. In P. H. Mussen, M. M. Haith, & J. J. Campos (Eds.), *Handbook of child psychology* (Vol. 2, pp. 783–915). New York: Wiley.

Chang, F., & Burns, B. M. (2005). Attention in preschoolers: Associations with effortful control and motivation. *Child Development, 76*, 247–263.

Critchley, H. D., Mathias, C. J., Josephs, O., O'Doherty, J., Zanini, S., Dewar, B. K., et al. (2003). Human cingulate cortex and autonomic control: Converging neuroimaging and clinical evidence. *Brain, 126*, 2139–2152.

Crockenberg, S. C., Leerkes, E. M. J., & Barrig, P. S. (2008). Predicting aggressive behavior in the third year from infant reactivity and regulation as moderated by maternal behavior. *Development and Psychopathology, 20*, 37–54.

Deater-Deckard, K., & Wang, Z. (2012). Anger and irritability. In M. Zentner & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 124–144). New York: Guilford Press.

Derryberry, D., & Reed, M. A. (1994). Temperament and attention: Orienting toward and away from positive and negative signals. *Journal of Personality and Social Psychology, 66*, 1128–1139.

Derryberry, D., & Reed, M. A. (2002). Anxiety-related attentional biases and their regulation by attentional control. *Journal of Abnormal Psychology, 111*, 225–236.

Derryberry, D., & Rothbart, M. K. (1997). Reactive and effortful processes in the organization of temperament. *Developmental Psychopathology, 9*, 633–652.

Diamond, A., & Lee, K. (2011). Interventions shown to aid Executive Function development in children 4–12 years old. *Science, 333*, 959–964.

Drevets, W. C., & Raichle, M. E. (1998). Reciprocal suppression of regional cerebral blood flow during emotional versus higher cognitive processes: Implications for interactions between emotion and cognition. *Cognition and Emotion, 12*, 353–285.

Eisenberg, N., Fabes, R. A., Nyman, M., Bernzweig, J., & Pinulas, A. (1994). The relations of emotionality and regulation to children's anger-related reactions. *Child Development, 65*, 109–128.

Etkin, A., Egner, T., Peraza, D. M., Kandel, E. R., & Hirsch, J. (2006). Resolving emotional conflict: A role for the rostral anterior cingulate cortex in modulating activity in the amygdala. *Neuron, 51*, 871–882.

Evans, D., & Rothbart, M. K. (2007). Developing a model for adult temperament. *Journal of Research in Personality, 41*, 868–888.

Fair, D. A., Cohen, A. L., Power, J. D., Dosenbach, N. U. F., Church, J. A., Miezin, F. M., et al. (2009). Functional brain networks develop from a local to distributed organization. *PLoS Computational Biology, 5*, e1000381.

Fan, J., Flombaum, J. I., McCandliss, B. D., Thomas, K. M., & Posner, M. I. (2003). Cognitive and brain consequences of conflict. *NeuroImage, 18*, 42–57.

Gao, W., Zhu, H., Giovanello, K. S., Smith, J. K., Shen, D., Gilmore, J. H., et al. (2009). Evidence on the emergence of the brain's default network from 2-week-old to 2-year-old healthy pediatric subjects. *Proceedings of the National Academy of Sciences USA, 106*, 6790–6795.

Gerardi-Caulton, G. (2000). Sensitivity to spatial conflict and the development of self-regulation in children 24–36 months of age. *Developmental Science, 3*, 397–404.

Goldstein, M., Brendel, G., Tuescheer, O., Pan, H., Epstein, J., Beutel, M., et al. (2007). Neural substrates of the interaction of emotional stimulus process and motor inhibitory control: An emotional linguistic go/no-go fMRI study. *NeuroImage, 36*, 1026–1040.

Gray, J. A., & McNaughton, N. (1996). *The neuropsychology of anxiety: Reprise.* Nebraska Symposium on Motivation, Perspectives on anxiety, panic and fear. Vol. 38, pp. 61–134.

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*, 281–291.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gunnar, M. R., Sebanc, A. M., Tout, K., Donzella, B., & van Dulmen, M. M. (2003). Peer rejection, temperament, and cortisol activity in preschoolers. *Developmental Psychobiology, 43*, 346–358.

Harman, C., Rothbart, M. K., & Posner, M. I. (1997). Distress and attention interactions in early infancy. *Motivation and Emotion, 21*, 27–43.

Huizenga, H. M., van der Molen, M. W., Bexkens, A., Bos, M. G., & van den Wildenberg, W. P. (2012). Muscle or motivation?: A stop-signal study on the effects of sequential cognitive control. *Frontiers in Cognition, 3*. [E-publication prior to print]

Ito, T. A., & Cacioppo, J. T. (2005). Variations on a human universal: Individual differences in positivity offset and negativity bias. *Cognition and Emotion, 19*, 1–26.

Johnson, M. H., Posner, M. I., & Rothbart, M. K. (1991). Components of visual orienting in early infancy: Contingency learning, anticipatory looking, and disengaging. *Journal of Cognitive Neuroscience, 3*, 335–344.

Jones, L. B., Rothbart, M. K., & Posner, M. I. (2003). Development of executive attention in preschool children. *Developmental Science, 6*, 498–504.

Kagan, J., & Fox, N. A. (2006). Biology, culture, and temperamental biases. In W. Damon & N. Eisenberg (Eds.), *Handbook of child psychology: Vol. 3. Social, emotional and personality development* (6th ed., pp. 167–225). New York: Wiley.

Kanske, P., Heissler, J., Schönfelder, S., Bongers, A., & Wessa, M. (2011). How to regulate emotion?: Neural networks for reappraisal and distraction. *Cerebral Cortex, 21*, 1379–1388.

Kanske, P., Heissler, J., Schönfelder, S., & Wessa, M. (2012). Neural correlates of emotion regulation deficits in remitted depression: The influence of regulation strategy, habitual regulation use, and emotional valence. *NeuroImage, 61*, 686–693.

Kanske, P., & Kotz, S. A. (2012). Effortful control, depression, and anxiety correlate with the influence of emotion on executive attentional control. *Biological Psychology, 91*, 88–95.

Kieras, J. E., Tobin, R. M., Graziano, W. G., & Rothbart, M. K. (2005). You can't always get what you want: Effortful control and children's responses to undesirable gifts. *Psychological Science, 16*, 391–396.

Kochanska, G. (1997). Multiple pathways to conscience for children with different temperaments: From toddlerhood to age 5. *Developmental Psychology, 33*, 228–240.

Kochanska, G., Murray, K. T., & Harlan, E. T.

(2000). Effortful control in early childhood: Continuity and change, antecedents, and implications for social development. *Developmental Psychology, 36*, 220–232.

Laptook, R. S., Klein, D. N., Olino, T. M., Dyson, M. W., & Carlson, G. (2010). Low positive affectivity and behavioral inhibition in preschool-age children: A replication and extension of previous findings. *Personality and Individual Differences, 48*, 547–551.

Larsen, H., van der Zwaluw, C. S., Overbeek, G., Granic, I., Franke, B., & Engels, R. C. (2010). A variable-number-of-tandem-repeats polymorphism in the dopamine D4 receptor gene affects social adaptation of alcohol use: Investigation of a gene–environment interaction. *Psychological Science, 21*, 1064–1068.

LeDoux, J. E. (1989). Cognitive–emotional interactions in the brain. *Cognition and Emotion, 3*, 267–289.

LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience, 23*, 155–184.

Lengua, L. J., & Wachs, T. D. (2012). Temperament and risk: Resilient and vulnerable responses to adversity. In M. Zentner & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 519–540). New York: Guilford Press.

McCrae, R. R., Costa, P. T., Jr., Ostendorf, F., Angleitner, A., Hrebícková, M., Avia, M. D., et al. (2000). Nature over nurture: Temperament, personality, and life span development. *Journal of Personality and Social Psychology, 78*, 173–186.

Norman, D. A., & Shallice, T. (1986). Attention to action: Willed and automatic control of behavior. In G. E. Schwartz & D. Shapiro (Eds.), *Consciousness and self-regulation* (Vol. 4, pp. 1–18). New York: Plenum.

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. E. (2002). Rethinking feelings: An FMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14*, 1215–1229.

Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D., et al. (2004). For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *NeuroImage, 23*, 483–499.

Paulus, M. P., Feinstein, J. S., Simmons, A., & Stein, M. B. (2004). Anterior cingulate activation in high trait anxious subjects is related to altered error processing during decision making. *Biological Psychiatry, 55*, 1179–1187.

Pérez-Edgar, K., McDermott, J. N. M., Korelitz, K., Degnan, K. A., Curby, T. W., Pine, D. S., et al. (2010). Patterns of sustained attention in infancy shape the developmental trajectory of social behavior from toddlerhood through adolescence. *Developmental Psychology, 46*, 1723–1730.

Posner, M. I. (2012). *Attention in a social world*. New York: Oxford University Press.

Posner, M. I., & Raichle, M. E. (1994). *Images of mind*. New York: Scientific American Library.

Posner, M. I., & Rothbart, M. K. (1998). Attention, self-regulation and consciousness. *Philosophical Transactions of the Royal Society of London B, 353*, 1915–1927.

Posner, M. I., & Rothbart, M. K. (2007a). *Educating the human brain*. Washington, DC: American Psychological Association Books.

Posner, M. I., & Rothbart, M. K. (2007b). Research on attentional networks as a model for the integration of psychological science. *Annual Review of Psychology, 58*, 1–23.

Posner, M. I., Rothbart, M. K. Sheese, B. E., & Voelker, P. (2012). Control networks and neuromodulators in early development. *Developmental Psychology, 48*, 827–835.

Putnam, S. P., Ellis, L. K., & Rothbart, M. K. (2001). The structure of temperament from infancy through adolescence. In A. Eliasz & A. Angleitner (Eds.), *Advances in research on temperament* (pp. 165–182). Lengerich, Germany: Pabst Science.

Rothbart, M. K. (2011). *Becoming who we are: Temperament and personality in development*. New York: Guilford Press.

Rothbart, M. K. (1988). Temperament and the development of inhibited approach. *Child Development, 59*, 1241–1250.

Rothbart, M. K., Ahadi, S. A., & Hershey, K. L. (1994). Temperament and social behavior in childhood. *Merrill–Palmer Quarterly, 40*, 21–39.

Rothbart, M. K., Ahadi, S. A., Hershey, K. L., & Fisher, P. (2001). Investigations of temperament at three to seven years: The children's behavior questionnaire. *Child Development, 72*, 1394–1408.

Rothbart, M. K., & Bates, J. E. (2006). Temperament in children's development. In W. Damon, R. Lerner, & N. Eisenberg (Eds.), *Handbook of child psychology: Vol 3. Social, emotional, and personality development* (6th ed., pp. 99–166). New York: Wiley.

Rothbart, M. K., & Derryberry, D. (1981). Devel-

opment of individual differences in temperament. In M. E. Lamb & A. L. Brown (Eds.), *Advances in developmental psychology* (Vol. 1, pp. 37–86). Hillsdale, NJ: Erlbaum.

Rothbart, M. K., Derryberry, D., & Hershey, K. (2000). Stability of temperament in childhood: Laboratory infant assessment to parent report at seven years. In V. J. Molfese & D. L. Molfese (Eds.), *Temperament and personality development across the life span* (pp. 85–119). Hillsdale, NJ: Erlbaum.

Rothbart, M. K., Ellis, L. K., Rueda, M. R., & Posner, M. I. (2003). Developing mechanisms of temperamental effortful control. *Journal of Personality, 71*, 1113–1143.

Rothbart, M. K., & Posner, M. I. (2006). Temperament, attention, and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Vol. 2. Developmental neuroscience* (2nd ed., pp. 465–501). New York: Wiley.

Rothbart, M. K., Ziaie, H., & O'Boyle, C. G. (1992). Self-regulation and emotion in infancy. In N. Eisenberg & R. A. Fabes (Eds.), *Emotion and its regulation in early development* (pp. 7–23). San Francisco: Jossey-Bass.

Rueda, M. R. (2012). Effortful control. In M. Zentner & R. L.Shiner (Eds.), *Handbook of temperament* (pp. 145–167). New York: Guilford Press.

Rueda, M. R., Fan, J., McCandliss, B. D., Halparin, J. D., Gruber, D. B., Lercari, L. P., et al. (2004). Development of attentional networks in childhood. *Neuropsychologia, 42*, 1029–1040.

Rueda, M. R., Rothbart, M. K., McCandliss, B. D., Saccomanno, L., & Posner, M. I. (2005). Training, maturation, and genetic influences on the development of executive attention. *Proceedings of the National Academy of Sciences USA, 102*, 14931–14936.

Rumbaugh, D. M., & Washburn, D. A. (1995). Attention and memory in relation to learning: A comparative adaptation perspective. In G. R. Lyon & N. A. Krasengor (Eds.), *Attention, memory and executive function* (pp. 199–219). Baltimore: Brookes.

Schimmack, U. (2005). Attentional interference effects of emotional pictures: Threat, negativity, or arousal? *Emotion, 5*, 55–66.

Sethi, A., Mischel, W., Aber, J. L., Shoda, Y., & Rodriguez, M. L. (2000). The role of strategic attention deployment in development of self-regulation: Predicting preschoolers' delay of gratification from mother–toddler interactions. *Developmental Psychology, 36*, 767–777.

Sheese, B. E., Voelker, P., Posner, M. I., & Rothbart, M. K. (2009). Genetic variation influences on the early development of reactive emotions and their regulation by attention. *Cognitive Neuropsychiatry, 14*, 332–355.

Sheese, B. E., Voelker, P. M., Rothbart, M. K., & Posner, M. I. (2007). Parenting quality interacts with genetic variation in dopamine receptor DRD4 to influence temperament in early childhood. *Development and Psychopathology, 19*, 1039–1046.

Sheese, B. E., Voelker, P. M., Rothbart, M. K., & Posner, M. I. (2012). The dopamine receptor D4 gene 7-repeat allele interacts with parenting quality to predict effortful control in four-year-old children. *Child Development Research, 86*, 32–42.

Shiner, R. L., & Caspi, A. (2012). Temperament and the development of personality traits, adaptations, and narratives. In M. Zentner & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 497–518). New York: Guilford Press.

Shoda, Y., Mischel, W., & Peake, P. K. (1990). Predicting adolescent cognitive and self-regulatory competencies from preschool delay of gratification: Identifying diagnostic conditions. *Developmental Psychology, 26*, 978–986.

Tang, Y.-Y., Lu, Q., Fan, M., Yang, Y., & Posner, M. I. (2012). Mechanisms of white matter changes induced by meditation. *Proceedings of the National Academy of Sciences USA, 109*, 10570–10574.

Tang, Y.-Y., Ma, Y., Wang, J., Fan, Y., Feng, S., Lu, Q., et al. (2007). Short term meditation training improves attention and self regulation. *Proceedings of the National Academy of Sciences USA, 104*, 17152–17156.

Tang, Y., & Posner, M. I. (2009). Attention training and attention state training. *Trends in Cognitive Science, 13*, 222–227.

Wolfe, C. D., & Bell, M. A. (2004). Working memory and inhibitory control in early childhood: Contributions from electrophysiology, temperament, and language. *Developmental Psychobiology, 44*, 68–83.

Zentner, M., & Shiner, R. L. (2012). *Handbook of temperament*. New York: Guilford Press.

Zuckerman, M. (2012). Models of adult temperament. In M. Zentner & R. L. Shiner (Eds.), *Handbook of temperament* (pp. 41–68). New York: Guilford Press.

CHAPTER 20

Three Approaches to Individual Differences in Affect Regulation: Conceptualizations, Measures, and Findings

Oliver P. John
Joshua Eng

Over the past two decades, interest in the way people can and do regulate their affective states has increased rapidly. Much of the research has been experimental (e.g., Webb, Miles, & Sheeran, 2012). However, to understand the antecedents and consequences of affect regulation in everyday life, outside the laboratory, one must study naturally occurring individual differences (John & Gross, 2007). Although it is now widely agreed that individuals differ systematically and consistently in affect regulation, these individual differences have been conceptualized and measured in many different ways. Different conceptualization and measures arose, in part, because individual differences in affect regulation are not uniquely associated with just *one* field within psychology. Instead, researchers in several distinct subdisciplines in psychology are keenly interested in this topic.

Personality and social psychologists are interested in positive and negative mood states, and how stable individual differences in self-control influence longer-term adaptation (e.g., Block & Block, 1980; Baumeister, Zell, & Tice, 2007). Stress and coping researchers study the myriad ways individuals differ when they encounter an intensely stressful life situation and try to maintain well-being (e.g., Carver & Scheier, 1994; Folkman & Lazarus, 1980). Clinical psychologists tend to focus on the dysregulation of affect (i.e., failures and problems with regulation) and its contribution to various forms of pathology (e.g., Kring & Sloan, 2009). Emotion researchers examine the specific experiential, expressive, and physiological components of the emotional response process and the role of regulation within this process (e.g., Gross & Levenson, 1993). Developmental psychologists are interested in how children learn to regulate emotional states in family and peer contexts and to do so in ways that are adaptive and socially appropriate, thus becoming emotionally competent adults (e.g., Morris, Silk, Steinberg, Myers, & Robinson, 2007).

With such a wide range of stakeholders and their particular interests, conceptual preferences, and measurement traditions, it is not surprising that research on individual differences in the regulation of affective states (e.g., stress, mood, specific emotions) has not proceeded as a unified science. Many researchers agree that this is not a desirable state of affairs. For example, Weinberg and Klonsky (2009, p. 616) noted,

"Despite growing consensus regarding the importance of emotion dysregulation in psychopathology, the field has not yet reached an agreement on the construct's definition. Multiple components of emotion regulation have been proposed. . . . " Similarly, Morris et al. (2007, p. 363) concluded, "Existing empirical studies of ER [emotion regulation] differ widely in the measures, methods, and levels of analyses employed."

More generally, research on individual differences

> in the emotion regulation domain is still in its infancy. A consequence is the lack of a definitive conceptualization and assessment methods of ER, with significant inconsistencies across studies. . . . In order to contribute to greater scientific and methodological rigor, there is a need to empirically examine these relationships within a valid theoretical framework that conceptualizes ER in a multifaceted way. . . . Utilization of a model, such as Gross' (1998), would do good service to this weakness in the existing literature. (Bariola, Gullone, & Hughes, 2011, p. 208)

Indeed, the process model proposed by Gross (1998, this volume; see also Gross & Thompson, 2007) has attracted much attention and has proven influential in research on individual differences as well (e.g., Gross & John, 2003; John & Gross, 2004), because it defines core constructs and provides a general terminology.

In the first major part of this chapter, we describe this model and the individual-differences research it has inspired, focusing on a small set of carefully specified processes used to regulate emotions. We begin with this approach to individual differences for two reasons, one personal and the other conceptual. First, having contributed to this research, we are obviously more familiar with this work than with other approaches. Second, Gross's process model is sufficiently general to serve as a framework that can help organize, interpret, and compare the various ways individual differences in affect regulation have been conceptualized and studied.

Our review of the existing research on individual differences suggests that, in addition to work based on the emotion regulation process model, two other major approaches address the regulation of affective states broadly defined, namely, individual differences in coping with stress and individual differences in emotional competence. We begin with a brief overview of these three approaches. Then we consider each approach in turn, focusing on conceptualization and major measures. In the final section we outline limitations, continuing issues, and future directions for research.

Overview: Three Approaches to Individual Differences in Affect Regulation

Humans have multiple kinds of affective (i.e., valenced) states that they may control or regulate, such as stress, negative affect, or distinct emotions (e.g., pride or sadness) (Gross & Thompson, 2007). Therefore, we designate individual differences in *affect regulation* as the superordinate domain; here we include all kinds of regulatory attempts to influence any valenced responses. Figure 20.1 shows that below that broad rubric, we may consider three somewhat overlapping subdomains, namely, individual differences (1) in *specific processes used to regulate emotions*, (2) in *coping with stress*, and (3) in *emotional competence*.

Most goal-directed actions and cognitions can serve to maximize pleasure or minimize pain and may therefore be said to regulate affective states. Nonetheless, the three approaches to the study of individual differences in Figure 20.1 do differ from each other, in terms of the particular affective responses subject to regulation, the relevant situational contexts, and the nature of the cognitive and behavioral processes included.

Broadly speaking, the *coping* approach can be distinguished from the *specific emotion regulation process* approach by the fact that coping focuses primarily on reducing negative affect rather than distinct emotions, and typically extends across longer periods of time (e.g., coping with loss). Stress responses also are less clearly defined than specific emotions in terms of behavioral response tendencies; thus, coping is more likely to involve changing experiential, rather than behavioral–expressive, components of the affective response.

Whereas the coping approach is narrowly focused on those situations that are stressful, individual differences in the *emotional*

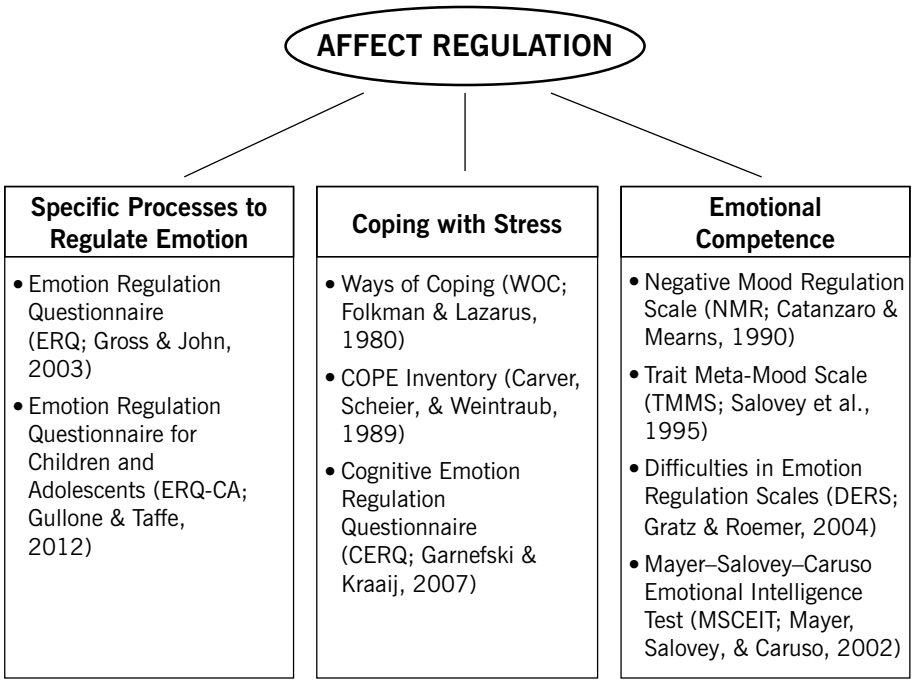

FIGURE 20.1. Three major approaches to studying individual differences in affect regulation via self-report and some of the major measures.

competence approach apply to a broader set of contexts. Furthermore, the emotional competence approach includes a host of processes, skills, and competencies (e.g., attention to feelings; clarity about feeling states) that do not directly regulate emotions but make it easier for the individual to behave in socioemotionally appropriate ways. Additionally, in the emotional competence approach, the specific processes used to regulate feelings or moods are not of core importance and thus tend to remain undifferentiated; individual differences in strategy use are often summarized in terms of a single concept, such as whether the individual makes greater or lesser efforts at "mood repair" (e.g., Salovey et al., 1995) or has great or only limited "access to effective regulation strategies" (e.g., Gratz & Roemer, 2004). Thus, the emotional competence approach is much broader than the specific emotion regulation process approach.

Each of these three broad conceptual approaches has generated a number of measures. Examples of the major measures associated with each approach are listed in Figure 20.1. We now describe each approach and the associated measures in some detail.

Individual Differences in Specific Processes Used to Regulate Emotions

The Process Model

We begin with an approach to individual differences that is based on the model of specific emotion regulation processes developed by Gross (1998). He derived this model from a generally accepted conception of the emotion-generative process, which holds that an emotion begins with an evaluation (or appraisal) of emotion cues. When attended to and evaluated in certain ways, emotion cues trigger a coordinated set of experiential, behavioral, and physiological response tendencies. Once these response tendencies arise, they may be modulated in various ways. Because emotion unfolds over time, emotion regulation strategies can be differentiated in terms of *when* they have their primary impact on the emotion-

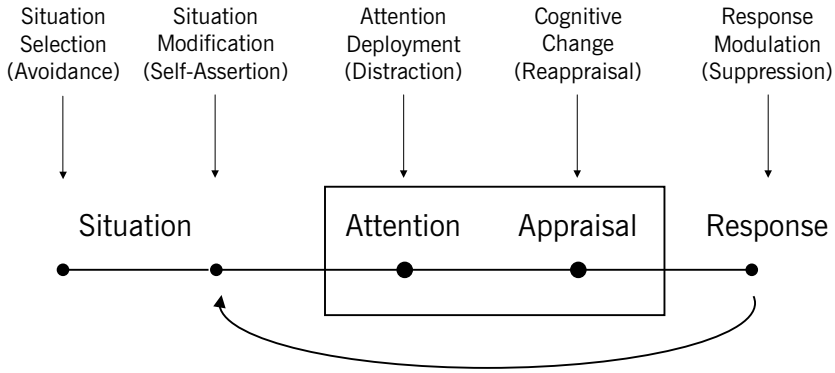

FIGURE 20.2. A process model of emotion regulation. Individual differences in emotion regulation may arise at five points in the emotion-generative process: (1) selection of the situation; (2) modification of the situation; (3) deployment of attention; (4) change of cognitions; and (5) modulation of experiential, behavioral, or physiological responses. Specific instantiations of these five families of regulatory strategies (given in parentheses) may be used for the down-regulation of negative emotion, as described in the text. From John and Gross (2007). Copyright 2007 by The Guilford Press. Reprinted by permission.

generative process. As shown in Figure 20.2, five families of more specific strategies can be located along the timeline of the emotion process (Gross, 1998).

Here we illustrate the use of these strategies for the most frequent goal of emotion regulation in everyday life, namely, down-regulating (decreasing) emotions that typically have a negative valence, such as anxiety/fear, sadness, and anger (Gross, Richards, & John, 2006). Specific forms of emotion down-regulation are indicated in Figure 20.2 in parentheses under each family name. In particular, *situation selection* refers to avoiding certain people, places, or activities so as to limit one's exposure to situations likely to generate negative emotion. Once a situation has been selected, *situation modification* operates to tailor or change a situation so as to decrease its negative emotional impact. Third, situations have many different aspects, so *attentional deployment* can be used to focus on less negatively valenced aspects of the situation. Once one has focused on a particular aspect of the situation, *cognitive change* refers to constructing a more positive meaning out of the many possible meanings that may be attached to that situation. Finally, *response modulation* refers to various kinds of attempts to influence emotion response tendencies once they already have been elicited.

Individual Differences Measured with the Emotion Regulation Questionnaire

Empirical research on individual differences has focused on two specific regulatory processes (for reviews, see John & Gross, 2004, 2007). *Cognitive reappraisal* is a form of cognitive change that involves construing a potentially emotion-eliciting situation in a way that changes its emotional impact. For example, during an admissions interview, one might view the give and take as an opportunity to find out how much one likes the school, rather than as a test of one's worth. *Expressive suppression* is a form of response modulation that involves inhibiting ongoing emotion-expressive behavior. For example, one might keep a poker face while holding a great hand during an exciting card game. Antecedent-focused strategies such as reappraisal influence whether or not particular emotion response tendencies *are triggered*, and are therefore expected to have generally positive implications for affective and social functioning. In contrast, response-focused strategies such as suppression influence how emotion response tendencies *are modulated once they have been triggered*, and are therefore expected to have generally more negative implications for affective and social functioning (cf. Gross, 1998).

To test whether there are reliable and systematic individual differences in these two emotion regulation processes, Gross and John (2003) developed the Emotion Regulation Questionnaire (ERQ), a brief 10-item measure of the habitual use of reappraisal and suppression. Items were derived rationally, starting with the experimental manipulations used in previous experimental research, and indicating clearly in each item the intended emotion regulatory process, such as "I control my emotions *by changing the way I think* about the situation I'm in" (reappraisal) and "I control my emotions *by not expressing them*" (suppression). These example items refer to emotion in general. In addition, the Reappraisal and Suppression scales each include at least one item asking about regulating *negative* emotion (illustrated for the participants by giving sadness and anger as examples) and one item about regulating *positive* emotion (exemplified by joy and amusement). Gross and John also were careful to limit the item content to the intended emotion regulatory strategy in order to avoid potential confounding by mentioning any positive or negative consequences on affect, social functioning, or well-being.

The structure of the ERQ is consistent across samples, ages, and cultures, indicating a clear two-factor structure. Factor analyses have shown two independent factors in multiple samples of young and older adults in the United States (John & Gross, 2004), Germany (Abler & Kessler, 2009), Italy (Balzarotti, John, & Gross, 2010), China (English & John, 2013), Japan (Eng, Akutsu, Gross, & John, 2013), and more than 15 other language communities in which the ERQ has been adapted (Matsumoto et al., 2008). Consistent with factor-analytic findings, the correlation between the ERQ Reappraisal and Suppression scales tends to be close to zero. That is, individuals who frequently use reappraisal are no more (or less) likely to use suppression than individuals who use reappraisal infrequently. These findings are important because they provide clear evidence against a single, general-factor model, in which some individuals regulate their emotions a lot using both reappraisal and suppression, whereas other individuals regulate their emotions rarely, using neither strategy frequently. Instead, reappraisal and suppression are two independent regulatory strategies that different individuals use to varying degrees.

In terms of mean differences, gender and culture differences emerged for suppression, which is expected to reflect culturally defined display rules for emotion (Ekman, 1972). The pattern of gender differences was highly consistent across cultures. Masculinity is generally associated with acting tough and unemotional, thus avoiding any indication of weakness or dependency (Knobloch & Metts, 2013). Indeed, emotion expression may be costly for men; for example, expression of negative emotion has been linked to lower social status for men but not for women (Anderson, John, Keltner, & Kring, 2001). In general, then, men should use suppression more often than women. As shown in Figure 20.3, men indeed reported lower levels of suppression use than women; in contrast, there were no gender differences in reappraisal use in any of the cultures studied.

Figure 20.3 also shows that among both men and women, the Japanese samples scored highest in suppression use. This is consistent with mean suppression scores in 23 nations (Matsumoto et al., 2008); individuals in Western countries that value independence were less likely to suppress their emotions than individuals in East Asian countries that value interdependence. However, cross-national comparisons cannot conclusively pinpoint the origin of the national–group differences, so Eng et al. (2013) tested for acculturation effects (1) between groups of European Americans and East Asian Americans and (2) within a group of Asian American immigrants who differed in their acculturation to Western culture (indexed by the length of their residence in the United States). Figure 20.4A shows that European Americans scored lower in suppression than did East Asian Americans. Figure 20.4B shows parallel suppression differences in the within-group analysis: The longer East Asian immigrants had lived in the United States and been exposed to Western cultural values and practices, the less suppression they reported, in effect becoming more like European Americans. How can these cultural group differences and acculturation effects be explained? As we will argue in the final section of this chapter, models of

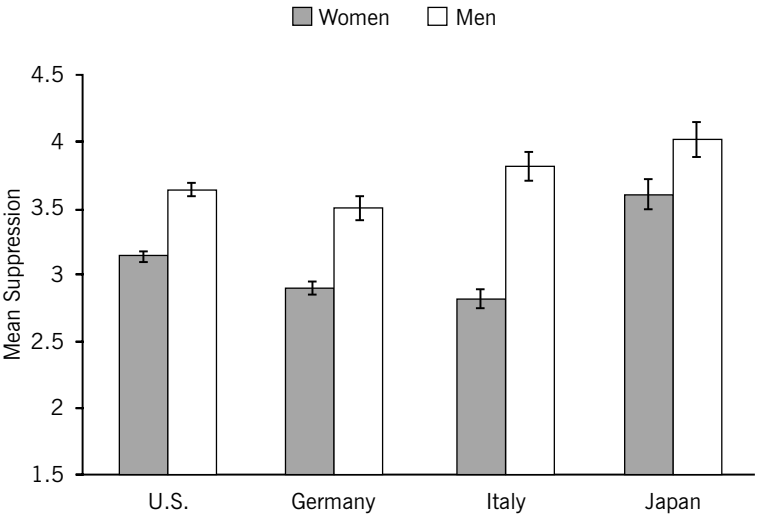

FIGURE 20.3. Men suppress more than women across four cultures. Mean ERQ suppression scores are shown for men and women in samples from the United States (Gross & John, 2003), Germany (Abler & Kessler, 2009), Italy (Balzarotti, John, & Gross, 2010), and Japan (Eng et al., 2013).

individual differences in emotion regulation need to consider values, goals, and other motivational factors, such as the culturally transmitted values associated with independent and interdependent self-construal.

Finally, it is important to comment on discriminant validity. The ERQ Reappraisal and Suppression scales were not related to various measures of cognitive ability (Gross & John, 2003; McRae, Jacobs, Ray, John, & Gross, 2012). Similarly, correlations with social desirability were small, probably because the ERQ items are worded fairly neutrally and do not mention adjustment or psychological health outcomes. Finally, correlations with the broad personality dimensions defined by the Big Five traits (e.g., John, Naumann, & Soto, 2008) showed only modest relations; highly neurotic individuals were slightly less likely to use reappraisal, and highly extraverted individuals were somewhat less likely to use suppression. Overall, these findings are consistent with a conceptualization of reappraisal and suppression as rather specific and narrowly defined individual differences in emotion regulation processes. The discriminant validity findings are particularly important here, because theory predicts habitual use of reappraisal should have generally favorable implications for adjustment and psychological health, whereas greater habitual use of suppression should have less favorable implications. Thus, it is important that any such adjustment effects cannot be attributed to other factors, such as intelligence, socially desirable responding, or general temperament and personality traits.

In terms of adjustment outcomes, the general process model (Gross, 1998) holds that reappraisal occurs early in the emotion-generative process, before emotion response tendencies have been fully generated, and thus permits the modification of the entire emotional sequence, including the experience of more positive and less negative emotion, without notable physiological, cognitive, or interpersonal costs. Suppression, by contrast, comes relatively late in the emotion-generative process and primarily modifies behavioral emotion response tendencies, without reducing the experience of negative emotion. Because suppression comes late in the emotion-generative process, it requires the individual to effortfully manage emotion response tendencies that arise continually, thus consuming cognitive resources that could otherwise be used for optimal performance in the social contexts in which the emotions occur. Moreover, in everyday life, the habitual suppression of one's true feelings is expected to create in the

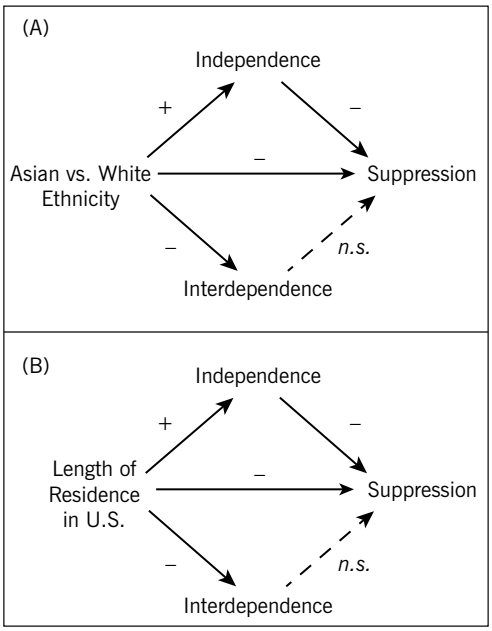

FIGURE 20.4. Explaining the effects of culture on emotion regulation: Ethnicity and acculturation effects on suppression were mediated by independent (but not interdependent) self-construal. Panel (A) shows that in a sample of Asian and White students, the between-group effect of ethnicity (coded Asian = 0, White = 1) on suppression was mediated through independence (but not through interdependence). Panel (B) shows parallel mediation effects within the group of Asian immigrants: length of acculturation (residence) in the United States was associated with less suppression use, and this effect was mediated through greater independence (but not interdependence) in self-construal. Signs indicate the relationship between variables: "+" indicates a positive effect, "-" a negative effect, and "*n.s.*" a null effect.

individual a sense of discrepancy between inner experience and outer expression, leading to negative feelings about the self and to interpersonal behavior that is distracted, strained, and avoidant, thus impeding the development of emotionally close relationships (see also English & John, 2013; English, John, & Gross, 2013; John & Gross, 2004).

In general, empirical evidence has supported the prediction that *more* regulation is not necessarily better but, instead, the effects of regulation should depend on the particular regulatory process used: Individual differences in the habitual use of reappraisal and suppression have differential implications for effective functioning (e.g., Abler & Kessler, 2009; Balzarotti et al., 2010; English & John, 2013; English, John, & Gross, 2013; Gross & John, 2003; Richards & Gross, 2000; Srivastava, Tamir, McGonigal, John, & Gross, 2009). Figure 20.5 illustrates typical findings. In terms of affective implications, habitual use of reappraisal was related to more positive emotion and less negative emotion. Cognitively, reappraisal does not appear to have reliable effects on social memory. In the domains of interpersonal functioning and well-being, reappraisal was generally associated with better psychological health. However, these relationships may turn out to be more complex; for example, the positive interpersonal effects of reappraisal may be more apparent in peer ratings of relationship quality than in self-ratings, and the well-being effects of reappraisal may vary somewhat depending on measures and samples (e.g., Aldao, Nolen-Hoeksema, & Schweizer, 2010; English & John, 2013; English, John, Srivastava, & Gross, 2012).

A rather different picture emerges for habitual use of suppression. As shown in Figure 20.5, suppression is related to less positive and more negative emotion experience. Cognitively, suppression is associated with degraded memory for socially relevant information. Socially, suppression is consistently related to relationships that are less close, satisfying, and supportive, even in highly interdependent cultures such as China (English & John, 2013). In terms of well-being, suppression again has unfavorable effects, although that may be limited to Western cultures that highly value independence and authenticity (English & John, 2013).

Whereas there is now considerable evidence for effects on adjustment and psychological health, much less is known about the development of individual differences in reappraisal and suppression (see John & Gross, 2004). For example, research on adults suggests that gender and cultural socialization processes are likely playing a role. As discussed earlier (see Figures 20.3 and 20.4), on average men report higher lev-

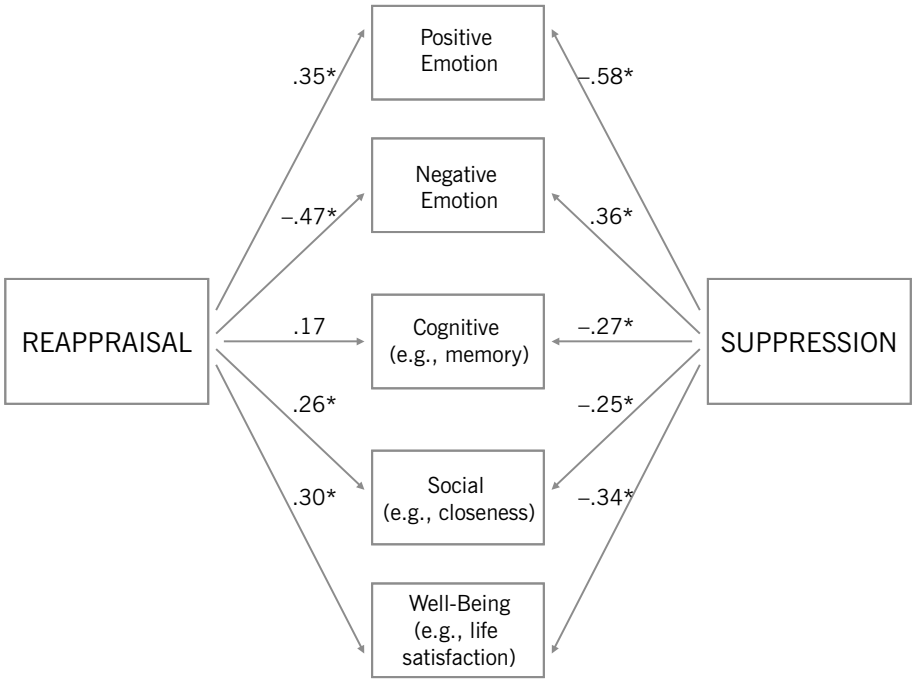

FIGURE 20.5. Individual differences in the habitual use of reappraisal and suppression are differentially associated with healthy adaptation in emotion experience, cognition, relationships, and well-being (adapted from John & Gross, 2004, 2007). Specific correlational findings in the figure are given only for illustrative purposes, and effect sizes vary across samples and measures used.

els of suppression use than women, and East Asians report higher levels of suppression use than Westerners. However, at this point we know little about when and how these differences in emotion regulation develop.

There is reason to expect this state of affairs to change for the better in the near future. A version of the ERQ adapted for children and adolescents (ERQ-CA) has recently been published (Gullone & Taffe, 2012). Using this instrument, promising findings on the development of reappraisal and suppression are beginning to appear (e.g., Bariola et al., 2011).

The Coping-with-Stress Approach and Its Major Measures

The literature on stress and coping originated from psychodynamic concepts and ideas on anxiety and defense mechanisms that have provided an important starting point for much thinking about affective states (e.g., Shaver & Mikulincer, 2007; Westen & Blagov, 2007). For several decades, stress researchers have studied individual differences in coping styles—the ways individuals attempt to deal with adversity (e.g., Lazarus & Folkman, 1984; see also Carver & Scheier, 1994). In their pioneering work, Folkman and Lazarus (1985) emphasized that coping has two major functions, namely, "the regulation of distressing emotions and doing something to change for the better the problem causing the distress" (p. 152). That is, coping includes regulatory efforts directed at internal emotional responses (e.g., anxiety) or at external problems facing the individual (e.g., a final exam). What are the major ways, or dimensions, of coping in which individuals differ from each other?

Ways of Coping Questionnaire

Folkman and Lazarus (1980, 1985, 1988) reasoned that multiple ways of coping could

be distinguished but, lacking an established theoretical framework, tried to discover the major dimensions of coping empirically. They initially assembled in the Ways of Coping Questionnaire 68 items intended to capture a wide variety of behavioral and cognitive coping responses, representing the things people commonly do when dealing with stress. Some of these items were inspired by previous theory and literature, including defense mechanisms (e.g., wishful thinking, denial), whereas others were added later at the suggestion of subjects in their studies (e.g., prayer). Exploratory factor analyses and rational item selection led eventually to eight scales assumed to measure distinct ways of coping: Confrontive, Distancing, Self-Controlling, Seeking Social Support, Accepting Responsibility, Escape-Avoidance, Planful Problem Solving, and Positive Reappraisal (Folkman & Lazarus, 1988). These eight coping styles constituted the first and "most commonly used measure of basic coping responses" (Parker, Endler, & Bagby, 1993, p. 361).

The inclusion of cognitive coping styles such as Planful Problem Solving and Accepting Responsibility shows that the conceptual domain of coping (see Figure 20.1) also includes processes that are not aimed at the regulation of affective states per se, such as "analyzing the problem in order to understand it better."

Some scales, however, do fit with the broader notion of affect regulation set out in Figure 20.1. Consider, for example, the scale initially labeled Emphasizing the Positive (Folkman & Lazarus, 1985). Its revised label, Positive Reappraisal, implies a conceptual link to the family of emotion regulation strategies that Gross (1998) called Cognitive Change, especially the Cognitive Reappraisal strategy. Do the seven items on Folkman and Lazarus's (1988) Positive Reappraisal coping scale measure cognitive reappraisal? They include "I was inspired to do something creative"; "I changed or grew as a person in a good way"; "I came out of the experience better than I went in"; "I found new faith"; "I rediscovered what is important in life"; "I changed something about myself"; and "I prayed." These items indeed describe a person who emphasizes positive experiences or behaviors in a stressful situation. However, they do not directly assess the particular process of *changing the personal meaning or appraisal of an emotion-eliciting event*. Instead, these items describe diverse, though generally positive, consequences arising from or after the stressful experience (e.g., "I came out of the experience better than I went in"), including personal growth, self-transformation, greater creativity, and even spiritual renewal.

This complex item content poses serious issues for research on the correlates and adaptive consequences of using a particular coping style. As Lazarus (2000, p. 666) explained, "The danger of confounding is that measures of coping could contain some of the same variables—for example, distress or psychopathology—as the outcome measure of mental health. Thus, if the antecedent and consequent measures are essentially the same, any correlation between them would represent some degree of tautology."

This problem—confounding the predictor or causal variable (i.e., regulation) with subsequent outcomes to be predicted (i.e., better adjustment) within the same item—is a common issue in research on individual differences in affect regulation. It highlights that in writing items researchers should focus item content specifically on the regulatory process of interest. Indeed, among the items listed earlier, only one ("I prayed") describes a potential emotion regulatory activity without mentioning some other, positive outcomes as well.

In addition to confounding, lack of specificity is another potential issue in these items. For example, the item "I prayed" is vague vis-à-vis the particular regulatory process at work: Individuals may pray in order to gain a new perspective or understanding of an emotion-eliciting event, but they may also pray to distract themselves, or to share their feelings with a greater power, or even to muster the inner strength to modify the situation. These uses of prayer and religion for affect regulation may well be distinct and show differential correlates and adjustment outcomes.

These issues also apply to items on other scales (see Folkman & Lazarus, 1985). For example, "I jog or exercise," scored on the Tension Reduction scale, may represent multiple specific regulation strategies, such as situation selection (e.g., avoiding the stressor), attentional deployment (e.g., dis-

tracting oneself), and even response modulation (e.g., going off to jog in order to avoid expressing one's emotion to another person). These items could be more specific regarding the specific regulatory processes intended.

In summary, most items on the Ways of Coping Questionnaire are worded broadly and lack specificity regarding the regulatory process involved. Many items suffer from a confounding problem where regulation and extraneous outcomes (e.g., psychosocial consequences) are combined in the same item. At the scale level, the lack of a process model led to a reliance on exploratory factor analyses of these broad and complex items to define the conceptual building blocks (i.e., the particular coping styles). The resulting concepts (as represented by the scales) are heterogeneous and often combine conceptually distinct regulatory processes. With such complex and global concepts, the scales are hard to interpret and have been criticized for lacking construct validity; discriminant validity has been a particular problem, because intercorrelations between some of the scales tend to be very high, with some exceeding .70 (e.g., Parker et al., 1993).

COPE Inventory

In an important contribution, Carver, Scheier, and Weintraub (1989) substantially reworked the original Folkman and Lazarus scales, using modern psychometric methods. They subdivided some of the most heterogeneous scales, added new scales, and improved the overall conceptual coherence and internal consistency. The resulting Coping Orientations to Problems Experienced Scale (COPE) is a self-report questionnaire that measures 14 coping styles with four items each. Even though the COPE scales are shorter, they are more reliable than the earlier Ways of Coping scales. The COPE covers a broad array of diverse coping styles, such as Planning, Active Coping, Mental Disengagement, Seeking Social Support—Instrumental, Seeking Social Support—Emotional, Positive Reinterpretation and Growth, Turning to Religion, Focus on and Venting of Emotion, and Denial.

However, some of the problems of the Ways of Coping Questionnaire remain. The lack of a process, structural, or conceptual model continues to be a problem. The scales thus continue to define coping styles at rather different levels of abstraction, with some scales (e.g., Turning to Religion) measuring very broad and complex constructs and others (e.g., Denial) measuring much more specific processes. It is not clear how the various coping styles being measured should be conceptually or causally related. And many of the scales remain conceptually heterogeneous; for example, the scale labeled Focus on and Venting of Emotions involves both being aware of one's distress and "letting it out."

Before using these scales, it is important to examine the item content of the COPE scales to understand what they are measuring (e.g., John & Gross, 2007). Some of them can be linked to the specific emotion regulation strategies defined in Gross's (1998) process model. For example, the COPE Active Coping and Planning scales include items that refer to regulatory processes involving situation selection and situation modification. Another example is the scale that Folkman and Lazarus (1988) had initially called Emphasizing the Positive and that Carver et al. (1989) relabeled Positive Reinterpretation and Growth (e.g., looking for the silver lining in stressful situations; trying to learn from difficult experiences). One would expect this coping style to be related to the specific emotion regulation strategy of Cognitive Reappraisal. However, this COPE scale also includes general optimism and longer-term positive changes and adaptations individuals might make later on, *following* the stressful experience. Indeed, the correlation of this COPE scale with Gross and John's (2003) Cognitive Reappraisal scale tends to be moderate (e.g., Gross & John, 2003; Balzarotti, John, & Gross, 2010), consistent with the broader definition of the COPE scale. That is, the inclusion of positive processes and long-term outcomes that have little to do with the immediate regulatory task at hand make the scale complex and conceptually heterogeneous.

This conceptual heterogeneity makes it difficult to establish whether and when a particular coping style is effective or dysfunctional. For example, Carver et al. (1989) had assumed that the Focus on and Venting of Emotions scale would be dys-

functional but instead found its adjustment correlates to be complex; the positive correlation with Social Support suggests some positive adjustment implications. Conceptually, we suggest, this scale combines two distinct processes with potentially different adjustment implications. On the one hand, items like "I get upset, and am really aware of it" are conceptually related to rumination (Nolen-Hoeksema, Parker, & Larson, 1994); focusing on a negative emotion will likely intensify the experience of that emotion further and thus make down-regulation more difficult, leading to *lower* adjustment and well-being. On the other hand, items like "I feel a lot of emotional distress and I find myself expressing those feelings a lot" describe an individual whose emotion experience and expression are congruent (Mauss et al., 2011); congruence between experience and expression, and sharing of emotions with others, can increase authenticity and generate greater closeness with others, leading to *higher* well-being (English & John, 2013). If the research goal is to understand individual differences relevant to affect regulation and how these individual differences are related to important life outcomes, such as well-being and relationship functioning, then less complex measures may be needed to provide conceptual clarity and coherence in findings.

A similar analysis applies to the two Seeking Social Support scales on the COPE—after all, talking to another person about one's problems requires some sharing and expression of feelings (i.e., low suppression) and will serve to focus the individual on these emotions rather than distract him or her from them (i.e., low distraction). In contrast, the Mental Disengagement scale (e.g., coping by turning to work; going to the movies) should relate most to distraction; however, as Carver et al. (1989) noted, these items represent a rather loose set of diverse activities. The inclusion of items about sleep and daydreaming suggest a potential link to situation selection as well, because these activities can be performed for reasons other than distraction (e.g., avoiding the stressful situation).

Drug and alcohol use "in order to think about it less" may also be related to distraction. As noted earlier in our discussion of prayer use, the Turning to Religion scale on the COPE could serve several regulatory strategies; it would be interesting to clarify empirically the processes that account for positive effects of religiosity on well-being, which may include antecedent processes, such as reappraisal or distraction, or even response modulation via lowering suppression, because prayer may offer a safe place for emotional expression by sharing feelings with a trusted higher entity.

Consistent with our earlier observation that coping styles are defined rather broadly, several COPE scales fall clearly outside the scope of the affect regulation domain. For example, the Suppression of Competing Activities scale and the Restraint Coping scale involve behavioral responses that reflect problem-focused coping, showing that coping efforts are often directed at problems or events in the external world that interfere with the individual's goals, even though they do not cause a specific emotional response. Several other COPE scales refer to long-term adjustments some individuals make as a consequence of the stressful experience, a timescale outside the more limited time frame of the immediate emotion regulatory episode. This temporal issue applies to Behavioral Disengagement ("I just give up trying to reach my goal"), Acceptance ("I learn to live with it"; "I get used to the idea that it happened"), Denial ("I act as though it hasn't even happened"), and the Growth component of Positive Reinterpretation and Growth ("I try to grow as a person as a result of the experience").

Cognitive Emotion Regulation Questionnaire

Although its name does not refer to stress and coping, Garnefski and Kraaij's (2007) Cognitive Emotion Regulation Questionnaire (CERQ) also originated in the coping approach. Like the COPE, the goal was to assess a set of regulatory strategies that help people "to keep control over their emotions during or after the experience of threatening or stressful events" (Garnefski & Kraaij, 2007, p. 141). However, the CERQ focuses more narrowly than the COPE on *cognitive* coping strategies, which are said to represent "the conscious, cognitive way of handling the intake of emotionally arousing information" (p. 141).

The CERQ has a format very similar to the COPE and also consists of relatively brief 4-item scales, "each referring to what someone thinks after the experience of threatening or stressful events" (Garnefski & Kraaij, 2007, p. 141). In other words, just like the coping styles on the COPE, these cognitive regulation dimensions focus on stressful situations and have the longer time horizons (e.g., *after* the actual emotional experience) that differentiate coping styles from the specific emotion-regulation processes defined in Gross's model.

Some of the nine CERQ scales were adapted from the COPE and other coping instruments: "These dimensions were defined either by taking out or reformulating the cognitive dimensions of existing coping measures (Carver et al., 1989; de Ridder, 1997), 'transforming' non-cognitive coping strategies into cognitive dimensions or adding new strategies on theoretical grounds" (Garnefski & Kraaij, 2007, p. 142). Coping styles that were clearly cognitive, such as Acceptance, Planning, and Positive Reinterpretation and Growth (renamed Positive Reappraisal) were retained from the COPE. Behavioral and overly complex coping styles from the COPE, such as Use of Social Support, Substance Abuse, Humor, Religious Coping, and even Focus on and Venting of Emotions were left out of the CERQ.

In addition, some regulatory processes from other literatures were added, such as Focus on Thought/Rumination (e.g., "I often think about how I feel about what I have experienced"), Catastrophizing (e.g., "I continually think how horrible the situation has been"), and Self-Blame (e.g., "I feel that I am the one to blame for it"). Again, as in the earlier coping measures, many of the scales do not focus on the regulation of internal experiences; rather, they focus on the external, stressful situation, as illustrated by the items on the Acceptance scale (e.g., "I think that I have to accept the situation"; "I think that I have to accept that this has happened").

In our view, the singular focus on "thinking" aspects in an instrument with nine distinct scales has two potential disadvantages. One is discriminant validity, in the sense that subsets of the scales become relatively similar and thus highly correlated. This problem has been criticized in the two earlier coping style measures as well. For example, the three CERQ scales—Positive Reappraisal ("I think that I can become a stronger person as a result of what has happened"), Refocus on Planning ("I think of what I can do best"), and Putting into Perspective ("I think that it hasn't been too bad compared to other things")—all share elements of cognitive change or reappraisal. Indeed, Garnefski and Kraaij (2007) reported that Positive Reappraisal and Refocus on Planning correlated close to .70 in their adult participants. Unfortunately, the ERQ (or COPE) was not included, thus providing no information about the convergent validity of these three new CERQ scales with earlier measures of cognitive reappraisal.

The second disadvantage of the purely cognitive focus is that emotion researchers agree that emotions have not only experiential but also behavioral–expressive components (e.g., Mauss et al., 2011), and the coherence of these emotion response systems is likely a function of regulatory processes. Therefore, the omission of any scales related to individual differences in response modulation, such as expressive suppression, would seem to make an instrument less useful for research on individual differences in affect regulation.

In conclusion, this comparison of three measures of coping styles and specific emotion regulation processes suggests that there is some overlap between the two approaches. Overall, however, the two approaches are surprisingly distinct. One core difference is that the constructs measured by all the coping questionnaires are defined in broader and more complex terms than the specific emotion regulatory strategies. Several coping styles, especially the problem-focused ones, fall outside the domain of emotion regulation processes. A great methodological strength of the coping approach is its clear focus on stressful situations. Much coping research has studied individual differences within the concrete context of a particular stressful encounter (e.g., preparing for an upcoming test), focusing on the individual's behavioral and cognitive responses in this situation. This contextually grounded approach emphasizes what individuals actually do, or try to do, in a specific context (even when reported on questionnaires or in interviews) and contrasts with global

approaches such as the Big Five trait model (e.g., John et al., 2008) that pay scant attention to the context and instead assess broad trends in usual or typical behavior.

Future coping research may take a more theoretically guided approach to resolve the conceptual complexity and heterogeneity of the coping scales in terms of their affect regulatory implications. Renewed scale construction efforts may address the confounding of regulatory processes and downstream adjustment outcomes within the same questionnaire item. Finally, because the coping approach and the specific emotion regulation processes approach differ in terms of their time horizons and relevant situational contexts, each has unique advantages and applications. The various measures of coping styles provide a useful focus on stressful situations and regulating negative affective states.

The Emotional Competence Approach and Its Major Measures

The *emotional competence* approach originated in developmental and clinical analyses of what a child needs to learn to become an emotionally and socially competent adult. Saarni (e.g., 1999, 2011) analyzed emotional functioning from the perspective of how well it serves the adaptive and instrumental goals of the individual, then defined emotional competence as a set of affect-oriented behavioral, cognitive, and regulatory skills. Simply put, the child needs to learn what it means to feel something and to do something about those feelings.

Saarni (1999, 2011) postulated eight broad skills that she considers to be prerequisites for emotional competence: (1) awareness of one's own emotional state; (2) the skills to discern and understand the emotions of others; (3) skill in using the common vocabulary of emotion and expression; (4) capacity for empathic and sympathetic involvement in others' emotional experiences; (5) ability to realize that one's inner emotional state need not correspond to outer expression; (6) capacity for adaptive coping with aversive or distressing emotions by using self- regulatory strategies that ameliorate the intensity or temporal duration of such emotional states; (7) awareness that relationships are defined by emotional genuineness of expressive display and reciprocity; and (8) capacity for emotional self-efficacy (i.e., individuals can accept their own emotional experiences and view themselves as generally feeling the way they want to feel). Although not necessarily accepted by all, these eight emotional competencies provide a formidable set of complex socioemotional skills that researchers in this tradition consider to be part of affect regulation. Note that from the perspective of the specific emotion regulation process approach, the capacity for adaptive coping would relate most directly to individual differences in affect regulation. The other competencies seem to specify processes that make emotion regulation possible or may help (rather than hinder) the enactment of effective regulatory strategies. For example, if an individual has no awareness of his or her current emotional state, then that person would hardly proceed with attempts to regulate that emotion.

Generalized Expectancies for Negative Mood Regulation Scale

The Generalized Expectancies for Negative Mood Regulation Scale (NMR) was developed by Catanzaro and Mearns (1990) and is one of the earliest measures to take the emotional competence approach. It now has fallen out of use. This 30-item self-report questionnaire focuses on individuals' *beliefs* that some "behavior or cognition will alleviate a negative state or induce a positive one" (p. 547), and asks them to indicate the extent to which they believe their attempts to alter their negative moods will work.

Many of the items focus on ways to eliminate or at least avoid negative emotions. Thus, the measure has been criticized for equating mood regulation with the avoidance of negative affect (e.g., Gratz & Roemer, 2004); simply avoiding negative states is assumed to be an indication of effective regulation, as shown by items such as "When I'm upset, I believe that I can forget about what's upsetting me pretty easily" versus "When I'm upset, I believe that I won't be able to put it out of my mind" (reverse scored). Moreover, the NMR does not assess aspects of mood regulation, such as awareness, clarity, or acceptance of emotions, that later emotional competence

researchers, such as Saarni (1999), Salovey et al. (1995), and others, deemed important. Finally, regulatory processes are not differentiated but are instead measured only in terms of whether the individual thinks he or she has access to mood regulation strategies perceived to be effective.

Trait Meta-Mood Scales

Consistent with their emotional competence perspective, Salovey et al. (1995) aimed to understand the reflective (or meta) processes that accompany many mood states. These "meta-mood" processes capture how individuals reflect on their feelings, including how they monitor, evaluate, and regulate them (Mayer & Gaschke, 1988). Salovey et al. (1995) assumed that emotions serve as an important source of information, and that individuals differ in how skilled they are at processing this kind of information, particularly in "their understanding of and ability to articulate their affective states" and their ability to "regulate such feelings and use them adaptively to motivate behavior" (p. 147). The Trait Meta-Mood Scales (TMMS) were designed to measure stable and general attitudes about moods and the degree to which individuals attempt to manage (or repair) mood experiences. The TMMS measures three constructs—people's tendency to attend to their moods and emotions (attention), to discriminate clearly among them (clarity), and to regulate them (repair)—each capturing individual differences considered "fundamental to the self-regulatory domain of emotional intelligence" (p. 147).

Given that the TMMS focuses on individual differences in meta-moods—that is, thoughts and attitudes that accompany mood experiences—we would not expect any links to regulatory strategies that occur earlier in the emotion process, prior to the onset of an emotional episode itself, such as situation selection or modification. However, attentional processes, cognitive change, and response modification should be of considerable relevance.

The TMMS Attention scale refers to paying close attention to feelings, accepting feelings, valuing them positively, and letting oneself experience them fully and intensively, using items such as "I often think about my feelings" versus "I don't think it's worth paying attention to your emotions or moods" (reverse scored). As expected, the Attention scale correlates with the Private Self-Consciousness scale, which measures awareness and attention to private aspects of the self (e.g., thoughts and feelings). One would expect the Attention scale to relate negatively to chronic use of the emotion regulation strategies distraction and suppression. The link to distraction is theoretically interesting, because paying close attention to negative mood states has been shown to magnify and intensify the experience of negative affect and the risk for depression (Scheier & Carver, 1977), whereas in our model, distraction is expected to decrease negative emotion experience. Indeed, in one of the studies by Salovey et al. (1995), the Attention scale was related to higher depression scores. Regarding the negative link to suppression, being intensely aware of and paying close attention to one's emotions should interfere with the considerable and ongoing cognitive effort required to suppress one's emotions effectively (Richards & Gross, 2000), and that should be especially true for individuals who experience their emotions intensely. Moreover, individuals scoring high on the Attention scale value their feelings and believe in letting them guide their behavior. That is the opposite of individuals who habitually use suppression and thus show expressive behavior that is inconsistent with their inner feelings (Gross & John, 2003). Indeed, the TMMS Attention scale correlated negatively with the ERQ Suppression scale.

The TMMS Clarity scale assesses clarity about one's feelings, and contrasts awareness and acceptance of feelings with confusion about feelings and their meaning and implications. True-scored item examples include "I feel at ease about my emotions" versus false-scored items such as "I can't make sense out of my feelings." Low clarity was related not only to ambivalence about emotional expression but also to vulnerability to negative affect, such as neuroticism, distress, and depression. Salovey et al. (1995) conceptually linked lack of clarity to ruminative thought processes (e.g., individuals who do not know how they feel about a negative event have to keep thinking about it) and showed empirically that, after watching a stressful video, individuals scoring high on

the Clarity scale exhibited a decline in ruminative thought and recovered their positive mood to a greater extent than low-scoring individuals. These findings suggest a positive relation between clarity and distraction (the ability to use attentional resources to move away from a negative emotion stimulus, rather than ruminating about it), and a negative relation with suppression. Individuals who are clear about and comfortable with their emotions should feel little need to suppress their behavioral expression of emotion, and the evidence supported this predicted negative relation (Gross & John, 2003).

The TMMS Repair scale assesses attempts to improve negative mood by thinking positively and taking an optimistic (rather than pessimistic) attitude more generally. Item examples include "Although I am sometimes sad, I have a mostly optimistic outlook" and "I try to think good thoughts no matter how badly I feel," contrasted with "Although I am sometimes happy, I have a mostly pessimistic outlook" (reverse scored). This scale seems conceptually similar to coping styles reviewed earlier, such as Emphasizing the Positive on the original Ways of Coping Questionnaire and Positive Reinterpretation and Growth on the COPE.

As expected, the Repair scale correlated substantially with general optimism and low vulnerability to distress and depression; it also predicted lower levels of distress after participants watched a stressful video. In terms of specific emotion regulation processes, one would expect the Repair scale to relate positively to the use of distraction, as well as reappraisal: The explicit mood repair efforts included in the scale involve (1) using thought to focus on something other (e.g., pleasant things; good thoughts) than the distressing stimulus, thus implicating distraction, and (2) also trying to think differently (e.g., more positively) about the situation, thus implicating reappraisal. In contrast, expressive suppression is hardly an optimistic process (Gross & John, 2003); its habitual use is related not only to negative emotion, as shown in Figure 20.5, but also to feelings of inauthenticity (English & John, 2013) and the pessimistic expectation that others will disapprove if shown one's "real self," as shown as in Table 20.1. Consistent with these considerations, Gross and John (2003) found that the TMMS Repair scale was differentially related to the ERQ Reappraisal (r = .36) and Suppression (r = −.26) scales.

TABLE 20.1. Examples of Beliefs and Motivations Predicting Individual Differences in the Habitual Use of Reappraisal and Suppression as Measured by the ERQ

	Emotion regulation strategy	
Construct studied	Reappraisal	Suppression
Incremental (vs. entity) theory of emotion[a] (If they want to, people can change the emotions they have)	.35	.04
Global emotion regulation self-efficacy[a] (I certainly can control my emotions when running into a former boyfriend/girlfriend)	.21	.00
Subjective authenticity[b] (It's important to me to be true to myself)	−.02	−.30
Fear of negative consequences[c] (I worry that if I express negative emotions, such as fear and anger, other people will not approve of me)	−.05	.31
Low self-efficacy for self-expression[c] (At times I am just not able to express what I am really feeling)	−.12	.45

Note. ERQ, Emotion Regulation Questionnaire (Gross & John, 2003).
[a]Tamir, John, Srivastava, and Gross (2007, Study 1).
[b]English and John (2013; mean across Studies 1–3).
[c]John (2010).

However, the correlations of the ERQ scales and the TMMS scales were not large in size. Suppression was related negatively to all three TMMS scales, most strongly to Attention ($r = -.41$) and least strongly to Repair ($r = -.26$). This is interesting and worth further study for two reasons. First, we need to understand better how individual differences in cognitive variables such as attention to emotion are linked to behavioral variables, such as emotion-expressive behavior. Second, we need to address questions about causal order: How are cognitive variables such as attention best conceptualized? In our view, some may be antecedents (see Figure 20.6 below): Individuals may use suppression frequently because they have learned to devalue and ignore their emotions as important sources of information, and because they have the pessimistic belief that others will respond negatively if they truly express their feelings. Other meta-mood processes may represent consequences: Frequent use of suppression leads to expressive behavior that is incongruent with internal feeling states, and may in turn lead to confusion and lack of clarity about one's emotions (and eventually an unclear self-concept and low self-esteem).

These considerations imply that research on individual differences in beliefs and motivational factors will be critical in helping us conceptualize how meta-mood processes and specific emotion regulatory processes are related. Table 20.1 summarizes the findings from several initial studies. Incremental beliefs that emotions can be changed, and self-efficacy beliefs about one's own ability to change one's emotions, predicted greater use of cognitive reappraisal, whereas subjective inauthenticity, fear of negative consequences, and low self-efficacy predicted greater use of suppression.

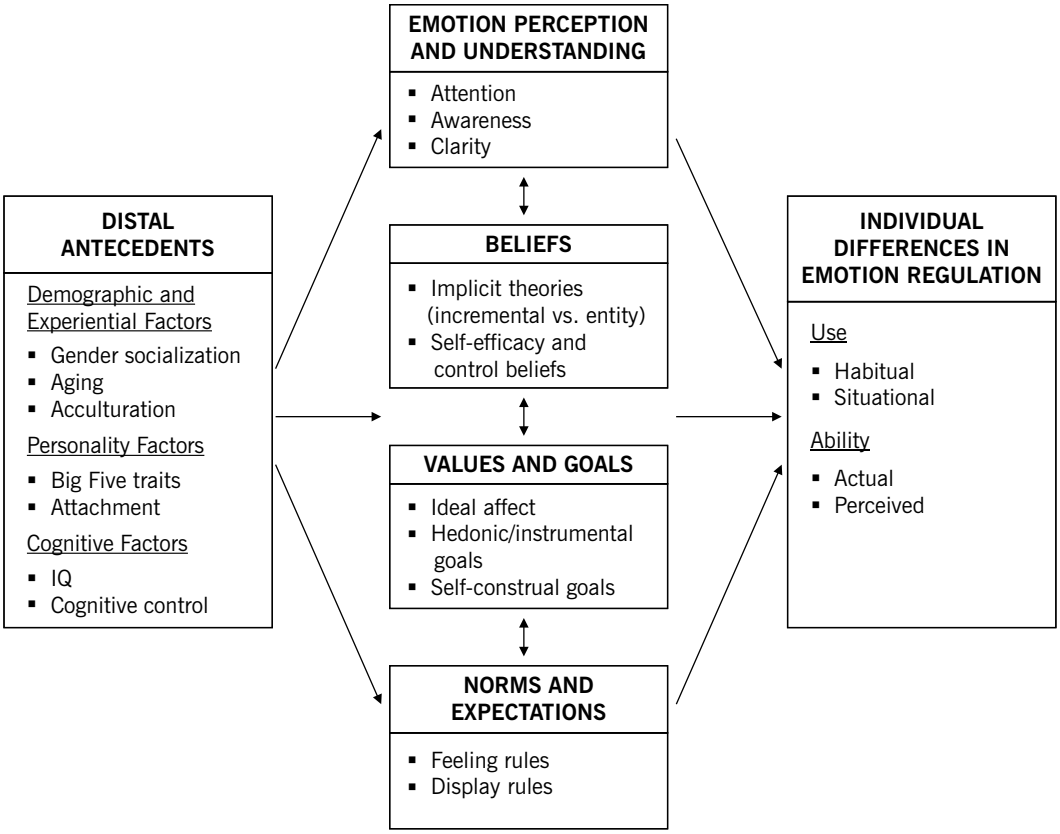

FIGURE 20.6. Examples of several types of individual differences in emotion regulation and their potential antecedents.

Difficulties in Emotion Regulation Scale

Gratz and Roemer (2004) defined emotion regulation competencies almost as broadly as did Saarni (1999), whose influence they acknowledge: "(a) awareness and understanding of emotions, (b) acceptance of emotions, (c) ability to control impulsive behaviors and behave in accordance with desired goals even when experiencing negative emotions, and (d) ability to use situationally appropriate regulation strategies flexibly to modulate emotional responses as desired in order to meet individual goals and situational demands" (p. 42).

In devising measures of these four competency constructs, Gratz and Roemer (2004, see p. 44) were influenced by the content of Salovey et al.'s (1995) TMMS scales, as well as by the older NMR, which they used as a template to structure the format of some of the items. Specifically, they wrote their items assessing difficulties with regulating emotions during times of distress using the same general format as the NMR. Thus, many of their items begin with the sentence stem "When I'm upset, I . . . " This focus on a global negative affect (upset) is a feature that both the NMR and the Difficulties in Emotion Regulation Scale (DERS) share with the coping scales discussed earlier.

The 36-item DERS has six subscales. Note that because the authors are interested in dysregulation and its link to psychopathology, they score all the subscales in terms of *difficulties* with various competencies considered important for emotion regulation. Possibly as a result of this naming convention, several of the DERS subscales have vague or confusing names. It is thus imperative to review the items that make up each scale to ascertain what is being measured.

Two subscales seem rather similar to existing TMMS scales but label the opposite pole: (1) Lack of Emotional Awareness (e.g., "I pay *attention* to how I feel"—reverse scored; emphasis added) captures the low pole of the TMMS Attention scale; (2) Lack of Emotional Clarity (e.g., "I am *clear* about my feelings"—reverse scored; emphasis added) is very similar to the low pole of the TMMS Clarity scale.

Two other subscales seem to assess coping styles: (3) Nonacceptance of Emotional Responses (e.g., "When I'm upset, I feel *ashamed* with myself for feeling that way"; emphasis added) seems similar to ineffective coping scales such as Self-Blaming, which might be a simpler yet clearer label for this subscale; (4) Difficulties Engaging in Goal-Directed Behavior (e.g., "When I'm upset, I have difficulty *thinking about anything else*"; emphasis added) seems to involve not goal-directed behavior per se but ineffective attentional deployment (Gross, 1998) and resembles what Nolen-Hoeksema et al. (1994) conceptualized as *ruminative coping* (i.e., difficulty shifting attention and disengaging from a preoccupying affective state).

Finally, (5) the Impulse Control Difficulties subscale (e.g., "When I'm upset, I become out of control") is similar to Impulse Strength or Intensity scales (e.g., Gross & John, 1997, 1998) that describe individuals who have very strong feelings that are hard to control; and (6) the subscale Limited Access to Effective Emotion Regulation Strategies (e.g., "When I'm upset, I *believe* that there is nothing *I can do* to make myself feel better" and "When I'm upset, I *believe* that wallowing in it is all *I can do*"; emphases added) is similar to the earlier NMR Belief scale and seems to capture the low pole of Tamir, John, Srivastava, and Gross's (2007) measure of Emotion Regulation Self-Efficacy Beliefs (see Table 20.1).

As with the NMR and the TMMS, these six subscales do not differentiate between kinds of regulatory strategies. As with the coping measures in the previous section, discriminant validity has been noted as a potential problem with the DERS, because some of the scale intercorrelations exceed .60 and the subscales tend to correlate similarly with indicators of distress or dysfunction (e.g., Weinberg & Klonsky, 2009). All six subscales are often simply summed to obtain an overall dysregulation score.

The DERS (and its Adolescent version) is increasingly being used in studies of adult pathology and child disorders; findings show that scores on the DERS are correlated with rather diverse psychological problems, such as depression, anxiety, suicidal ideation, eating disorders, alcohol use, and drug use (e.g., Weinberg & Klonsky, 2009). Such a sweeping pattern of maladjustment correlates may indicate that the DERS assesses a rather undifferenti-

ated self-presentation as a troubled person: Such individuals are overwhelmed by strong affective impulses they do not understand, and they feel no confidence (self-efficacy) that they can control these impulses. Without some evidence of discriminant validity, it is difficult to interpret correlations of the DERS with indicators of psychopathology. Overall, it is not clear which of these six subscales was intended to capture the core regulatory concept defined as the "ability to use situationally appropriate regulation strategies flexibly to modulate emotional responses" (Gratz & Roemer, 2004, p. 42). Certainly, use of the DERS for individual cases in clinical and applied contexts seems premature.

Emotional Intelligence Test

We have seen repeatedly that the emotional competence approach conceptualizes emotion regulation in terms of a number of specific *abilities*. Yet all the measures we have discussed so far have used self-report questionnaire methodology: that is, researchers assess self-perceptions (including self-efficacy beliefs) and typical experiences and behaviors. In fact, we have seen that some of these "competence" scales can be difficult to differentiate from coping styles. This is hardly a foolproof way to assess ability constructs, which are usually measured in terms of carefully controlled maximum performance tests of the behavior or process in question.

Considerable credit is due to Mayer, Salovey, and Caruso (2002), who acknowledged this methodological inconsistency and tackled the difficult task of constructing a test of emotional intelligence that is scored in terms of correct and incorrect answers, namely, the Mayer–Salovey–Caruso Emotional Intelligence Test (abbreviated MSCEIT). They define *emotional intelligence* as a set of skills involved in the processing of emotion-relevant information. Due to space limitations, of the four MSCEIT components, we briefly address only the component that is most relevant here, namely, *emotion management ability*, which they define as the capacity to reduce, increase, or maintain particular emotions in both oneself and other people. The tasks they use to measure these abilities require respondents to react to hypothetical scenarios and evaluate the effectiveness of various behaviors and subjective construals for emotion management purposes. For example, participants are asked to judge the effectiveness of strategies to help a friend enhance a joyful mood or reduce feelings of sadness.

Unfortunately, scientific evaluation and research use of the MSCEIT are hindered, because the test is owned by a commercial "test publisher (who) does not authorize reproduction of actual test items" (e.g., Lopes, Salovey, Côté, & Beers, 2005, p. 114); thus, we are allowed to provide here only two abridged examples that were published in this form by Lopes et al. (2005, pp. 114–115), each consisting of a vignette paired with separate response options:

> Debbie just came back from vacation. She was feeling peaceful and content. How well would each action preserve her mood? (1) She started to make a list of things at home that she needed to do. (2) She began thinking about where and when to go on her next vacation. (3) She called a friend to tell her about the vacation . . .

> Ken and Andy have been good friends for over 10 years. Recently, however, Andy was promoted and became Ken's manager. Ken felt that the new promotion had changed Andy in that Andy had become very bossy to him. How effective would Ken be in maintaining a good relationship, if he chose to respond in each of the following ways? (1) Ken tried to understand Andy's new role and tried to adjust to the changes in their interactions. (2) Ken approached Andy and confronted him regarding the change in his behavior . . .

These examples are from the fourth (Managing Emotions) "branch" of the MSCEIT, which comprises two distinct tasks. Five vignettes measure *ability in emotion management*, and each describes a person (like Debbie) who is experiencing a mood or emotion. For each of the five vignettes, the respondent rates (on a 5-point scale) how effective four different actions would be for obtaining a specified effect on the person's experience (here, to preserve Debbie's good mood), yielding a total of 20 separate ratings. The second task measures *emotional relationship abilities* and comprises three vignettes describing relationships between persons (like Ken and Andy). In each

vignette, the respondent rates how effective three different actions would be *to maintain a good relationship* between the persons, for a total of nine separate ratings. Each of the 29 individual ratings is scored according to normative effectiveness ratings provided by a panel of emotion experts or the group consensus.

Although abbreviated, these two examples are very instructive. First, they illustrate that the total emotion management ability score includes more than 30% of ratings that assess not emotional but relational skills, raising questions about content validity. Second, each vignette and action includes a lot of detailed contextual information specific to that rating, which adds error and keeps interitem correlations, and thus reliability, low; with 29 ratings aggregated into the total score, reliability in this study was a measly .63, and that is higher than in other studies (see Føllesdal & Hagtvet, 2009, for a thoughtful psychometric analysis and critique). Third, as the MSCEIT authors readily acknowledge, these vignette ratings do not actually measure individual differences in skillful or effective regulation scored or observed objectively in an emotional situation; instead they tap the individual's *knowledge, and capacity to reason*, about emotions and emotional situations (e.g., Lopes et al., 2005, p. 114). Fourth, the emphasis on knowledge and complex reasoning processes is likely to introduce correlations with measures of other abilities, creating discriminant validity problems. Fifth, because there are interpersonal themes even in the emotion management vignettes (e.g., calling a friend to share one's mood, thus capitalizing on the experience), performance on these items may yield surprising correlations with personality variables, again introducing potential problems with discriminant validity. In response to these discriminant validity concerns, the test authors and their collaborators have tried to demonstrate that the MSCEIT predicts social, emotional, and leadership outcomes even when intelligence and broad personality traits are controlled. So far, however, many researchers have remained unconvinced; the MSCEIT, and emotional intelligence research more generally, is viewed with some skepticism among researchers (e.g., Landy, 2005; Joseph & Newman, 2010).

For example, Lopes et al. (2005) obtained eight criterion measures (e.g., interpersonal sensitivity; socioemotional competence; friendship nominations) with self-ratings or peer nominations. When the personality traits measured by the Big Five Inventory (John et al., 2008) were controlled, the MSCEIT emotion management ability scale still significantly predicted two of these criteria. However, with two out of eight significant correlations, it is hard to say whether the predictive validity goblet is a quarter full or three quarters empty. Of greater interest to personality researchers is a finding not highlighted by the authors: By far the highest correlation (r = .40) was not found with any of the eight predicted socioemotional outcome measures but, somewhat unexpectedly, with the Big Five trait of Agreeableness.

In conclusion, the MSCEIT, though an admirable and conceptually interesting undertaking, has not proven to be the decisive fix for the less compelling self-report measures of emotional "competencies" that have come before it. Even though, as outsiders, we do not know much about the inner workings of the MSCEIT, it seems unlikely that a measure consisting of only five emotion management vignettes and three relationship vignettes can provide the conceptual building blocks needed to construct a comprehensive conception of emotion regulation in terms of specific competencies. More generally, the emotional competence approach (e.g., Saarni, 1999) includes all manner of cognitive, behavioral, self-perception, and emotion perception processes under one broad rubric. This conceptual richness and reach, and the resulting complexity, is theoretically appealing but may be keeping this approach from achieving its full empirical potential. Fewer constructs, more narrowly delineated distinctions, and tighter links between construct definitions and actual measures (and items) may prove a fruitful avenue for future research on the vast differences in people's emotional competence.

Continuing Issues, Limitations, and Future Directions

In this brief and necessarily limited review, we have covered almost 50 scales purporting to measure individual differences in affec-

tive regulation. Our review suggests that the most recent arrivals show a great deal of redundancies with their forebears, such as the newer CERQ with the older COPE, and the newer DERS with the older NMR and TMMS. In our view, the time has come to stop the proliferation of self-report scales and to begin a moratorium that refocuses on solid theory development.

The Need for an Integrative Framework and for Mediation Research

In addition to a great number of affect regulation concepts and scales, we have touched upon a number of factors that predict or influence individual differences in emotion regulation. As a first, modest step toward a broader framework, we have organized these variables in the first two columns of Figure 20.6. In the first column we have included broad person factors that we expect to shape the acquisition and use of particular emotion regulation strategies, such as gender socialization, acculturation, and aging, all of which have been shown to predict individual differences in emotion regulation in adulthood. Broad personality factors, attachment, and cognitive ability factors also need to be considered, even if only as control variables, as we have seen in the debate over the validity and utility of the MSCEIT.

The effects of gender, acculturation, and aging on regulation are unlikely to be direct and thus will need to be explained. In research on naturally occurring individual differences, explanatory research usually takes the form of mediation designs, such as the one depicted in Figure 20.4. In this example, the increasing adoption of culturally transmitted values associated with independent (but not interdependent) self-construal is the mechanism through which greater acculturation to Western practices and values leads Asian immigrants to use suppression less and less frequently the longer they live in the United States. Another example is a recent series of studies showing that the link between habitual suppression and negative social consequences (see Figure 20.5; consequences of regulation are not shown in Figure 20.6) can be explained by individual differences in authenticity in young and older adults, as well as in samples from the United States and China (English & John, 2013).

More generally, we recommend that the field increase recent efforts (see Table 20.1) to link values, goals, and other motivational factors, as well as beliefs, norms, and expectations, to emotion regulation, both conceptually and empirically. We have organized these social-cognitive processing characteristics into four boxes in the middle column of Figure 20.6, with arrows indicating that these concepts have the greatest *promise* for explanatory accounts.

Finally, Figure 20.6 also illustrates our hypotheses about where cognitive meta-mood processes may belong in such an integrative framework. In particular, attention to emotional experience, awareness of emotional experience, and clarity of emotional experience do not seem to us to be isomorphic with regulatory processes. Rather, these cognitive meta-mood processes seem to be important antecedents that can trigger, help, or hinder the use of various, more or less effective regulatory processes. For example, Scheier and Carver (1977) showed that focusing attention on an emotional state can result in both intensification of that state and clarification; intensification may make subsequent regulation efforts less effective, whereas greater clarity may have the opposite effect. In other words, the nature and causal direction of the relationships among the concepts in Figure 20.6 needs to be tested empirically.

The Need to Expand Beyond Habitual Measures of Individual Differences

It is time to acknowledge some limitations in our coverage and in the work presented here. We have focused almost exclusively on habitual differences in affective regulation, which implies that the individual differences under study show at least some consistency over time and may in fact be chronic in nature. It is true that in all three approaches reviewed here, habitual concepts represent the lion's share of the work, but other kinds of individual differences in emotion regulation have been studied (see the third column of Figure 20.6). For example, a number of recent studies have examined situationally

flexible and spontaneous use of emotion regulation as well as abilities. Using a longitudinal design, Srivastava et al. (2009) explicitly differentiated habitual or stable use from dynamic use of suppression during the transition from high school and home town to college; they found considerable change during this transition, with suppression use temporarily increasing as students constructed new friendship networks. Feinberg, Willer, Antonenko, and John (2012) studied spontaneous, rather than habitual, emotion regulation. In one study, they had subjects make moral judgments about a variety of disgusting (but not unethical) behaviors; coding thought protocols, they tested whether some individuals had spontaneously used reappraisal to down-regulate their disgust response and, in turn, made less affectively driven and therefore less moralistic judgments about the behaviors.

We have also not been able to cover recent efforts to extend ERQ work to the domain of individual differences in emotion regulation *ability*, such as actual differences in the effectiveness of reappraisal as assessed with laboratory procedures (e.g., McRae et al., 2012; Troy, Willhelm, Shallcross, & Mauss, 2010) and self-perceived differences in effectiveness in implementing an instructional set in the laboratory (e.g., Gruber, Harvey, & Gross, 2012). This is exciting work, and we are looking forward to seeing more diverse individual-difference measures that go beyond the existing measures of habitual emotion regulation.

Limitations and Future Directions in Research on Specific Emotion Regulation Processes

Finally, lest we forget, we must acknowledge some fundamental limitations of previous research using the ERQ. Whereas much of the work in the coping approach has focused specifically on negative affect and stressful situations, work using the ERQ (e.g., English et al., 2012) has studied individual differences more generally, measuring reappraisal and suppression at the global regulatory process level, shown as Level 2 in the hierarchical model in Figure 20.7. Certainly, the level of specific regulatory processes is preferable to the even more abstract level of *general emotional control* (Level 1). However, the choice of Level 2 (e.g., Gross & John, 2003) should not be taken as an indication that theorists believe regulatory processes operate exactly in the same way for positive and negative emotions (Level 3), or that there are no important regulatory differences (at Level 4) between discrete negative emotions, such as anger and fear, or positive emotion, such as pride and joy (e.g., Shiota, Keltner, & John, 2006). Indeed, theoretical accounts of Asian–White differences in emotion suggest that Asians' greater use of suppression is particularly pronounced for emotions that are positive and highly activated, such as pride and love. Along similar lines, Larsen (2000) suggested that suppression might be "more effective for controlling affects with clear expressive components, such as angry moods, compared to affects that are not clearly expressive, such as loneliness" (p. 138). Webb et al. (2012) note that even in the experimental literature few studies compared the effectiveness of a particular strategy in regulating different emotions. This is certainly an important new direction in research on individual differences as well.

Indeed, as shown in Figure 20.7, our conceptual framework needs to be extended even further downward to include contextual factors (at Level 5) that change the meaning of the emotion for the individual; for example, an Asian American feeling pride in a collective context (e.g., feeling proud of the achievements of a younger sibling) should suppress much less than he or she would in an individualistic context (e.g., having won a prize for personal achievement). Finally, research on individual differences with the ERQ has focused on two important regulatory processes, reappraisal and suppression. Future research needs to extend this focus along the horizontal dimension of Level 2 shown in Figure 20.7 and add exemplars of the other three families of regulatory processes in Gross's process model (see Figure 20.2), namely, situation selection, situation modification, and attention deployment. We have discussed these processes conceptually throughout this chapter, but we have not yet measured them in terms of individual differences. In brief, there is plenty left to do. It is an exciting time to be working on individual differences in affect regulation.

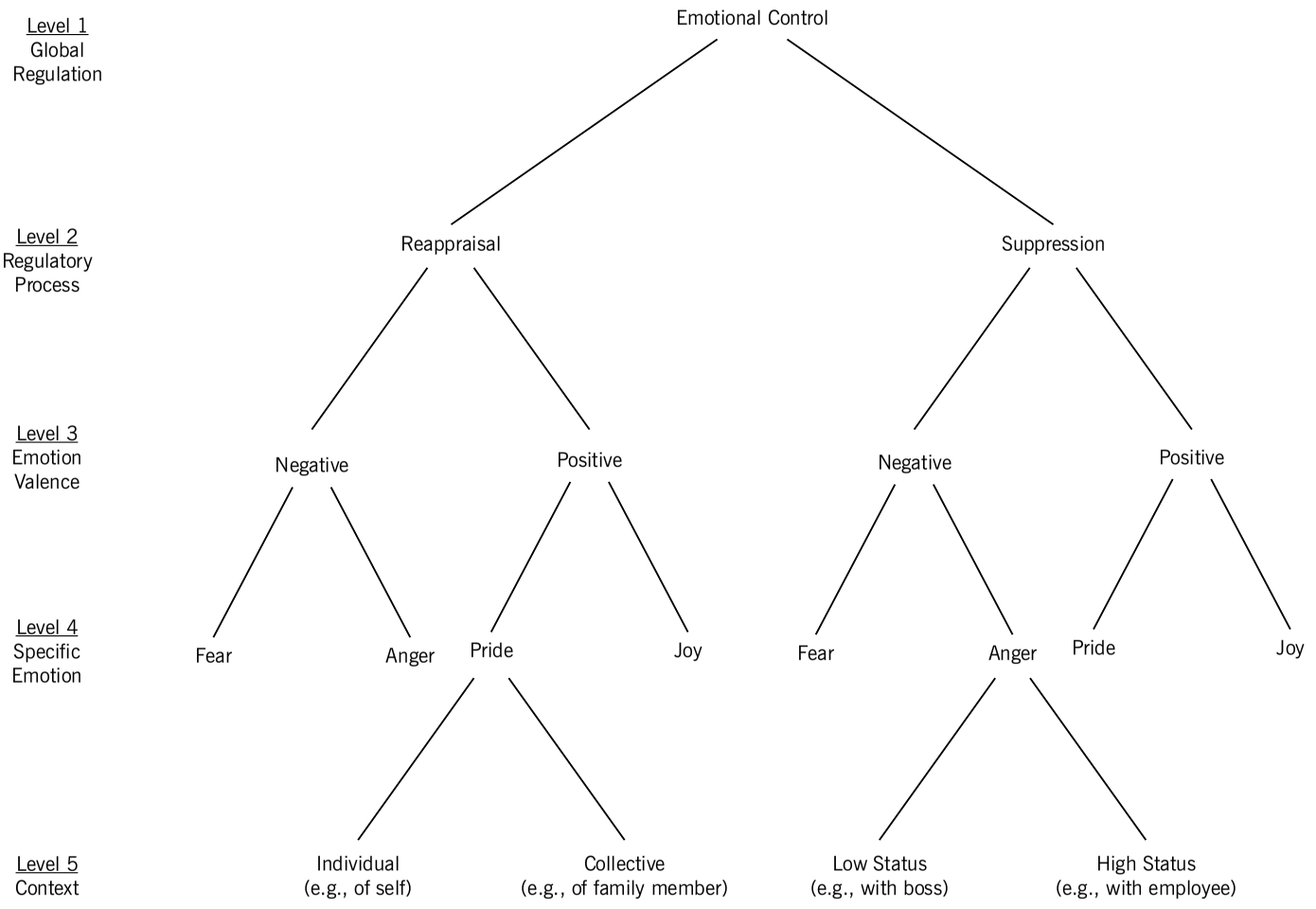

FIGURE 20.7. Hierarchical framework for the study of individual differences in emotion regulation. Emotion regulation is conceptualized at five levels of abstraction: (1) global emotional control, (2) particular regulatory processes, (3) regulation of positive and negative emotion, (4) regulation of specific emotions, and (5) regulation of specific emotions in particular contexts.

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References

Abler, B., & Kessler, H. (2009). Emotion Regulation Questionnaire: A German version of the ERQ by Gross & John. *Diagnostica, 55*, 144–152.

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*, 217–237.

Anderson, C., John, O. P., Keltner, D., & Kring, A. M. (2001). Who attains social status? Effects of personality and physical attractiveness in social groups. *Journal of Personality and Social Psychology, 81*, 116–132.

Balzarotti, S., John, O. P., & Gross, J. J. (2010). An Italian adaptation of the Emotion Regulation Questionnaire. *European Journal of Psychological Assessment, 26*, 61–67.

Bariola, E., Gullone, E., & Hughes, E. K. (2011). Child and adolescent emotion regulation: The role of parental emotion regulation and expression. *Clinical Child and Family Psychological Review, 14*, 198–212.

Baumeister, R. F., Zell, A. L., & Tice, D. M. (2007). How emotions facilitate and impair self-regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 408–426). New York: Guilford Press.

Block, J. H., & Block, J. (1980). The role of ego-control and egoresiliency in the organization of behavior. In W. A. Collins (Ed.), *Minnesota Symposia on Child Psychology* (Vol. 13, pp. 39–101). Hillsdale, NJ: Erlbaum.

Carver, C. S., & Scheier, M. F. (1994). Situational coping and coping dispositions in a stressful transaction. *Journal of Personality and Social Psychology, 66*, 184–195.

Carver, C. S., Scheier, M. F., & Weintraub, J. K. (1989). Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology, 56*, 267–283.

Catanzaro, S. J., & Mearns, J. (1990). Measuring generalized expectancies for negative mood regulation: Initial scale development and implications. *Journal of Personality Assessment, 54*, 546–563.

de Ridder, D. (1997). What is wrong with coping assessment?: A review of conceptual and methodological issues. *Psychology and Health, 12*, 417–431.

Ekman, P. (1972). Universals and cultural differences in facial expression of emotion. In J. Cole (Ed.), *Nebraska Symposium on Motivation* (pp. 207–283). Lincoln: University of Nebraska Press.

Eng, J., Akutsu, S., Gross, J. J., & John, O. P. (2013). *Emotion regulation in East Asian and European Americans: Effects of regulatory strategy, emotion, and independent construal.* Unpublished manuscript, Department of Psychology, University of California, Berkeley.

English, T., & John, O. P. (2013). Understanding the social effects of emotion regulation: The mediating role of authenticity for individual differences in suppression. *Emotion, 13*, 314–329.

English, T. E., John, O. P., & Gross, J. J. (2013). Emotion regulation in close relationships. In J. A. Simpson & L. Campbell (Eds.), *The Oxford handbook of close relationships* (pp. 500–513). Oxford, UK: Oxford University Press.

English, T. E., John, O. P., Srivastava, S., & Gross, J. J. (2012). Emotion regulation and peer-rated social functioning: A 4-year longitudinal study. *Journal of Research in Personality, 46*, 780–784.

Feinberg, M., Willer, R., Antonenko, O., & John, O. P. (2012). Liberating reasons from passion: Overriding intuitionist moral judgments through emotion reappraisal. *Psychological Science, 23*, 788–795.

Folkman, S., & Lazarus, R. S. (1980). An analysis of coping in a middle-aged community sample. *Journal of Health and Social Behavior, 21*, 219–239.

Folkman, S., & Lazarus, R. S. (1985). If it changes it must be a process: Study of emotion and coping during three stages of a college examination. *Journal of Personality and Social Psychology, 48*, 150–170.

Folkman, S., & Lazarus, R. S. (1988). *Manual for the Ways of Coping Questionnaire.* Palo Alto, CA: Consulting Psychology Press.

Føllesdal, H., & Hagtvet, K. A. (2009). Emotional intelligence: The MSCEIT from the perspective of generalizability theory. *Intelligence, 37*, 94–105.

Garnefski, N., & Kraaij, V. (2007). The Cogni-

tive Emotion Regulation Questionnaire: Psychometric features and prospective relationships with depression and anxiety in adults. *European Journal of Psychological Assessment, 23*, 141–149.

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the Difficulties in Emotion Regulation Scale. *Journal of Psychopathology and Behavioral Assessment, 26*, 41–54.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*, 271–299.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gross, J. J., & John, O. P. (1997). Revealing feelings: Facets of emotional expressivity in self-reports, peer ratings, and behavior. *Journal of Personality and Social Psychology, 72*, 435–448.

Gross, J. J., & John, O. P. (1998). Mapping the domain of emotional expressivity: Multimethod evidence for a hierarchical model. *Journal of Personality and Social Psychology, 74*, 170–191.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*, 348–362.

Gross, J. J., & Levenson, R. W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64*, 970–986.

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York: Guilford Press.

Gross, J. J., Richards, J. M., & John, O. P. (2006). Emotion regulation in everyday life. In D. K. Snyder, J. A. Simpson, & J. N. Hughes (Eds.), *Emotion regulation in families and close relationships: Pathways to dysfunction and health* (pp. 13–35). Washington, DC: American Psychological Association.

Gruber, J., Harvey, A. G., & Gross, J. J. (2012). When trying is not enough: Emotion regulation and the effort-success gap in bipolar disorder. *Emotion, 12*, 997–1003.

Gullone, E., & Taffe, J. (2012). The Emotion Regulation Questionnaire for Children and Adolescents (ERQ–CA): A psychometric evaluation. *Psychological Assessment, 24*, 409–417.

John, O. P., & Gross, J. J. (2004). Healthy and unhealthy emotion regulation: Personality processes, individual differences, and lifespan development. *Journal of Personality, 72*, 1301–1334.

John, O. P., & Gross, J. J. (2007). Individual differences in emotion regulation strategies: Links to global trait, dynamic, and social cognitive constructs. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 351–372). New York: Guilford Press.

John, O. P., Naumann, L. P., & Soto, C. J. (2008). Paradigm shift to the integrative Big Five trait taxonomy: History, measurement, and conceptual issues. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (pp. 114–158). New York: Guilford Press.

Joseph, D. L., & Newman, D. A. (2010). Emotional intelligence: An integrative meta-analysis and cascading model. *Journal of Applied Psychology, 95*, 54–78.

Knobloch, L. K., & Metts, S. (2013). Emotion in relationships. In J. A. Simpson & L. Campbell (Eds.), *The Oxford handbook of close relationships* (pp. 514–534). Oxford, UK: Oxford University Press.

Kring, A. M., & Sloan, D. S. (Eds.). (2009). *Emotion regulation and psychopathology: A transdiagnostic approach to etiology and treatment*. New York: Guilford Press.

Landy, F. (2005). Some historical and scientific issues related to research on emotional intelligence. *Journal of Organizational Behavior, 26*, 411–424.

Larsen, R. J. (2000). Toward a science of mood regulation. *Psychological Inquiry, 11*, 129–141.

Lazarus, R. S. (2000). Toward better research on stress and coping. *American Psychologist, 55*, 665–673.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.

Lopes, P. N., Salovey, P., Côté, S., & Beers, M. (2005). Emotion regulation ability and the quality of social interaction. *Emotion, 5*, 113–118.

Matsumoto, D., Yoo, S. H., Nakagawa, S., Alexandre, J., Altarriba, J., Anguas-Wong, A. M.,

et al. (2008). Culture, emotion regulation, and adjustment. *Journal of Personality and Social Psychology, 94*, 925–937.

Mauss, I. B., Shallcross, A. J., Troy, A. S., John, O. P., Ferrer, E., Wilhelm, F. H., et al. (2011). Don't hide your happiness! Positive emotion dissociation, social connectedness, and psychological functioning. *Journal of Personality and Social Psychology, 100*, 738–748.

Mayer, J. D., & Gaschke, Y. N. (1988). The experience and meta-experience of mood. *Journal of Personality and Social Psychology, 55*, 102–111.

Mayer, J. D., Salovey, P., & Caruso, D. R. (2002). *MSCEIT user's manual*. Toronto: Author.

McRae, K., Jacobs, S. E., Ray, R. D., John, O. P., & Gross, J. J. (2012). Individual differences in reappraisal ability: Links to reappraisal frequency, well-being, and cognitive control. *Journal of Research in Personality, 7*, 253–262.

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotionregulation. *Social Development, 16*, 362–388.

Nolen-Hoeksema, S., Parker, L. E., & Larson, J. (1994). Ruminative coping with depressed mood following loss. *Journal of Personality and Social Psychology, 67*, 92–104.

Parker, J. D. A., Endler, N. S., & Bagby, R. M. (1993). If it changes, it might be unstable: Examining the factor structure of the ways of coping questionnaire. *Psychological Assessment, 5*, 361–368.

Richards, J. M., & Gross, J. J. (2000). Emotion regulation and memory: The cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology, 79*, 410–424.

Saarni, C. (1999). *The development of emotional competence*. New York: Guilford Press.

Saarni, C. (2011). Emotional development in childhood. In R. E. Tremblay, R. G. Barr, R. DeV. Peters, & M. Boivin (Eds.), *Encyclopedia on early childhood development: Emotions* (pp. 1–7). Montréal: Centre of Excellence for Early Childhood Development and Strategic Knowledge Cluster on Early Child Development.

Salovey, P., Mayer, J. D., Golman, S. L., Turvey, C., & Palfai, T. P. (1995). Emotional attention, clarity, and repair: Exploring emotional intelligence using the Trait Meta-Mood Scale. In J. W. Pennebaker (Ed.), *Emotion, disclosure, and health* (pp. 125–154). Washington, DC: American Psychological Association.

Scheier, M. F., & Carver, C. S. (1977). Self-focused attention and the experience of emotion: Attraction, repulsion, elation, and depression. *Journal of Personality and Social Psychology, 35*, 625–636.

Shaver, P. R., & Mikulincer, M. (2007). Adult attachment strategies and the regulation of emotion. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 446–465). New York: Guilford Press.

Shiota, L., Keltner, D., & John, O. P. (2006). Positive emotion dispositions differentially associated with Big Five personality and attachment style. *Journal of Positive Psychology, 1*, 61–71.

Srivastava, S., Tamir, M., McGonigal, K. M., John, O. P., & Gross, J. J. (2009). The social costs of emotional suppression: A prospective study of the transition to college. *Journal of Personality and Social Psychology, 96*, 883–897.

Tamir, M., John, O. P., Srivastava, S., & Gross, J. J. (2007). Implicit theories of emotion: Affective and social outcomes across a major life transition. *Journal of Personality and Social Psychology, 92*, 731–744.

Troy, A. S., Willhelm, F. H., Shallcross, A. J., & Mauss, I. B. (2010). Seeing the silver lining: Cognitive reappraisal ability moderates the relationship between stress and depressive symptoms. *Emotion, 10*, 783–795.

Weinberg, A., & Klonsky, E. (2009). Measurement of emotion dysregulation in adolescents. *Psychological Assessment, 21*, 616–621.

Webb, T. L., Miles, E., & Sheeran, P. (2012). Dealing with feeling: A meta-analysis of the effectiveness of strategies derived from the process model of emotion regulation. *Psychological Bulletin, 138*, 775–808.

Westen, D., & Blagov, P. S. (2007). A clinical–empirical model of emotion regulation: From defense and motivated reasoning to emotional constraint satisfaction. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 373–392). New York: Guilford Press.

CHAPTER 21

Desire and Desire Regulation: Basic Processes and Individual Differences

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Desires pervade everyday life. Accordingly, our language is filled with metaphors of how we experience and deal with desire:

> We burn and are aflame with desire; we are pierced by or riddled with desire; we are sick or ache with desire; we are tortured, tormented, and racked by desire; we are possessed, seized, ravished, and overcome by desire; . . . our desire is fierce, hot, intense, passionate, incandescent, and irresistible; . . . We battle, resist, and struggle with, or succumb, surrender to, and indulge our desires. (Belk, Ger, & Askegaard, 2000, p. 99)

Even though the described struggle with desire is primarily mental, the downstream implications of poor desire regulation can be enormous, such as when political leaders lose their jobs over a scandalous love affair. Additionally, public health statistics show that 40% of deaths in the United States each year are associated with unhealthy behaviors—behaviors that are at least partially attributable to the way people deal with certain appetitive desires, such as those for unhealthy foods, tobacco, alcohol, unprotected sex, and illicit drugs (Schroeder, 2007). From this perspective, gaining a better understanding of how and when desires impact behavior and how unwanted desires may be successfully regulated is clearly needed.

With this chapter, we hope to show that some new insights may be gleaned by treating desires as emotions and, consequently, desire regulation as an instance of emotion regulation. In our reading, the issues of desire and desire regulation have been studied primarily from the vantage point of addiction research, clinical research, and cognitive neuroscience. Applying some of the insights gained from the burgeoning emotion regulation literature to the desire case may broaden our understanding of the problem at hand and open up new avenues of inquiry.

We believe that an emotion regulation perspective on desire may also benefit the field of self-control. That is, a focus on desire and its regulation, may be helpful, wherever the motivational conflicts under study can be framed as a struggle between immediate desires (e.g., for tasty food, alcoholic drinks, sexual interaction) and self-regulatory goals and values (e.g., maintaining one's weight, not driving drunk, staying faithful). Such conflicts arguably make up a great deal of self-control situations in everyday life. Yet until recently, they have been mainly studied under the rubric of goal conflicts (e.g., a short-term goal to eat tasty chocolate vs. a long-term goal to lose weight). While such a terminology is parsimonious and helpful in many respects, it may not get at a deeper

understanding of the specific characteristics of the two opponents involved in such motivational struggles (two opponents, which we argue, are quite different). That is, by framing the short-term goal as "just" another goal, we might miss a closer analysis of the driving force (i.e., of desire) behind that short-term motivation, the specific laws that trigger this driving force, and the most effective strategies to tame it. By treating desire as an essentially emotional phenomenon (involving motivational and cognitive components), we can begin to ask more specific questions about how desire waxes and wanes in close interaction with environmental characteristics and how it can be strategically up- or down-regulated.

Accordingly, self-control researchers have begun to scrutinize the driving forces underlying many self-control struggles (Hofmann, Friese, & Strack, 2009; Hofmann & Van Dillen, 2012) and to look at how factors commonly thought to impact people's control capacity might also affect the strength of desires and cravings (Schmeichel, Harmon-Jones, & Harmon-Jones, 2010; Vohs et al., 2012). By and large, the science of self-control appears to be shifting toward the more balanced view that for many self-control situations, the role of desire is just as important and vital to our understanding as the role of restraint (Hofmann & Van Dillen, 2012).

To advance an emotion regulation perspective on desire, we address the following three major questions in this chapter:

1. What are the main characteristics of desire?
2. How can desire be successfully regulated?
3. How does personality shape desire experiences and desire regulation?

Desire Characteristics

Colloquially, there are many usages of the word *desire*, especially as a verb to express all kinds of wishes and wants (e.g., "I desire that there be more rainbows next year"). Here, we focus exclusively on so-called *appetitive* desires, which are those motivations that propel us to approach and consume objects or otherwise engage in activities that provide a *relative* gain in immediate pleasure (including relief from discomfort). Appetitive desires, in the narrow sense, are typically rooted in primary or acquired physiological need states, such as the desires for food and drink, alcohol, sex, a cigarette, and sleep (no story line intended here); however, we would also include more secondary, socially learned desires (e.g., the urge to check one's smartphone for new messages or the desire to watch a favorite TV show) in a broader definition of appetitive desires. We therefore use the term *desire* to refer to appetitive desires of all sorts. Moreover, we use the term *craving* to refer to desires across domains that are particularly high-intensity (e.g., drug craving, food craving).

Desire as Emotion

In this chapter, we want to make a strong case for treating desires as emotions. Why? Because desires, in our view, share many of the major hallmark characteristics of an emotion (see Franken, 2003). Most important, like emotions, desires are multifaceted phenomena combining affective, motivational, and cognitive components (see Table 21.1).

First, desires have a clearly affective component, consisting of a phenomenological feeling of "wanting" of varying intensity. On the stimulus side, we perceive objects of desire as appealing; desires signal to us that a given thing, person, or activity has high momentary relevance against the backdrop of our current goals, bodily need states, and learning history. Although enacting or thwarting desires may have various emo-

TABLE 21.1. Components of Desire

Component	Description
Affective	A feeling of "wanting" toward certain appealing objects of desire (things, activities, people).
Motivational	A prepotent behavioral driving force to acquire, consume, or be close to the object of desire.
Cognitive	Accompanying thoughts (e.g., expectations, images, and fantasies) regarding the enactment of desire.

tional consequences (more on this later), desires, intrinsically, have a distinct emotional experience to them, which is that phenomenological feeling of wanting.

Second, like other emotions, desires prepare and motivate behavior. Desiring something means wanting to have, consume, or do something that we expect will yield pleasure (or reduce discomfort). When we desire a cupcake, we expect its consumption to provide us with a highly pleasurable experience. When a tobacco addict craves a cigarette, he or she expects that smoking it will alleviate distress (including, somewhat ironically, the discomfort of deprivation created by the addiction). Desires therefore motivate us through their more or less explicit promise of pleasure or relief. Even though hedonic motives may not be the only reasons we pursue desires, we consider the striving for a relative gain in immediate pleasure the primary motivational underpinning of desire. Because desires are directed toward certain objects (i.e., things, persons, activities), they share a sense of "aboutness" (Higgins, 1998) with all other emotions (e.g., being happy, sad, angry, disgusted *about* something). Their target-oriented nature distinguishes them, among other things, from more diffuse affective states such as mood.[1]

Third, desires also have an important cognitive component that is strongly intertwined with the aforementioned affective–motivational components: According to Kavanagh, Andrade, and May's (2005) elaboration–intrusion theory of desire, desire is typically accompanied by intrusive thoughts about the object of desire. Such cognitions comprise expectations about the consequences of desire enactment and the feasibility of attaining the desired object, as well as imagination and fantasies. These cognitions may vary on a continuum, from very realistic to very unrealistic (i.e., overly optimistic), with stronger desires typically leading to more biased and distorted cognitions (Kavanagh et al., 2005). Moreover, the interplay between cognition and affect is recursive and dynamical; as a person mentally elaborates a desire, its strength increases, and more and more mental resources are allocated to the desire, thus lending desire its well-known potential to escalate. As we elaborate below, this state of affairs can reach a point where desire can fully crowd out opposing mental representations such as those of self-control goals (Hofmann, Friese, Schmeichel, & Baddeley, 2011; Hofmann & Van Dillen, 2012; Kavanagh et al., 2005).

Not All Desires Are Temptations

It is important to clarify that *desires* and *temptations* are not synonyms. In our view, temptations are a special subset of desires. To say that somebody is "tempted" by something means that the person has a desire to do *A* on one hand, *and simultaneously* has reason not to do *A* (Mele, 2001). (In this chapter we use the term *temptation* to refer to the ambivalent psychological state of being tempted, whereas we use *tempting stimuli* to refer specifically refer to the external objects that give rise to temptation.) Whether a person has reason not to do *A* depends on whether the behavior implied by the desire conflicts with a person's background set of endorsed values and self-regulatory goals (Hofmann, Baumeister, Förster, & Vohs, 2012). For instance, the desire for mousse au chocolat seems harmless unless one subscribes to a low-fat diet. Similarly, the desire for physical closeness with an attractive acquaintance may be completely unproblematic unless one is subscribed to traditional Western marriage, and—to add some further intricacies—that acquaintance is also one's best friend's spouse. The problem that our appetitive desires are so strongly connected to whatever provides us with immediate pleasure or relief from discomfort on a physiological basis, and that their implications may deviate from what is rationally regarded as optimal, proper, or virtuous, renders occasional self-control conflict an inevitable feature of the human condition.

A recent experience-sampling project called the "Everyday Temptations Study" set out to collect base rate information on the prevalence of desires and temptations in everyday life (Hofmann, Baumeister, et al., 2012; Hofmann, Vohs, & Baumeister, 2012). Participants were equipped with smartphones for a week. On multiple random occasions each day, they received a questionnaire via these smartphones and were asked whether they were currently experiencing a desire from a list of 15 desire domains, including food, nonalcoholic drinks, sleep,

sex, social contact, leisure, sports, spending, media, alcohol, tobacco, and other drugs. Participants reported a current desire about 50% of the time they were signaled. The lion's share of desires were reported to be unproblematic, that is, not in conflict with other important goals (Hofmann, Baumeister, et al., 2012). However, more than one-third of desires were experienced as conflicting with important self-regulatory goals, such as health goals (e.g., healthy eating, bodily fitness), abstinence/restraint goals (e.g., remaining abstinent, saving money, being faithful), achievement-related goals (e.g., educational achievements), social goals (e.g., social recognition, moral integrity), and time use goals (e.g., not delaying things) (Hofmann, Vohs, et al., 2012). Although there were some prominent connections between specific desires and specific opposing goals (e.g., desire for tobacco ↔ reducing health damage; spending ↔ saving expenses), desire–goal conflicts on average were very multifaceted. Thus, one and the same desire can be experienced as a temptation for many different reasons.

Linked Emotions

As noted earlier, the *inherent* affective core of desire (the feeling of "wanting") needs to be distinguished from the emotional *consequences* of desire enactment–nonenactment, which we call *linked emotions*. The enactment of desire is typically linked to a relative short-term gain in pleasure resulting from need satisfaction or relief from discomfort. Conversely, the prolonged nonenactment of desire typically leads to a period of frustration, until the motivational system manages to disengage from the desire altogether (Carver & Scheier, 1998). Note, however, that the primary emotions of pleasure and frustration need not be the only linked emotions. In the case of temptation, things may become more complicated: Because enacting a temptation implies that one may have violated important values relevant for one's identity, giving in to temptation may lead to feelings of guilt or shame (Hofmann & Fisher, 2012; Shiffman et al., 1997), on top of pleasure. Conversely, not enacting a temptation may trigger feelings of pride on top of frustration. Thus, in contrast to unproblematic desires, enacting temptations may at times yield a combination of (positive and negative) primary and secondary emotions (pleasure and guilt; frustration and pride). As we argue elsewhere (Hofmann, Kotabe, & Luhmann, in press), the net affective outcome of such mixed emotional states may be a function of the relative intensities of the linked emotions involved. Enacting a certain temptation (e.g., desire to eat cake when on a diet) may therefore result in a "spoiled pleasure" effect, a reduction in the overall gain in affect as compared with the enactment of the equivalent desire in an unproblematic context (e.g., desire to eat cake when not on a diet).

Although not a focus of the present chapter, the linked emotions of desire enactment and nonenactment can themselves, of course, be subject to emotion regulation. For instance, to increase their affective well-being, people may try to down-regulate the frustration ensuing from unproblematic desires that cannot be enacted because of external obstacles, or to cherish the pleasure ensuing from their enactment for as long as possible. In the case of temptation, a person may try to down-regulate guilt after a lapse has occurred. Likewise, people may try to maintain or up-regulate pride ensuing from successful resistance—a self-conscious emotion that some have argued is often not "harvested" enough for bolstering self-control (Hofmann & Fisher, 2012).

How Does Desire Emerge and Impact Behavior?

Desire emerges in a relatively automatic manner as reward processing centers in midbrain regions (e.g., the ventral striatum) evaluate external stimuli (or mental images thereof[2]) against the backdrop of internal need states and an individual's learning history (Hofmann et al., 2009; Hofmann & Van Dillen, 2012). This early reward processing may have the potential to trigger fast impulsive, habitual responses (e.g., Mogenson, Jones, & Yim, 1980; Winkielman, Berridge, & Wilbarger, 2005). However, most typically, reward signals from midbrain regions are forwarded to prefrontal regions in the brain involved in reward representation and integration, with the orbitofrontal cortex (OFC) being among the regions most consistently implicated (Van der Laan, de

Ridder, Viergevera, & Smeetsa, 2011). Even though somewhat speculative, we believe that the involvement of prefrontal regions such as the OFC is an important element in the conscious representation of desires and cravings.

As desire gains access to consciousness and, therefore, the global workspace of the mind, it gains the potential to "broadcast" its message to a wide range of participating systems, including those that generate thoughts and behavioral intentions (Baars & Franklin, 2003; Baumeister, Masicampo, & Vohs, 2011; Hofmann & Wilson, 2010). Like market criers who try to gather attention of those around them, contents of the global workspace of consciousness compete for mental processing resources. Once a desire becomes conscious, it can function very much like an attractor state (Carver & Scheier, 2002) that exerts increasing influence on conscious processing the more supporting working memory resources it is able to acquire, along the lines of "whoever has will be given more." In other words, desire-related processing can be subject to a vicious circle of reprocessing and rumination as people harbor increasingly elaborated, conscious mental representations of desire that bias attentional, affective, and cognitive processing.

As desire grows in consciousness, so does its potential to instigate concrete action plans and behavioral intentions to consume the object of desire. In the case of temptation, desires may lead to the inhibition of self-regulatory values and goals (Hofmann, Koningsbruggen, Stroebe, Ramanathan, & Aarts, 2010; Stroebe, Mensink, Aarts, Schut, & Kruglanski, 2008). Moreover, as people become increasingly occupied by a certain desire (Kavanagh et al., 2005), they may engage in processes of motivated reasoning that license and justify indulgence (e.g., "I deserve a special treat today because . . ."; "This is going to be my last cigarette, and then I'll quit!"; Kunda, 1990; Sayette & Griffin, 2011). Unless people manage to down-regulate desire effectively and/or allocate attention away from desire-related (hot) cognitions toward competing (cooler) attractor states such as those of self-control goals, desires can hijack the very mechanisms that may otherwise support "reasoned" action. In extreme cases of desire escalation, a desire may have gathered so much clout in working memory that it may be able to fully crowd out all other opposing, goal-related representations—a state we describe as "all-consuming passion" in everyday language.

How Can Desire Be Successfully Regulated?

Given that desires and cravings may sometimes be experienced as conflicting with one's set of self-regulatory goals and values, the question that emerges is how can tempting desires be effectively controlled? In the remainder of this chapter, we therefore focus on the down-regulation of tempting desires. Note, however, that the taming of temptations is only one special case of desire regulation, because desires may be up- or down-regulated for a variety of other reasons. For instance, in order to enjoy a special dinner even more, one may deliberately up-regulate one's appetitive desire for food by refraining from the regular afternoon snack. Or in order to stay awake on a train ride from Tokyo to Osaka to view Mt. Fuji from the window, one may deliberately down-regulate one's desire to sleep on the train by getting a good night's rest the night before.

As laid out by Gross (this volume), emotion regulation refers to shaping which emotions one has, when one has them, and how one experiences them. Applied to tempting desires, we focus on regulatory processes and strategies affecting (1) whether a given tempting desire is consciously experienced to begin with, and, if so, (2) how such a tempting desire can be effectively down-regulated. The underlying assumption is that the likelihood of a given tempting desire to impact behavior, all else being equal, is a function of the desire's intensity (more intense desire is more capable of hijacking working memory resources in its favor). Therefore, all mechanisms that help keep the intensity of tempting desire below a critical enactment level, either by preventing tempting desire from emerging in consciousness in the first place, or by helping the person down-regulate tempting desire, can be considered effective desire regulation strategies. Conversely, mechanisms that increase desire intensity, contrary to the desire down-regulation goal, can be considered ineffective.

In close accordance with the process model of emotion regulation (Gross, 1998), we review emotion regulation mechanisms and strategies pertaining to situation and stimulus control, attention allocation, cognitive reappraisal, and suppression. The emotion regulation strategies reviewed vary in the extent to which they may prevent the emergence of conscious desire (i.e., by preventing exposure to triggering cues or interfering with the conscious processing of tempting stimuli) or aid the down-regulation of conscious desire experiences.

Situation and Stimulus Control

Availability begets desire (Carter & Tiffany, 2001). Without any doubt, the most effective strategy to prevent desire is to avoid exposure to tempting situations or stimuli altogether through techniques of situation and stimulus control (Mahoney & Thoresen, 1972). Based on the notion that external stimuli, in their interaction with a person's learning history and current need states, play a seminal role in the generation of desire as "impellers" (Finkel & Eckhardt, in press; Hofmann & Van Dillen, 2012), these techniques can greatly alter the odds that people will experience temptation. Situation and stimulus control techniques can either be learned and applied by a person directly (e.g., keeping one's home free of unhealthy but tempting foods) or be applied through paternalistic "nudges" (Thaler & Sunstein, 2010) that reduce the availability of temptations in people's environments (e.g., "no smoking" bans in restaurants and bars; cafeterias that primarily offer healthy choice options). Because situation and stimulus control are not always feasible (e.g., when one is "stuck" in a temptation-rich environment), however, situation and stimulus control cannot be the sole answer to effective desire regulation.

Attention Allocation

Our previous analysis of how desire emerges suggests a fundamental role of attention in desire regulation. Attention can be parsed into both automatic, bottom-up attention (i.e., attention driven by salient stimulus properties) and controlled, top-down attention (i.e., the goal-driven allocation of attention toward goal-relevant information in working memory) (Knudsen, 2007). Both types of attention shape desire experiences; therefore, both mechanisms may be harnessed to regulate desire effectively, though we argue that top-down attention is ultimately more important.

Regarding bottom-up attention, a growing number of studies in the addiction domain have tried to reduce conscious cravings and associated behaviors by modifying automatic attentional biases toward tempting stimuli (see also MacLeod & Grafton, this volume). However, the results of these attentional retraining studies have been mixed. Sometimes the effects generalized to other contexts, but at other times the effects did not extend much beyond the retraining task (Attwood, O'Sullivan, Leonards, Mackintosh, & Munafo, 2008; Fadardi & Cox, 2009; Field, Duka, Tyler, & Schoenmakers, 2009; Schoenmakers, Wiers, Jones, Bruce, & Jansen, 2007). The most encouraging results were obtained when several attentional retraining sessions were used and participants were explicitly told about the purpose and theoretical rationale of the retraining task.

Increasing evidence suggests, however, that top-down attention may be more decisive in desire regulation. Top-down attention has been shown to modulate bottom-up attention (Rauss, Schwartz, & Pourtois, 2011; Van Dillen & Koole, 2009). Top-down attention enables the selective processing of information relevant for one's current goal, preventing distracting information from entering awareness (Knudsen, 2007). Hence, when a current goal directs attention in such a way that tempting information becomes irrelevant, the attentional capture of tempting stimuli should be substantially reduced or not occur at all. Conversely, people should process tempting cues more strongly in the absence of a specific goal (i.e., when their top-down attention is free to be captured by salient cues), or when their current goal focus prompts attentional processing of such cues. Indeed, recent research has shown that cognitively demanding tasks (unrelated to the temptation at hand), can actually prevent the emergence of desire in response to tempting stimuli (Kemps, Tiggemann, & Christianson, 2008; Van Dillen, Papies, & Hofmann, 2013). For instance, in the Van Dillen et al.

(Study 1), participants categorized pictures of tempting (e.g., brownies) versus neutral (e.g., radishes) food stimuli according to their spatial location on the screen while rehearsing either a one-digit number (low load) or an eight-digit number (high load) during each task trial. Participants under low cognitive load allocated more attention to tempting than to neutral stimuli, as evidenced by slower spatial categorizations of attractive food pictures compared to neutral food pictures. Participants under high cognitive load were equally fast to respond to tasty and neutral food items, suggesting that they may not have processed the hedonic relevance of the attractive food (as much). Consequently, participants in the high load condition reported lower snack cravings following the categorization task. Hence, the desire to consume attractive food temptations may only surface in consciousness to the extent that people have sufficient attentional resources to assess their hedonic value.[3] Early powerful distraction may therefore facilitate desire regulation by preventing the emergence of desire and—by implication—the conscious pursuit of desire satisfaction.

The powerful role of top-down attention in desire regulation suggests that teaching people how to distract themselves from cravings through mentally engaging activities may be an effective element in craving management and relapse prevention (Florsheim, Heavin, Tiffany, Colvin, & Hiraoka, 2008). The power of attention is also exemplified in recent applied interventions showing how mindfulness- and acceptance-based interventions can reduce cravings in problem populations (Alberts, Mulkens, Smeets, & Thewissen, 2010; Forman et al., 2007). The concept of mindfulness (see also Farb, Anderson, Irving, Segal, this volume) has been tightly linked with effective top-down attention (Chambers, Lo, & Allen, 2008). One mechanism through which mindfulness interventions may work is by increasing people's ability to maintain their self-regulatory goals in working memory and shield them from interference through desire-related processing (Hofmann, Gschwendner, Friese, Wiers, & Schmitt, 2008; Hofmann, Schmeichel, & Baddeley, 2012). In addition, the down-regulation of ongoing desires and cravings may be facilitated by the acceptance component contained in these interventions: Accepting desires and cravings as transient (i.e., fleeting) states instead of trying to suppress them may make it easier for people to disengage from the maladaptive vicious circle of reprocessing and rumination.

Reappraisal

Rather than avoid tempting situations and stimuli or allocate attention in strategic ways, people can also employ strategies that modify how they appraise tempting stimuli. For instance, in *On Desire: Why We Want What We Want*, William Irvine (2006) relates advice provided by a Buddhist monk on how to deal with tantalizing sexual desire by thinking about the human body in less favorable terms: "Don't think about her full breasts and flaxen hair; think instead about her lungs, . . . phlegm, pus, spittle." According to the monk, the aim of the mediation "is not to produce aversion and disgust but detachment, to extinguish the fire of lust by removing its fuel" (p. 187).

Early pioneering research on delay of gratification by Walter Mischel has shown that cognitive reappraisal of tempting stimuli can have a large impact on how well schoolchildren are able to resist immediate rewards, such as marshmallows, by cognitively transforming the rewards in nonconsummatory ways (e.g., imagining the marshmallows as white puffy clouds) (Mischel & Baker, 1975). Recent work applying this idea has demonstrated that reappraisal can have a profound impact on affective responses to tempting stimuli (Fujita & Han, 2009; Hofmann, Deutsch, Lancaster, & Banaji, 2010). Engaging people in an abstract rather than concrete mindset (Fujita & Han, 2009) or imagining tempting stimuli in nonconsummatory ways (Hofmann, Deutsch, et al., 2010) appear to interfere with the early reward processing of these stimuli, thus taking the edge of temptation.

Findings that cognitive reappraisal can have far-reaching effects on how people initially appraise tempting stimuli align well with recent accounts arguing that emotion regulation can often operate via powerful automatic or implicit processes (see Gyurak & Etkin, this volume). For instance, several lines of work have suggested that, at least

under certain circumstances, mental representations of self-control goals may exert an automatic inhibiting effect on mental representations of desire, implying that tempting stimuli may lose their desire-activating potential. For instance, Fishbach, Friedman, and Kruglanski (2003) argued that tempting stimuli can trigger the automatic activation of an overriding self-control goal in successful self-regulators (e.g., the sight of a cake activating a dieting goal).[4] Thus, strong and easily accessible self-control goals may help to reduce desire-related processing because of the automatic inhibition of goal-irrelevant information (Shah, Friedman, & Kruglanski, 2002), which typically implies a relative devaluation of the tempting stimulus (Veling, Holland, & van Knippenberg, 2007, 2008). Automatic self-regulatory processes may thus contribute to desire down-regulation and—depending on their potency—may even make the need for more effortful self-control obsolete.

The discovery of automatic self-regulatory processes has raised the intriguing question of whether the initial appraisal of tempting stimuli (i.e., the reward signal triggered upon exposure) may be altered through training and intervention techniques. The underlying rationale is that people should have an easier time keeping desires at bay if the tempting stimuli that typically elicit strong desires and cravings are appraised in less positive terms. One method to shape the initial appraisal of tempting stimuli is evaluative conditioning (Hofmann, De Houwer, Perugini, Baeyens, & Crombez, 2010). Specifically, pairing a tempting stimulus (e.g., alcohol) repeatedly with an affectively negative unconditioned stimulus can decrease desire for alcohol among problematic drinkers (Houben, Havermans, & Wiers, 2010; Van Gucht, Baeyens, Vansteenwegen, Hermans, & Beckers, 2010). A second promising method is avoidance training, in which people are trained to respond routinely to tempting stimuli with avoidance responses (Wiers, Eberl, Rinck, Becker, & Lindenmeyer, 2011; Wiers, Rinck, Kordts, Houben, & Strack, 2010). In one intriguing study, alcoholic inpatients who underwent cognitive-behavioral therapy (CBT) and four 15-minute training sessions to avoid alcohol stimuli showed reduced cravings after the avoidance treatment, as well as reduced relapse 1 year after treatment when compared with control patients who only did CBT (Wiers et al., 2011). Relatedly, laboratory work has shown that implementation intentions to avoid tempting stimuli can reduce automatic affective reponses to these stimuli, presumably because such implementation intentions reprogram the mind to appraise the tempting stimulus more negatively (Hofmann, Deutsch, et al., 2010).

Suppression

If accepting a desire may facilitate desire regulation as noted earlier, how does the opposite strategy fare? Is the willful suppression or negation of cravings an effective desire regulation strategy? The bulk of the literature on appetitive thought suppression across domains suggests not (Barnes & Tantleff-Dunn, 2010; Erskine, 2008; Johnston, Bulik, & Anstiss, 1999; Mann & Ward, 2001). The problem with the forced suppression of desire is that even though suppression may provide some short-term relief, suppression may often backfire, leading to ironic rebound effects (Wegner, 1994). According to Wegner's theory, when one tries actively to suppress something, attention may be repeatedly redirected toward the very mental content one is trying to avoid. This feature of suppression may contribute to keeping the desire–processing loop alive, or even lead to the hyperaccessibility of desire-related thoughts. As we argue below, these (main) effects do not imply that everybody suffers from ironic rebound effects to the same extent. Suppression may well be effective for those individuals who are particularly skilled at directing their attention in a top-down manner (Brewin & Smart, 2005).

Individual Differences in Desire and Its Regulation

Our main focus, up to this point, has been on general mechanisms that facilitate or hinder successful desire regulation. In this last part of this chapter, we address the research on how individual differences may shape the way people experience and deal with appetitive desires.

Regarding the intensity of desire experiences, there is strong evidence that individuals high in behavioral approach system (BAS) sensitivity experience stronger desires and cravings than do those low in BAS. This effect was found both in cue-reactivity paradigms in the laboratory (Franken, 2002) and when aggregating average self-reported desire strength across many desire domains (Hofmann, Baumeister, et al., 2012). These findings clearly support Gray's (1982) reinforcement sensitivity theory, arguing that the BAS is responsible for the reward processing in appetitive motivation, and suggest an important role for related constructs such as sensation seeking.

The Everyday Temptations Study also showed that individuals high in trait self-control (TSC) reported lower average desire strength (as well as lower average conflict and less use of active resistance to control desire) (Hofmann, Baumeister, et al., 2012). Even though somewhat speculative, we believe this surprising pattern of findings may reflect the joint impact of two mechanisms. First, individuals with high TSC may make more use of preventive situation and stimulus control strategies, thus avoiding tempting desires more often than their low-TSC counterparts (and hence reducing the need for effortful control). In support, independent raters rated the desires reported by high-TSC participants as less problematic for the "average person" than the desires reported by low-TSC participants (Hofmann, Baumeister, et al., 2012). In other words, high-TSC individuals seem to be navigating an objectively less tempting desire landscape. Second, it is conceivable that those with high TSC have more powerful automatic desire regulation processes at their disposal. Some support for this assumption comes from studies revealing that people who report to be successful self-regulators in a given domain, such as dieting, tend to show more effective automatic self-regulation than do unsuccessful self-regulators (Papies, Stroebe, & Aarts, 2008a). These findings are intriguing because they suggest that, in everyday life, TSC may not so much be about how well temptations are resisted (the type of "late-stage" self-control typically studied in the laboratory situations) as about how well temptations are being avoided or appraised at early stages in the self-control process. Furthermore, we believe that TSC is distinct from individual differences in what has been called the *restraint bias* (Nordgren, van Harreveld, & van der Pligt, 2009), which taps into people's overoptimistic—and thus unrealistic—beliefs about their capacity at self-control. As a consequence of their inflated beliefs, people high in the restraint bias tend to overexpose themselves to tempting situations, leading to more frequent self-control failures in the heat of the moment (Nordgren et al., 2009). Thus, we predict that individuals high in TSC harbor more realistic impulse control beliefs than do individuals low in TSC. This more realistic willpower self-assessment (and a more realistic appreciation of the power of the situation) may lead them to prudently avoid high-risk situations and remove tempting stimuli from their environment when they can.

Individuals also differ considerably in their command of top-down attention. In the cognitive literature on executive functioning, top-down attention has been strongly linked with the concept of working memory capacity (WMC; Kane, Bleckley, Conway, & Engle, 2001), which represents the ability to maintain and update relevant information in working memory, and to shield it from interfering processing or distraction (Engle, 2002; Kane et al., 2001). This ability may be highly instrumental to a variety of strategies important for effective desire regulation (Hofmann et al., 2011). First, WMC may help people disengage faster and be less influenced by attention-grabbing desire-related cues. Eye-tracking studies, for instance, have shown that people low in WMC are prone to bias eye direction toward tempting cues, with the degree of bias correlating with their automatic affective reactions toward those cues (Friese, Bargas-Avila, Hofmann, & Wiers, 2010; Friese & Hofmann, 2012). Those high in WMC, in contrast, were much less influenced by their automatic affective reactions, which suggests that they were faster at disengaging attention from salient cues.

Second, WMC may aid a host of emotion regulation strategies, such as the efficient use of distraction, reappraisal, and emotion suppression (Hofmann et al., 2008, Study 3; Hofmann, Schmeichel, et al., 2012; McRae, Jacobs, Ray, John, & Gross, 2012;

Schmeichel, Volokhov, & Demaree, 2008). Third, individuals high in WMC seem to be better at controlling their own thoughts, with fewer thought intrusions (Brewin & Smart, 2005; Kane et al., 2007). Even though more direct evidence for the moderating role of WMC in the regulation of desires and cravings is missing, it does not appear to be great leap to suggest that individuals with high WMC are more effective than those with low WMC at breaking the desire-processing cycle.

Because executive functions such as working memory can be trained, at least to some extent (for a discussion, see Shipstead, Redick, & Engle, 2012), there is a large potential for intervention research aimed at finding ways to improve the management of unwanted desires and cravings. Furthermore, fascinating new research suggests that repeated high-frequency (i.e., excitatory) stimulation of the dorsolateral prefrontal cortex via transcranial magnetic stimulation (TMS) or transcranial direct current stimulation (tDCS) may reduce cravings (e.g., Amiaz, Levy, Vainiger, Grunhaus, & Zangen, 2009; Boggio et al., 2008; Camprodon, Martinez-Raga, Alonso-Alonso, Shih, & Pascual-Leone, 2007). One highly plausible explanation is that the high-frequency stimulation leads to an additional mobilization of executive control processes implied in the down-regulation of cravings.

Regarding further personality traits, the Everyday Temptations Study showed that a measure of perfectionism (tapping primarily into negative, dysfunctional perfectionism) was associated with stronger desire intensity in daily life (Hofmann, Baumeister, et al., 2012), more intense feelings of conflict, and more frequent desire resistance. Even though somewhat speculative, this finding suggests that people high in this sort of perfectionism may become overly preoccupied with regulating their desires, perhaps making too much use of counterproductive strategies such as suppression, consistent with research on the trait's connection with emotion suppression (Bergman, Nyland, & Burns, 2007).

Finally, when discussing the positive effects of mindfulness on desire regulation, we highlighted the possible mediating role of desire acceptance as an effective regulation strategy. The case of acceptance raises the intriguing issue of whether there are reliable individual differences in the way people relate to their desires. A number of personality characteristics, such as faith in intuition, trait hedonism, tolerance for ambiguity, as well as demographic variables, such as religiosity, may moderate the extent to which people see their appetitive desires as integral parts of themselves (leading to high acceptance) as opposed to "dark and sinister" forces that lack integration with the self and thus need to be kept at bay. In the absence of empirical work that has explored such possible links between acceptance of appetitive desires and personality traits, we predict that such personality characteristics should (1) relate to how often people go about regulating their appetitive desires and (2) facilitate regulation of appetitive desires to the extent that they promote acceptance-based rather than suppression-based emotion regulation strategies.

Conclusion

As humans we experience all sorts of appetitive desires. Sometimes, these stand in strong opposition to our self-regulatory goals or values, forcing us to face the challenge of how best to "get a grip" on such "unwanted wants." In this chapter, we have argued that desires can be conceptually treated as emotions, because, like emotions, desires have affective, motivational, and cognitive components. Hence, it may be beneficial to address the issue of desire regulation from an emotion regulatory perspective.

Our review of the available literature suggests that, as with emotion regulation, effective desire regulation can take place at various phases of desire processing. Desire regulation strategies can be roughly divided into those that prevent exposure to desire-eliciting stimuli (situation and stimulus control), those that may prevent a desire from reaching conscious awareness (e.g., attention allocation), and those that prevent a desire from becoming overly dominant (e.g., reappraisal). Mapping a given strategy to just one of these different phases may be overly simplistic, however. For example, take attention allocation: Sometimes, powerful distractors (resulting in mental preoccupation) may prevent a desire from reach-

ing conscious awareness; at other times, attention allocation may be used strategically to down-regulate a desire only after it has reached conscious awareness. In a similar vein, it is possible that automatic down-regulation may sometimes be so powerful that it may even prevent the onset of a real desire, whereas at other times, it may make a desire less potent, so that it does not take up as many mental resources as it otherwise would. Moreover, it is possible that multiple strategies may complement each other in important ways that are worthy of future study. For instance, powerful automatic regulation should reduce the demand for more effortful emotion regulation processes.

Finally, people appear to differ as to how intensely they experience desire and how they go about regulating it. Individuals high in TSC seem to make more use of early preventive strategies such as situation and stimulus control. (Dysfunctional) perfectionists may focus too much on suppression, thus becoming overly obsessed with their desires. Being dispositionally high in executive functions such as WMC may make it a lot easier to resist the attentional pull of tempting stimuli. Taking a closer look at what strategies work for what types of people should help practitioners to devise more effective customized treatments for those who suffer from tempting desires and cravings. As technological possibilities mature, we expect to see fascinating new developments in the years to come with regard to one of the central challenges of what it means to be human: how to shape one's internal landscape of appetitive desires in a way that strikes a healthy balance between desire enactment and the occasional need to curb one's passion for what might be a pretty good reason.

Notes

1. Even though the two are often correlated, desires are also conceptually distinct from attitudes, because liking does not necessarily imply wanting and vice versa (Berridge, 1996). For instance, one may like lobster in general but have no desire to eat one right now. A long-term smoker may dislike cigarettes ("They're unhealthy and expensive") yet have a craving for one.
2. Whereas external stimuli are typically involved and sometimes strategically employed by those wanting to instigate certain desires in others (e.g., companies using scents to better sell their products; a person wearing sexy clothing on a date), it is also possible that desires can be activated in a top-down fashion in the absence of any external stimuli by willfully activating certain memories or simulating certain experiences (Kavanagh et al., 2005).
3. Note that the effects of cognitive load are quite the opposite of the effects of resource depletion. Recent work by Vohs et al. (2012) shows that people in a depleted state experience stronger emotional reactions to valenced stimuli, intensified pain in a cold pressor task, and an intensified desire to eat cookies presented during a taste and rate test.
4. The reverse process (i.e., inhibition of the self-control goal) may take place among less successful self-regulators (see Papies, Stroebe, & Aarts, 2008b; Stroebe, Mensink, Aarts, Schut, & Kruglanski, 2008), suggesting important individual differences with regard to automatic desire-regulation processes.

References

Alberts, H. J. E. M., Mulkens, S., Smeets, M., & Thewissen, R. (2010). Coping with food cravings. Investigating the potential of a mindfulness-based intervention. *Appetite, 55*, 160–163.

Amiaz, R., Levy, D., Vainiger, D., Grunhaus, L., & Zangen, A. (2009). Repeated high-frequency transcranial magnetic stimulation over the dorsolateral prefrontal cortex reduces cigarette craving and consumption. *Addiction, 104*, 653–660.

Attwood, A. S., O'Sullivan, H., Leonards, U., Mackintosh, B., & Munafo, M. R. (2008). Attentional bias training and cue reactivity in cigarette smokers. *Addiction, 103*, 1875–1882.

Baars, B. J., & Franklin, S. (2003). How conscious experience and working memory interact. *Trends in Cognitive Sciences, 7*, 166–172.

Barnes, R. D., & Tantleff-Dunn, S. (2010). Food for thought: Examining the relationship between food thought suppression and weight-related outcomes. *Eating Behaviors, 11*, 175–179.

Baumeister, R. F., Masicampo, E. J., & Vohs, K. D. (2011). Do conscious thoughts cause

behavior? *Annual Review of Psychology, 62*, 331–361.

Belk, R. W., Ger, G., & Askegaard, S. (2000). The missing streetcar named desire. In S. Ratneshwar, D. G. Mick, & C. Huffman (Eds.), *The why of consumption: Contemporary perspectives on consumer motives, goals and desires* (pp. 98–119). London: Routledge.

Bergman, A. J., Nyland, J. E., & Burns, L. R. (2007). Correlates with perfectionism and the utility of a dual process model. *Personality and Individual Differences, 43*, 389–399.

Berking, M., & Schwarz, J. (this volume, 2014). Affect regulation training. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 529–547). New York: Guilford Press.

Berridge, K. C. (1996). Food reward: Brain substrates of wanting and liking. *Neuroscience and Biobehavioral Reviews, 20*, 1–25.

Boggio, P. S., Sultani, N., Fecteau, S., Merabet, L., Mecca, T., Pascual-Leone, A., et al. (2008). Prefrontal cortex modulation using transcranial DC stimulation reduces alcohol craving: A double-blind, sham-controlled study. *Drug and Alcohol Dependence, 92*, 55–60.

Brewin, C. R., & Smart, L. (2005). Working memory capacity and suppression of intrusive thoughts. *Journal of Behavior Therapy and Experimental Psychiatry, 36*, 61–68.

Camprodon, J. A., Martinez-Raga, J., Alonso-Alonso, M., Shih, M. C., & Pascual-Leone, A. (2007). One session of high frequency repetitive transcranial magnetic stimulation (rTMS) to the right prefrontal cortex transiently reduces cocaine craving. *Drug and Alcohol Dependence, 86*, 91–94.

Carter, B. L., & Tiffany, S. T. (2001). The cue-availability paradigm: The effects of cigarette availability on cue reactivity in smokers. *Experimental and Clinical Psychopharmacology, 9*, 183–190.

Carver, C. S., & Scheier, M. F. (1998). *On the self-regulation of behavior.* Cambridge, UK: Cambridge University Press.

Carver, C. S., & Scheier, M. F. (2002). Control processes and self-organization as complementary principles underlying behavior. *Personality and Social Psychology Review, 6*, 304–315.

Chambers, R., Lo, B. C. Y., & Allen, N. B. (2008). The impact of intensive mindfulness training on attentional control, cognitive style, and affect. *Cognitive Therapy and Research, 32*, 303–322.

Engle, R. W. (2002). Working memory capacity as executive attention. *Current Directions in Psychological Science, 11*, 19–23.

Erskine, J. A. K. (2008). Resistance can be futile: Investigating behavioural rebound. *Appetite, 50*, 415–421.

Fadardi, J. S., & Cox, W. M. (2009). Reversing the sequence: Reducing alcohol consumption by overcoming alcohol attentional bias. *Drug and Alcohol Dependence, 101*, 137–145.

Farb, N. A. S., Anderson, A. K., Irving, J. A., & Segal, Z. V. (this volume, 2014). Mindfulness interventions and emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 548–568). New York: Guilford Press.

Field, M., Duka, T., Tyler, E., & Schoenmakers, T. (2009). Attentional bias modification in tobacco smokers. *Nicotine and Tobacco Research, 11*, 812–822.

Finkel, E. J., & Eckhardt, C. I. (in press). Intimate partner violence. In J. A. Simpson & L. Campbell (Eds.), *The Oxford handbook of close relationships.* New York: Oxford University Press.

Fishbach, A., Friedman, R. S., & Kruglanski, A. W. (2003). Leading us not unto temptation: Momentary allurements elicit overriding goal activation. *Journal of Personality and Social Psychology, 84*, 296–309.

Florsheim, P., Heavin, S., Tiffany, S., Colvin, P., & Hiraoka, R. (2008). An experimental test of a craving management technique for adolescents in substance-abuse treatment. *Journal of Youth and Adolescence, 37*, 1205–1215.

Forman, E. M., Hoffman, K. L., McGrath, K. B., Herbert, J. D., Brandsma, L. L., & Lowe, M. R. (2007). A comparison of acceptance- and control-based strategies for coping with food cravings: An analog study. *Behaviour Research and Therapy, 45*, 2372–2386.

Franken, I. H. (2003). Drug craving and addiction: Integrating psychological and neuropsychopharmacological approaches. *Progress in Neuropsychopharmacological Biological Psychiatry, 27*, 563–579.

Franken, I. H. A. (2002). Behavioral approach system (BAS) sensitivity predicts alcohol craving. *Personality and Individual Differences, 32*, 349–355.

Friese, M., Bargas-Avila, J., Hofmann, W., & Wiers, R. W. (2010). Here's looking at you, Bud: Alcohol-related memory structures predict eye movements for social drinkers with low executive control. *Social Psychological and Personality Science, 1*, 143–151.

Friese, M., & Hofmann, W. (2012). Just a little bit longer: Viewing time of erotic material from a self-control perspective. *Applied Cognitive Psychology, 26*, 489–496.

Fujita, K., & Han, H. A. (2009). Moving beyond deliberative control of impulses: The effect of construal levels on evaluative associations in self-control conflicts. *Psychological Science, 20*, 799–804.

Gray, J. A. (Ed.). (1982). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system*. New York: Oxford University Press.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*, 271–299.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gyurak, A., & Etkin, A. (this volume, 2014). A neurobiological model of implicit and explicit emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 76–90). New York: Guilford Press.

Higgins, E. T. (1998). The aboutness principle: A pervasive influence on human inference. *Social Cognition, 16*, 173–198.

Hofmann, W., Baumeister, R. F., Förster, G., & Vohs, K. D. (2012). Everyday temptations: An experience sampling study of desire, conflict, and self-control. *Journal of Personality and Social Psychology, 102*, 1318–1335.

Hofmann, W., De Houwer, J., Perugini, M., Baeyens, F., & Crombez, G. (2010). Evaluative conditioning in humans: A meta-analysis. *Psychological Bulletin, 136*, 390–421.

Hofmann, W., Deutsch, R., Lancaster, K., & Banaji, M. R. (2010). Cooling the heat of temptation: Mental self-control and the automatic evaluation of tempting stimuli. *European Journal of Social Psychology, 40*, 17–25.

Hofmann, W., & Fisher, R. R. (2012). How guilt and pride shape subsequent self-control. *Social Psychological and Personality Science, 3*, 682–690.

Hofmann, W., Friese, M., Schmeichel, B. J., & Baddeley, A. D. (2011). Working memory and self-regulation. In K. D. Vohs & R. F. Baumeister (Eds.), *The handbook of self-regulation: Research, theory, and applications* (Vol. 2, pp. 204–226). New York: Guilford Press.

Hofmann, W., Friese, M., & Strack, F. (2009). Impulse and self-control from a dual-systems perspective. *Perspectives on Psychological Science, 4*, 162–176.

Hofmann, W., Gschwendner, T., Friese, M., Wiers, R. W., & Schmitt, M. (2008). Working memory capacity and self-regulatory behavior: Toward an individual differences perspective on behavior determination by automatic versus controlled processes. *Journal of Personality and Social Psychology, 95*, 962–977.

Hofmann, W., Koningsbruggen, G. M., Stroebe, W., Ramanathan, S., & Aarts, H. (2010). As pleasure unfolds: Hedonic responses to tempting food. *Psychological Science, 21*, 1863–1870.

Hofmann, W., Kotabe, H., & Luhmann, M. (in press). The spoiled pleasure of giving in to temptation. *Motivation and Emotion*.

Hofmann, W., Schmeichel, B. J., & Baddeley, A. D. (2012). Executive functions and self-regulation. *Trends in Cognitive Sciences, 3*, 174–180.

Hofmann, W., & Van Dillen, L. F. (2012). Desire: The new hotspot in self-control research. *Current Directions in Psychological Science, 21*, 317–322.

Hofmann, W., Vohs, K. D., & Baumeister, R. F. (2012). What people desire, feel conflicted about, and try to resist in everyday life. *Psychological Science, 23*, 582–588.

Hofmann, W., & Wilson, T. D. (2010). Consciousness, introspection, and the adaptive unconscious. In B. Gawronski & B. K. Payne (Eds.), *Handbook of implicit social cognition: Measurement, theory, and applications* (pp. 197–215). New York: Guilford Press.

Houben, K., Havermans, R. C., & Wiers, R. W. (2010). Learning to dislike alcohol: Conditioning negative implicit attitudes toward alcohol and its effect on drinking behavior. *Psychopharmacology, 211*, 79–86.

Irvine, W. B. (2006). *On desire. Why we want what we want*. New York: Oxford University Press.

Johnston, L., Bulik, C. M., & Anstiss, V. (1999). Suppressing thoughts about chocolate. *International Journal of Eating Disorders, 26*, 21–27.

Kane, M. J., Bleckley, M. K., Conway, A. R. A., & Engle, R. W. (2001). A controlled-attention view of working-memory capacity. *Journal of Experimental Psychology: General, 130*, 169–183.

Kane, M. J., Brown, L. H., McVay, J. C., Silvia, P. J., Myin-Germeys, I., & Kwapil, T. R. (2007). For whom the mind wanders, and

when: An experience-sampling study of working memory and executive control in daily life. *Psychological Science, 18*, 614–621.

Kavanagh, D. J., Andrade, J., & May, J. (2005). Imaginary relish and exquisite torture: The elaborated intrusion theory of desire. *Psychological Review, 112*, 446–467.

Kemps, E., Tiggemann, M., & Christianson, R. (2008). Concurrent visuo-spatial processing reduces food cravings in prescribed weight-loss dieters. *Journal of Behavior Therapy and Experimental Psychiatry, 39*, 177–186.

Knudsen, E. I. (2007). Fundamental components of attention. *Annual Review of Neuroscience, 30*, 57–78.

Kunda, Z. (1990). The case for motivated reasoning. *Psychological Bulletin, 108*, 480–498.

MacLeod, C., & Grafton, B. (this volume, 2014). Regulation of emotion through modification of attention. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 508–528). New York: Guilford Press.

Mahoney, M. J., & Thoresen, C. E. (1972). Behavioral self-control: Power to the person. *Educational Researcher, 1*, 5–7.

Mann, T., & Ward, A. (2001). Forbidden fruit: Does thinking about a prohibited food lead to its consumption? *International Journal of Eating Disorders, 29*, 319–327.

McRae, K., Jacobs, S. E., Ray, R. D., John, O. P., & Gross, J. J. (2012). Individual differences in reappraisal ability: Links to reappraisal frequency, well-being, and cognitive control. *Journal of Research in Personality, 46*, 2–7.

Mele, A. (2001). *Autonomous agents. From self-control to autonomy.* Oxford, UK: Oxford University Press.

Mischel, W., & Baker, N. (1975). Cognitive appraisals and transformations in delay behavior. *Journal of Personality and Social Psychology, 31*, 254–261.

Mogenson, G. J., Jones, D. L., & Yim, C. Y. (1980). From motivation to action: Functional interface between the limbic system and the motor system. *Progress in Neurobiology, 14*, 69–97.

Nordgren, L. F., van Harreveld, F., & van der Pligt, J. (2009). The restraint bias: How the illusion of self-restraint promotes impulsive behavior. *Psychological Science, 20*, 1523–1528.

Papies, E. K., Stroebe, W., & Aarts, H. (2008a). The allure of forbidden food: On the role of attention in self-regulation. *Journal of Experimental Social Psychology, 44*, 1283–1292.

Papies, E. K., Stroebe, W., & Aarts, H. (2008b). Healthy cognition: Processes of self-regulatory success in restrained eating. *Personality and Social Psychology Bulletin, 34*, 1290–1300.

Rauss, K., Schwartz, S., & Pourtois, G. (2011). Top-down effects on early visual processing in humans: A predictive coding framework. *Neuroscience and Biobehavioral Reviews, 35*, 1237–1253.

Sayette, M. A., & Griffin, K. M. (2011). Self-regulatory failure and addiction. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications* (Vol. 2, pp. 505–521). New York: Guilford Press.

Schmeichel, B. J., Harmon-Jones, C., & Harmon-Jones, E. (2010). Exercising self-control increases approach motivation. *Journal of Personality and Social Psychology, 99*, 162–173.

Schmeichel, B. J., Volokhov, R. N., & Demaree, H. A. (2008). Working memory capacity and the self-regulation of emotional expression and experience. *Journal of Personality and Social Psychology, 95*, 1526–1540.

Schoenmakers, T., Wiers, R. W., Jones, B. T., Bruce, G., & Jansen, A. T. M. (2007). Attentional retraining decreases attentional bias in heavy drinkers without generalization. *Addiction, 102*, 399–405.

Schroeder, S. A. (2007). We can do better—Improving the health of the american people. *New England Journal of Medicine, 357*, 1221–1228.

Shah, J. Y., Friedman, R. S., & Kruglanski, A. W. (2002). Forgetting all else: On the antecedents and consequences of goal shielding. *Journal of Personality and Social Psychology, 83*, 1261–1280.

Shiffman, S., Hickcox, M., Paty, J. A., Gnys, M., Kassel, J. D., & Richards, T. J. (1997). The abstinence violation effect following smoking lapses and temptations. *Cognitive Therapy and Research, 21*(5), 497–523.

Shipstead, Z., Redick, T. S., & Engle, R. W. (2012). Is working memory training effective? *Psychological Bulletin, 138*, 628–654.

Stroebe, W., Mensink, W., Aarts, H., Schut, H., & Kruglanski, A. (2008). Why dieters fail: Testing the goal conflict model of eating. *Journal of Experimental Social Psychology, 44*, 26–36.

Thaler, R. H., & Sunstein, C. R. (2010). *Nudge: Improving decisions about health, wealth, and happiness.* New York: Penguin Books.

Van der Laan, L. N., de Ridder, D. T. D., Viergevera, M. A., & Smeetsa, P. A. M. (2011). The first taste is always with the eyes: A meta-analysis on the neural correlates of processing visual food cues. *NeuroImage, 55*, 296–303.

Van Dillen, L. F., & Koole, S. L. (2009). How automatic is oautomatic vigilance?: The role of working memory in attentional interference of negative information. *Cognition and Emotion, 23*, 1106–1117.

Van Dillen, L. F., Papies, E., & Hofmann, W. (2013). Turning a blind eye to temptation. How task load can facilitate self-regulation. *Journal of Personality and Social Psychology, 3*, 427–443.

Van Gucht, D., Baeyens, F., Vansteenwegen, D., Hermans, D., & Beckers, T. (2010). Counter-conditioning reduces cue-induced craving and actual cue-elicited consumption. *Emotion, 10*, 688–695.

Veling, H., Holland, R. W., & van Knippenberg, A. (2007). Devaluation of distracting stimuli. *Cognition and Emotion, 21*, 442–448.

Veling, H., Holland, R. W., & van Knippenberg, A. (2008). When approach motivation and behavioral inhibition collide: Behavior regulation through stimulus devaluation. *Journal of Experimental Social Psychology, 44*, 1013–1019.

Vohs, K. D., Baumeister, R. F., Mead, N. L., Hofmann, W., Ramanathan, S., & Schmeichel, B. J. (2012). *Engaging in self-control heightens urges and feelings*. Unpublished manuscript.

Wegner, D. M. (1994). Ironic processes of mental control. *Psychological Review, 101*, 34–52.

Wiers, R. W., Eberl, C., Rinck, M., Becker, E. S., & Lindenmeyer, J. (2011). Retraining automatic action tendencies changes alcoholic patients' approach bias for alcohol and improves treatment outcome. *Psychological Science, 22*, 490–497.

Wiers, R. W., Rinck, M., Kordts, R., Houben, K., & Strack, F. (2010). Retraining automatic action-tendencies to approach alcohol in hazardous drinkers. *Addiction, 105*, 279–287.

Winkielman, P., Berridge, K. C., & Wilbarger, J. L. (2005). Unconscious affective reactions to masked happy versus angry faces influence consumption behavior and judgments of value. *Personality and Social Psychology Bulletin, 31*, 121–135.

CHAPTER 22

Emotion Goals: How Their Content, Structure, and Operation Shape Emotion Regulation

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Much of human behavior is purposeful, or goal directed (Bandura, 1986; Carver & Scheier, 2000; Deci & Ryan, 1985; Fishbach & Ferguson, 2007; Gollwitzer, 1990; Mischel, Cantor, & Feldman, 1996). Goals can target our appearance (e.g., to be thin), our mind (e.g., to be smart), and our behavior (e.g., to spend more time with family or to work harder). As we argue in this chapter, some of our most important goals target emotions (e.g., to be happy). A *goal* is a "cognitive representation of a desired endpoint" (Fishbach & Ferguson, 2007, p. 491). Therefore, we define an *emotion goal* as the cognitive representation of a particular emotional state that is the desired endpoint. Although any goal may indirectly involve desired emotional endpoints (e.g., "I want to buy a car in order to feel happy as a result"), we focus on goals that directly target emotions as the desired endpoint (e.g., "I want to feel happy"). Emotion goals are a specific type of affect goal. Whereas affect goals target states of pleasure or pain, more generally (e.g., "I want to feel pleasant"), emotion goals target specific emotional states (e.g., "I want to feel joyful, proud, or amused"). Therefore, in this chapter, we distinguish *nonaffect goals* (i.e., goals that do not involve affective states as the direct desired endpoint), *affect goals* (i.e., goals that involve pleasure or pain as the direct desired endpoint), and *emotion goals* (i.e., goals that involve specific emotional states as the direct desired endpoint), and focus mainly on the latter.

It follows from this definition that people's emotion goals are foundational to emotion regulation. In fact, the activation of an emotion goal is necessary for emotion regulation (Gross, this volume; Mauss, Bunge, & Gross, 2007). Emotion goals determine whether people engage in emotion regulation, which emotions they attempt to regulate, when they cease their emotion regulatory efforts, and people's satisfaction with their emotion regulation attempts. Thus, understanding emotion goals has crucial implications for understanding emotion regulation and its effects on well-being.

To date, research on emotion regulation has focused on understanding different emotion regulation strategies and their outcomes (Gross & Thompson, 2007; Webb, Miles, & Sheeran, 2012). As a function of this focus, research to date has examined the "how" (e.g., What strategies do people use to regulate their emotions?) more than the "why" and "what" of emotion regulation (e.g., When do people decide to regulate an emotion? What emotional states do people want to attain?). The goal framework we outline here emphasizes the importance of understanding not only the "how" but also the "why" and "what" of emotion regulation.

A Goal Framework for Emotion Regulation

Although relatively little research has directly examined emotions as goals, there is ample research on goals and self-regulation in general (e.g., Baumeister & Heatherton, 1996; Carver & Scheier, 2000; Custers & Aarts, 2010). Applying this body of knowledge to the emotion domain could greatly advance the understanding of emotion regulation (cf. Harmon-Jones, Harmon-Jones, Amodio, & Gable, 2011; Koole, van Dillen, & Sheppes, 2011; Webb, Schweiger Gallo, Miles, Gollwitzer, & Sheeran, 2012). People's goals determine their actions as they attempt to decrease perceived discrepancies between current and desired states. As Figure 22.1 illustrates, when applied to emotions, this idea can help us understand what determines the initiation and course of emotion regulation. More specifically, perceived discrepancies between current emotional states (e.g., "I feel sad") and emotion goals ("I want to feel less sad") initiate emotion regulation, which is set in motion to bring current emotional states closer to desired emotion states. Thus, the emotion goals people hold determine whether or not they engage in emotion regulation, which emotions they attempt to regulate, and in which direction (i.e., increase or decrease). In this chapter we discuss emotion goals, building on available knowledge about goal pursuit. We organize this chapter around three features of goals that have been highlighted in research on goal pursuit (Fishbach & Ferguson, 2007): their content, their hierarchical structure, and their operation. First, we consider the content of emotion goals. We examine what emotion goals people adopt and highlight two factors that determine these goals: hedonic benefits (i.e., greater pleasure and less pain) and nonhedonic benefits (e.g., to prepare an organism for fight) of particular emotions. Second, we consider the structure of emotion goals. Multiple goals, including emotion and nonemotion ones, coexist at any given point in time and are hierarchically organized. We consider possible features of this organization and their implications. Third, we consider the operation of emotion goals that unfolds as people regulate their emotions, distinguishing relatively automatic from deliberate types of emotion regulation. Finally, we highlight the goal framework's implications for understanding the links between emotion regulation and well-being.[1]

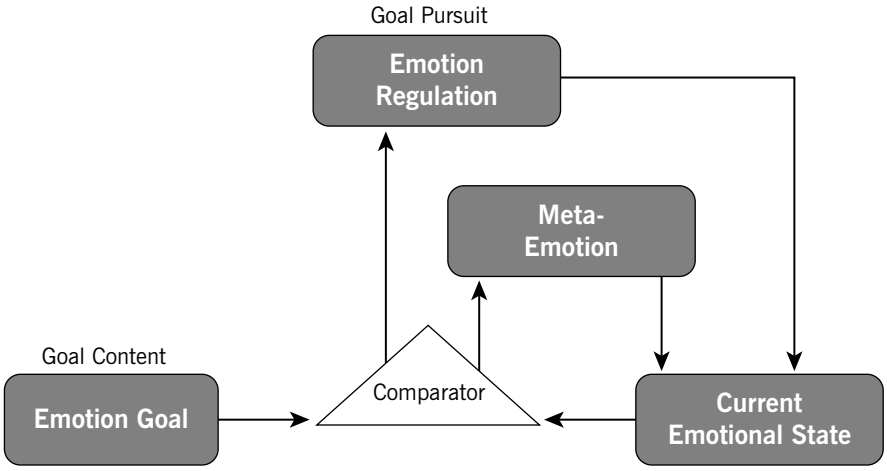

FIGURE 22.1. Hypothesized operation of emotion goals. Perceived discrepancy between current emotional states and emotion goals initiates and directs emotion regulation, which influences current emotional states. The evaluation of the difference between current emotional states and emotion goals is emotional in nature (e.g., contentment when discrepancy decreases, distress when discrepancy increases). We therefore refer to the output of this evaluation as meta-emotion.

The Content of Emotion Goals: Emotions as Desired States

What emotion goals do people have and what determines these goals? Because all creatures strive to attain pleasure and avoid pain, and because emotions are pleasant or painful subjective states, emotion goals are often determined by the immediate hedonic benefits of emotions. For example, people may be motivated to increase happiness because it is pleasant, and they may be motivated to decrease fear because it is unpleasant. Pleasant emotions generally are preferred to unpleasant emotions by people from different cultures (Diener, 2000; Tsai, Knutson, & Fung, 2006) and with different personality dispositions (Rusting & Larsen, 1995).

The immediate hedonic benefits of emotions are a powerful determinant of emotion goals. However, they are not the sole determinant. Functional theories of emotions hold that emotions serve to promote a broad array of nonhedonic benefits (Frijda, 1986; Nesse & Ellsworth, 2009). According to the instrumental approach to emotion regulation (Bonanno, 2001; Parrott, 1993; Tamir, 2009), people may be motivated to experience emotions to attain either hedonic or nonhedonic benefits. When hedonic benefits are prioritized, people are motivated to experience pleasant emotions and avoid unpleasant ones. However, when nonhedonic benefits are prioritized, people may be motivated to experience either pleasant or unpleasant emotions, depending on their instrumental implications (Tamir, 2009).

Emotions can offer at least three types of nonhedonic benefits. First, emotions can offer performance benefits. By engaging various physiological, cognitive, and motivational processes, emotions can change how effectively we deal with situational demands (Frijda, 1986). Second, emotions can have epistemic benefits. They provide us with important information regarding our state in the world (Clore, Gasper, & Garvin, 2001). Third, emotions can carry cultural benefits. In group contexts, emotions signal group membership and support of cultural values (Keltner & Haidt, 1999).

We believe that people may be motivated to experience emotions to attain any one of these benefits. First, emotions can orient behavior to deal with situational demands as effectively as possible (Frijda, 1986). For instance, joy may promote creativity (Fredrickson, 2001). Therefore, when performance is likely to benefit from increased creativity, joy might be useful for performance. People may be motivated to experience an emotion to attain its performance-related benefits. In support of this hypothesis, we have recently shown that people want to experience emotions they believe would promote their performance (Tamir, Salerno, Rhodes, & Schreier, 2012). In a series of studies, we led participants to expect anger to be either useful, irrelevant, or harmful for performance on an upcoming task. Participants were motivated to increase the experience of anger when they expected it be useful for performance, even though it was unpleasant to experience. This effect was obtained even when beliefs about usefulness were manipulated outside of conscious awareness. These findings demonstrate that emotion goals can be determined by the expected benefits of emotions for performance.

Second, emotions provide people with important information about themselves and their state in the world (Clore et al., 2001). Such information can support or conflict with core assumptions about who we are and what the world is like. People are generally motivated to preserve these core assumptions (Swann & Schroeder, 1995). Just as people seek feedback that maintains their self-image (Swann & Schroeder, 1995), people may be motivated to experience emotions that maintain their self-image (i.e., that have epistemic benefits). In support of this proposition, Wood and her colleagues showed that people with low self esteem are motivated to maintain sad feelings, because such feelings are familiar to them and because they reinforce their low sense of personal value (Wood, Heimpel, Manwell, & Whittington, 2009).

Third, emotions can reinforce one's commitment to and investment in particular cultural values. For instance, pride is tied to the value of personal achievement, whereas shame is tied to the value of social harmony (Kitayama, Mesquita, & Karasawa, 2006). Because cultures generally seek to preserve specific values, they prescribe certain emotions as more normative than others (Eid & Diener, 2001). In support of this notion,

Tsai and her colleagues (2006) have shown that cultures vary in the extent to which they value different emotional states. Whereas pleasant high-arousal states are more highly valued in individualistic cultures, pleasant low-arousal states are more highly valued in collectivistic cultures. Thus, beyond emotions' hedonic benefits, emotion goals may be determined by values that are prevalent in people's cultural context.

As the research reviewed here demonstrates, people are often motivated to experience emotions for their immediate hedonic benefits, but they can also be motivated to experience emotions for their performance-related benefits, epistemic benefits, or cultural benefits. This list of determinants of emotion goals is not exhaustive (for a more complete list, see Tamir & Bigman, in press). But it demonstrates that people can be motivated to experience almost any emotion. Thus, and perhaps surprisingly, emotion goals are not limited to pleasant emotions.

The Structure of Emotion Goals

Up to this point, we have treated emotion goals as though they are singular, isolated entities. However, people pursue many different goals at any given moment. As Figure 22.2 illustrates, goals can be ordered hierarchically according to their importance, centrality, and abstraction, with some goals assuming superordinate and others assuming subordinate positions (Carver & Scheier, 2000; Fishbach & Ferguson, 2007). To fully understand emotion goals, it is important to

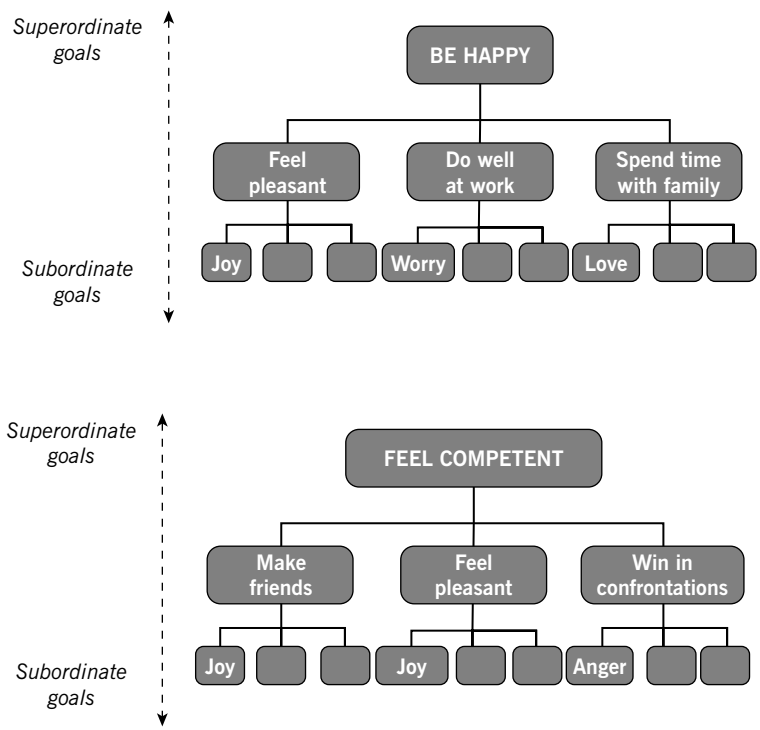

FIGURE 22.2. Hypothesized organization of emotion goals. Multiple goals are hierarchically organized, with some goals assuming superordinate and others assuming subordinate positions. Goals can be emotional or nonaffective in nature, and they can be compatible or in conflict with one another. As the examples illustrates, goal structure can be characterized by multifinality and equifinality. The goal of "joy" may serve the superordinate goal to be happy or to feel competent (multifinality). The superordinate goal to feel competent can be subserved by either joy or anger (equifinality). Conflict can arise when one goal (e.g., feel anger) subserves one superordinate goal (feel competent) but not another (be happy).

examine them within the hierarchical structure of a broader goal system.

Goal systems can be characterized by multifinality and equifinality (Kruglanski et al., 2002). *Multifinality* refers to the idea that a given subordinate goal may serve multiple superordinate goals (see Figure 22.2). In the context of emotion goals, this implies that a subordinate emotion goal (e.g., to feel joy) may serve superordinate affect goals (e.g., to feel pleasant) as well as superordinate nonaffect goals (e.g., to make friends). *Equifinality* refers to the idea that a given superordinate goal may be subserved by multiple subordinate goals (see Figure 22.2). In the context of emotion goals, this implies that a superordinate goal (e.g., to be happy) may be served by various affect (e.g., to feel good, to feel less bad) and nonaffect (e.g., to do well at work, to spend time with family) goals.

The pursuit of superordinate goals can automatically activate related subordinate goals (e.g., Fishbach & Ferguson, 2007). Adapting this principle to the study of emotion goals, we have recently shown that emotion goals can be activated by priming related superordinate goals (Tamir, Ford, & Ryan, 2013). Building on the idea that anger can impair collaboration, we showed that participants who were nonconsciously primed with the goal of collaboration became less motivated to experience anger before a social interaction. These findings could not be explained by concurrent emotional experiences and demonstrate that emotion goals can operate in the service of superordinate nonaffect goals.

Multifinality and equifinality of goals can thus facilitate goal pursuit because they offer multiple ways to achieve goals. However, as Figure 22.2 illustrates, the multifinality of goals can also give rise to goal conflict when subordinate goals simultaneously promote the pursuit of some superordinate goals and impair the pursuit of others. This may be particularly salient in the context of emotion goals, because emotions have both hedonic and nonhedonic implications. Pleasant emotions can either promote or impair the attainment of superordinate nonaffect goals (e.g., joy might help people make friends but also lead them to spend less time studying for an exam). However, pleasant emotions generally promote the attainment of superordinate affect goals (i.e., they feel good). Thus, goal conflict may be less likely when a pleasant emotion promotes both affect and nonaffect superordinate goals, and more likely when a pleasant emotion promotes an affect goal but impairs a nonaffect goal.

Goal conflict may be particularly likely when pursuing unpleasant emotions. Like pleasant emotions, unpleasant emotions can either promote or impair the attainment of superordinate nonaffect goals (e.g., anger might help people win a fight but it might also impair friendships). However, unlike pleasant emotions, unpleasant emotions often impair the attainment of superordinate affect goals (i.e., they feel bad). Therefore, the pursuit of unpleasant emotions depends on the relative strength of competing superordinate nonaffect and affect goals. People may be more likely to pursue subordinate unpleasant emotion goals when superordinate nonaffect goals become salient, and such pursuits likely involve some degree of goal conflict.

These ideas lie at the heart of the instrumental approach to emotion regulation (Tamir, 2009). According to this approach, people can pursue either pleasant or unpleasant emotion goals, to the extent that such goals serve salient superordinate goals. For instance, to the extent that anger promotes successful confrontation (e.g., Frijda, 1986), people may be motivated to feel angry when it is important for them to win a fight. In such cases, people would be motivated to experience unpleasant emotions, despite the immediate hedonic cost of doing so.

There is now a body of empirical evidence in support of these ideas. For instance, Tamir, Mitchell, and Gross (2008) tested whether people wanted to increase their anger when preparing for confrontation. Participants were given salient confrontational or nonconfrontational goals (e.g., kill enemies or build an empire in a virtual computer game). To assess emotion goals, participants indicated the extent to which they preferred to engage in various emotion-inducing activities, including those that were neutral, exciting, and anger-inducing. Although participants acknowledged that the anger-inducing activities would be unpleasant, they nonetheless preferred to engage in them when pursuing the confrontational, but not the nonconfrontational, goals. A similar pattern was found in an examination of people's prefer-

ences for anger before a face-to-face negotiation task (Tamir & Ford, 2012b). Participants who thought they were preparing for a confrontational negotiation showed stronger preferences for anger before the negotiation. In contrast, participants who thought they were preparing for a collaborative negotiation showed weaker preferences for anger and stronger preferences for happiness. Such preferences, in turn, were fully mediated by the belief that anger would promote or impair successful performance. These studies suggest that emotion goals can subserve nonaffect goals, and that people pursue unpleasant emotions when they expect them to subserve salient superordinate goals.

Interestingly, the relative importance of affect and nonaffect goals may shift systematically across the lifespan. Specifically, the importance of affect goals seems to change with age, with affect goals gaining in relative importance over nonaffect goals (Charles & Carstensen, this volume; Carstensen, Isaacowitz, & Charles, 1999). If people are likely to pursue unpleasant emotions when they subserve superordinate nonaffect goals, unpleasant emotion goals should be more prevalent in younger than in older adults. Indeed, wanting to maintain or increase unpleasant emotions and decrease pleasant emotions is more prevalent in younger than in older adults (Riediger, Schmiedek, Wagner, & Lindenberger, 2009).

In this section, we have discussed the structure of emotion goals, highlighting the fact that emotion goals operate within a broader goal system, which includes both affect and nonaffect goals that are hierarchically organized. The features of this organization have important implications for understanding how emotion goals interact with one another and with other types of goals. In the next section, we focus on the operation of emotion goals.

The Operation of Emotion Goals: Automatic and Deliberate Emotion Regulation

As illustrated in Figure 22.1, according to feedback, or cybernetic, models of self regulation, people initiate self-regulatory attempts when they perceive discrepancies between their current state and their goal (Baumeister & Heatherton, 1996; Carver & Scheier, 2000). When applied to emotion goals, these models direct our attention to an important element of emotion regulation. Goals can be represented outside of conscious awareness, as well as consciously (Custers & Aarts, 2010; Fishbach & Ferguson, 2007). People pursue conscious goals deliberately, whereas they pursue nonconscious goals relatively effortlessly and with little or no conscious awareness (automatically). It follows that emotion goals can set in motion relatively automatic as well as deliberate emotion regulation attempts. Research shows that nonemotion goals, such as achievement and cooperation, can be pursued automatically (e.g., Bargh, Gollwitzer, Lee-Chai, Barndollar, & Trötschel, 2001). Might emotion goals also be pursued in an automatic manner, with little effort and outside of conscious awareness?

While it has long been hypothesized that people can engage in emotion regulation unconsciously (Freud, 1936), only recently has empirical research begun to explore this possibility (see Gyurak & Etkin, this volume; Fitzsimons & Bargh, 2004; Gyurak, Gross, & Etkin, 2011; Mauss, Bunge, et al., 2007). To examine individual differences in automatic emotion regulation, we developed a variant of the Implicit Association Test (IAT; Greenwald, McGhee, & Schwartz, 1998) that estimates implicit evaluation of emotion control versus expression, the Emotion Regulation IAT (ER-IAT; Mauss, Evers, Wilhelm, & Gross, 2006, Study 1). We reasoned that people who implicitly evaluate emotion control positively would tend to engage in automatic emotion regulation (Aarts & Dijksterhuis, 2000; Custers & Aarts, 2005).

To examine how automatic emotion regulation predicts emotional responding we assessed whether ER-IAT scores were associated with responses to a laboratory anger provocation (Mauss et al., 2006, Study 2). While most participants became angry during the provocation, those with greater ER-IAT scores (i.e., those who implicitly evaluated emotion control more positively) reported relatively less anger experience. In addition, and in line with the notion that regulation attempts had taken place, they exhibited a challenge cardiovascular activation pattern, characterized by greater

cardiac output and lower total peripheral resistance (cf. Tomaka, Blascovich, Kelsey, & Leitten, 1993). The relative reduction of anger experience appeared to have happened without conscious effort, because associations between ER-IAT scores and anger responding held when researchers controlled for self-reported effortful emotion control. In summary, these findings are consistent with the idea that people who implicitly valued emotion control tended to regulate their emotion automatically and experienced less anger.

To examine the possible causal role of automatic emotion regulation, we (Mauss, Cook, & Gross, 2007) manipulated nonconscious emotion regulation goals by priming emotion control versus emotion expression with a sentence-unscrambling task (Srull & Wyer, 1979). Participants primed with emotion control in the laboratory responded with less anger to a subsequent anger provocation than did participants primed with emotion expression. The fact that participants were not aware of the purpose of the priming task suggests that these effects were not conscious. These conclusions were confirmed in a study in which participants were either explicitly instructed or primed outside of awareness to engage in emotion regulation. Participants in the priming condition achieved the same decrease in physiological reactivity to an anxiety induction as those explicitly instructed to regulate their emotion (Williams, Bargh, Nocera, & Gray, 2009).

Work on implementation intentions also suggests that emotion regulation can unfold automatically. An *implementation intention* is a plan that links situations to specific goal-directed behaviors (Gollwitzer, 1999), such as "After I get up in the morning, I will run 2 miles." By putting goal-directed behavior under the control of the situation in this way, the execution of the goal is removed from effortful and conscious control and rendered relatively automatic (Webb & Sheeran, 2007). Recent research suggests that implementation intentions can be used in the service of emotion regulation (for a review, see Webb, Schweiger Gallo, et al., 2012). For example, Schweiger-Gallo, Keil, McCulloch, Rockstroh, and Gollwitzer (2009) showed spider phobics images that included spiders. Participants were given no instructions, were asked to form a goal intention ("I will not get frightened!"), or were asked to form an implementation intention ("If I see a spider, then I will remain calm and relaxed!"). Afterward, when viewing spider pictures, participants who formed implementation intentions reported less negative affect and exhibited less physiological arousal compared to both other groups. This research further supports the idea that emotion regulation can take place without conscious awareness.

It appears, then, that the nonconscious pursuit of emotion goals may be just as effective as the conscious pursuit of emotion goals. Unlike conscious goal pursuit, however, nonconscious goal pursuit "eats up" less cognitive resources and is less effortful (Custers & Aarts, 2010; Fishbach, Friedman, & Kruglanski, 2003). Automatic emotion regulation, therefore, could help people cope with powerful negative situations without conscious effort. Given how important emotion regulation is to psychological health (Rottenberg & Johnson, 2007), the intriguing possibility arises that the automatic pursuit of emotion goals might play a beneficial role in psychological health. A recent study tested whether this might be the case (DeWall et al., 2011). It showed that after social exclusion, participants low in depressive symptoms or high in self-esteem automatically (without conscious intent) initiated the up-regulation of positive emotion. Similar findings have been obtained for individuals with healthy traits such as high action orientation or secure attachment (for review, see Koole & Rothermund, 2011). These findings suggest that automatic emotion regulation may be part of the *psychological immune system:* in healthy individuals a threat sets in motion an automatic emotion regulation process that leads to increased positive and decreased negative emotions.

This does not imply that automatic emotion regulation is *always* associated with beneficial outcomes. After all, defensiveness and repression are based on implicitly represented goals, yet they are associated with negative outcomes (Freud, 1936; Vaillant, 1977; Weinberger, 1995). The goal framework may help explain when automatic emotion regulation is beneficial versus harmful. As we discuss in the next section, automatic

emotion regulation may be beneficial to the extent that people (1) use effective regulation strategies and (2) pursue adaptive emotion goals (Hopp, Troy, & Mauss, 2011).

Implications for Well-Being

The goal framework leads to several novel predictions regarding when and why emotion regulation is associated with healthy versus unhealthy outcomes. Next, we examine two particularly important sets of predictions and empirical evidence to support them.

Emotion Goals and Emotion Regulation Strategies

People can use a multitude of emotion regulation strategies to attain emotion goals. For example, to feel less angry, Person *A* might think of something else whereas Person *B* may vent. The study of emotion regulation strategies and their relative adaptiveness (i.e., to what extent different types of emotion regulation strategies are associated with greater well-being) has paid relatively little attention to the emotion goals people pursue. We argue that it is fruitful to examine emotion regulation strategies in the context of emotion goals. The goal framework offers three specific hypotheses. First, the adaptiveness of emotion regulation strategies should depend on the extent to which they help a person achieve their emotion goals (i.e., to the extent that they are effective). Second, the adaptiveness of emotion regulation strategies should depend on not only their inherent features but also the extent to which they are used in a goal-sensitive, flexible manner. Third, the adaptiveness of emotion regulation strategies should depend on the extent to which they are used in the service of adaptive emotion goals.

Much research has focused on classifying the various emotion regulation strategies that exist (Gross & Thompson, 2007; Koole, 2009; Totterdell & Parkinson, 1999). One of the most prominent models distinguishes different emotion regulation strategies based on the stage in the emotion process in which they intervene (Gross & Thompson, 2007). According to this model, emotion regulation strategies can be characterized depending on whether they target the emotional situation, a person's attention to it, or appraisal of it (antecedent-focused) versus a later component of the emotional response, such as emotion-expressive behaviors (response-focused).

Which emotion regulation strategies are most effective for attaining emotion goals (e.g., decrease anger)? Antecedent-focused emotion regulation strategies should be relatively more effective at altering emotional responses because they have the advantage of a preventive strategy: they take place before the emotional response fully unfolds and thus should be more effective than response-focused emotion regulation strategies. A recent meta-analysis of 190 studies is broadly consistent with this hypothesis (Webb, Miles, et al., 2012). Are the most effective emotion regulation strategies also the most adaptive? Research comparing antecedent-focused emotion regulation (i.e., cognitive appraisal) to response-focused emotion regulation (i.e., expressive suppression) suggests that, indeed, on average reappraisal is associated with better psychological health (Garnefski & Kraaij, 2006; Gross & John, 2003; Troy, Wilhelm, Shallcross, & Mauss, 2010). Thus, there is some evidence that some of the most effective emotion regulation strategies are also relatively adaptive.

Importantly, from a goal perspective, the adaptiveness of an emotion regulation strategy should be determined by its inherent properties, but also by how flexibly it is used to support an individual's changing emotion goals (Bonanno & Burton, in press; Brandtstaedter & Rothermund, 2002; Cheng, 2001; Kashdan & Rottenberg, 2010). Findings in support of this notion have been obtained from daily diary studies in which participants reported on stressful life events and how many different coping strategies they used (Cheng, 2001). Flexibility was operationalized as participants' ability to vary coping strategies with the demand of the stressful event. Participants demonstrating greater flexibility exhibited greater well-being compared to participants who adhered more rigidly to particular coping strategies, regardless of the particular type of coping strategy.

Bonanno, Papa, Lalande, Westphal, and Coifman (2004) tested a related idea by deriving a laboratory measure of how well

participants were able to match their emotion regulation efforts to changing goals (either increase or decrease emotional expression). Participants who were better able to regulate their emotions in pursuit of their concurrent goals reported greater psychological health after the September 11, 2001 attacks (Bonanno et al., 2004) and greater well-being after high life stress (Westphal, Seivert, & Bonanno, 2010).

From a goal framework, emotion regulation strategies are therefore adaptive to the extent that they help people attain their concurrent emotion goals. However, the adaptiveness of emotion regulation more generally depends on which emotion goals people are trying to achieve. If people hold maladaptive emotion goals, even the most effective and flexible emotion regulation strategies should not be adaptive. Which emotion goals are adaptive? Perhaps those that are sensitive to situational demands and are consistent with superordinate goals and basic needs (Deci & Ryan, 1985; Troy, Shallcross, & Mauss, in press) (e.g., increase joy in the service of successfully collaborating with others, and increase anger in the service of successfully confronting others). Consistent with these ideas, we found that the more people wanted to feel angry and the less they wanted to feel happy in confrontational situations, the higher their psychological well-being. The converse pattern was found the more angry and the less happy people wanted to feel in collaborative situations (Tamir & Ford, 2012a).

In summary, the goal framework can help us understand when and why emotion regulation strategies are adaptive or maladaptive. Emotion regulation strategies are adaptive to the extent that they help individuals attain their emotion goals, are used in a goal-sensitive and flexible manner, and are used in the context of adaptive emotion goals.

Feedback Processing: Emotion Goals, Evaluation, and Meta-Emotion

Emotion regulation does not operate in a linear, one-directional fashion. Rather, as Figure 22.1 illustrates, it involves recursive feedback loops. Feedback models of self-regulation (Carver & Scheier, 2000) propose that people monitor the discrepancy between their current state and the desired end state, as well as the progress of their efforts to decrease discrepancies between the two. Importantly, the output of this monitoring process is emotional in nature. When people progress faster than expected toward their goal, they feel positively (e.g., contentment); when people progress more slowly than expected they feel negatively (e.g., distress). Because these emotional states are about an emotional state, they can be referred to as meta-emotion. Considering emotion regulation in the context of this feedback loop leads to two interesting predictions. First, decreasing the discrepancy between current and desired state should yield better well-being, whether the discrepancy reduction occurs by changing one's current emotional state or by changing one's desired emotional state. Conversely, an increased discrepancy between current and desired state should yield negative outcomes, whether it is due to one's current or desired emotional state. Second, meta-emotion may play an important role in the effects of emotion regulation on well-being.

The hypothesis that adjusting one's goals (in addition to or instead of one's current state) plays an important role in well-being has been supported in the context of coping with stressors (e.g., Brandtstaedter & Rothermund, 2002; Wrosch, Scheier, Miller, Schulz, & Carver, 2003). This research suggests that goal adjustment (e.g., failing to disengage from impossible goals) is at least as important to well-being as the effective pursuit of goals. This principle also appears to apply to emotion goals. In one experimental study, researchers manipulated participants' emotion goals by instructing them to make themselves feel as happy as possible while they listened to emotionally ambiguous music (i.e., their desired emotional end state was set to a highly positive state). In line with the idea that unrealistic emotion goals can lead to negative emotional outcomes, participants in this condition were less happy compared to participants who were not given an emotion goal (Schooler, Ariely, & Loewenstein, 2003). In another study, researchers manipulated emotion goals more subtly by presenting to participants a sham newspaper article discussing the advantages of happiness. Participants induced in this way to assume a happiness goal were less happy than control participants after subse-

quently watching a happy film clip (Mauss, Tamir, Anderson, & Savino, 2011, Study 2). This research converges to support the hypothesis that holding unrealistic emotion goals (e.g., high levels of happiness) can lead to decreased positive emotion.

Do people who chronically hold unrealistic emotion goals experience more negative well-being outcomes? We examined this question by measuring the extent to which participants held the goal to be happy, with items such as "Feeling happy is extremely important to me." On average, the more participants valued happiness, the lower their emotional well-being (Mauss et al., 2011, Study 1), and the higher their likelihood of being diagnosed with major depressive disorder (Ford, Shallcross, Mauss, Floerke, & Gruber, under review). These studies make the point that one's emotion goals play an important role in well-being and psychological health.

The perspective that adjusting one's emotion goals is one important avenue to well-being brings insight to a puzzling area of research: that of emotional acceptance. *Acceptance* is defined as the process of nonjudgmentally engaging with negative emotions (Teasdale et al., 2000). Correlational and experimental research on acceptance has consistently found it to be negatively correlated with negative emotion and mood disorder (Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Kashdan, Barrios, Forsyth, & Steger, 2006; Roemer, Salters, Raffa, & Orsillo, 2005; Shallcross, Troy, Boland, & Mauss, 2010). The inverse relationship between acceptance and negative affect may appear paradoxical at first: How can *engaging with* negative emotions be associated with *less* negative emotion? The goal perspective suggests one solution to this apparent paradox: Acceptance may involve more realistic emotion goals, which in turn lead to greater well-being. Well-being, therefore, is a function of not only effective emotion regulation but also having attainable emotion goals. Or, in the words of the adage, "Happiness is not having what you want, but wanting what you have."

As there are with nonemotion goals, there are likely costs for inflexibly pursuing emotion goals that are difficult to attain. However, unlike nonemotion goals, because it leads to meta-emotion, the pursuit of unattainable *emotion* goals can be self-defeating. For instance, in the study discussed earlier (Mauss et al., 2011, Study 2), compared to participants in the control condition, participants who were led to pursue happiness goals ended up feeling more disappointed in their emotional state, which resulted in less happiness. Interestingly, these effects were only observed for participants who watched a happy film clip, not for those who watched a sad film clip. This may be because in relatively negative situations (e.g., when watching a sad film clip), people have a good reason *not* to feel happy, and are less likely to feel disappointed if they fail to meet their happiness goal. Conversely, in relatively positive situations (e.g., when watching a happy film clip), people have every reason to feel happy and ironically end up feeling disappointed when they do not. Whereas the pursuit of nonemotion goals influences behavior and results in emotions, the pursuit of emotion goals influences emotions and results in emotions. Thus, when pursuing emotion goals, meta-emotional experiences can interfere with successful goal pursuit.

In summary, the goal framework highlights the fact that people's well-being is determined by not only how they pursue emotion goals but the goals themselves. Setting exceedingly positive emotion goals or failing to adjust emotion goals can, ironically, lead to less positive emotion and to lower well-being. These effects are due in part to people's meta-emotion (how they feel about their feelings).

Conclusions and Future Directions

In this chapter, we argue that it is crucial to consider emotion regulation in the context of the emotions people want to feel. Understanding the content of emotion goals helps us better understand the initiation and course of emotion regulation; understanding the structure of emotion goals helps us better understand how emotion and other goals interact with one another in goal hierarchies; and understanding the operation of emotion goals helps us better understand the emotion regulation process.

Taking a goal perspective can offer answers to questions such as the following:

When do people regulate their emotions? How do multiple, potentially conflicting goals interact with one another? What role do automatic processes play in the pursuit of emotion goals? How do emotion goals and pursuit of them affect well-being? In addition to guiding us to approach these novel questions, the proposed goal framework helps us critically examine some core assumptions in research on emotion regulation. For example, one core assumption is that people want to feel pleasant emotions and avoid unpleasant emotions. However, the goal framework challenges this assumption (Tamir, 2009; Tamir & Ford, 2009). Positioning emotion goals in a broader goal hierarchy leads us to predict that individuals will seek more unpleasant emotions in pursuit of superordinate nonaffect goals.

Another core assumption in research on emotion regulation is that there is something inherently beneficial or harmful about particular emotion regulation strategies. The goal framework suggests that emotion regulation and its implications for well-being can be fully understood only in the context of a person's broader goal hierarchy (Bonanno & Burton, in press; Thompson, 2011; Troy et al., in press).

While some research has already adopted a goal framework in emotion regulation, more work needs to be done on each of the three domains of emotion goals on which we have focused (i.e., their content, structure, and operation). In terms of the content of emotion goals, it will be important to develop a systematic approach to measuring these goals, whether they are transient or more chronically held. More work is necessary especially to measure implicitly represented emotion goals (those not readily accessible to introspection). In addition, important open questions remain about what biological, psychological, and cultural factors shape people's emotion goals.

In terms of the structure of emotion goals, it will be important to obtain a systematic and comprehensive understanding of how emotion and nonaffect goals interact with one another, and to identify the implications of different types of goal conflict. For example, how malleable are the associations between subordinate and superordinate emotion and nonaffect goals? What are the short- and long-term implications of conflict among goals? Of particular interest here might be conflict among explicit (relatively conscious) and implicit (relatively unconscious) emotion goals. Understanding conflict among explicit and implicit goals has been fruitful in domains such as achievement motives (Brunstein, Schultheiss, & Grässman, 1998); it would be promising in the domain of emotion goals as well.

In terms of the operation of emotion goals, there is still much to learn about their nonconscious representation and automatic pursuit. For instance, what gives rise to individual differences in automatic emotion regulation? What are its costs and benefits? In addition, the goal framework makes several predictions about emotion goals' implications for well-being. For example, it implies that healthy functioning hinges on the selection of appropriate emotion goals, interactions among emotion regulation strategies and emotion goals, and the meta-emotions that arise as a function of emotion goal pursuit. These features and implications of emotion goals have yet to be fully explored.

Note

1. Our review is selective. We do not cover concepts such as emotional effects of goals and goal pursuit (e.g., feelings of disappointment when not attaining a nonemotion goal, positive evaluation of goals) or goals that are infused with a lot of emotion (e.g., a goal about which one feels passionate). While these phenomena are important, they are distinct from the focus of this chapter.

References

Aarts, H., & Dijksterhuis, A. (2000). Habits as knowledge structures: Automaticity in goal-directed behavior. *Journal of Personality and Social Psychology, 78*, 53–63.

Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory.* Upper Saddle River, NJ: Prentice Hall.

Bargh, J. A., Gollwitzer, P. M., Lee-Chai, A., Barndollar, K., & Trötschel, R. (2001). The automated will: Nonconscious activation and pursuit of behavioral goals. *Journal of Personality and Social Psychology, 81*, 1014–1027.

Baumeister, R. F., & Heatherton, T. F. (1996).

Self-regulation failure: An overview. *Psychological Inquiry, 7*, 1–15.

Bonanno, G. A. (2001). Emotion self-regulation. In T. J. Mayne & G. A. Bonanno (Eds.), *Emotions: Current issues and future directions* (pp. 251–285). New York: Guilford Press.

Bonanno, G. A., & Burton, C. (in press). Regulatory flexibility: An individual differences perspective on coping and emotion regulation. *Perspectives on Psychological Science.*

Bonanno, G. A., Papa, A., Lalande, K., Westphal, M., & Coifman, K. (2004). The importance of being flexible: The ability to both enhance and suppress emotional expression predicts long term adjustment. *Psychological Science, 15*, 482–487.

Brandtstaedter, J., & Rothermund, K. (2002). The life-course dynamics of goal pursuit and goal adjustment: A two-process framework. *Developmental Review, 22*, 117–150.

Brunstein, J. C., Schultheiss, O. C., & Grässman, R. (1998). Personal goals and emotional well-being: The moderating role of motive dispositions. *Journal of Personality and Social Psychology, 75*, 494–508.

Campbell-Sills, L., Barlow, D. H., Brown, T. A., & Hofmann, S. G. (2006). Effects of suppression and acceptance on emotional responses of individuals with anxiety and mood disorders. *Behaviour Research and Therapy, 44*, 1251–1263.

Carstensen, L. L., Isaacowitz, D. M., & Charles, S. T. (1999). Taking time seriously: A theory of socioemotional selectivity. *American Psychologist, 54*, 165–181.

Carver, C. S., & Scheier, M. F. (2000). On the structure of behavioral self-regulation. In M. Boekaerts, P. R. Pintrich, & M. Zeidner (Eds.), *Handbook of self-regulation* (pp. 41–84). San Diego, CA: Academic Press.

Charles, S. T., & Carstensen, L. L. (this volume, 2014). Emotion regulation and aging. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 203–218). New York: Guilford Press.

Cheng, C. (2001). Assessing coping flexibility in real-life and laboratory settings: A multimethod approach. *Journal of Personality and Social Psychology, 80*, 814–833.

Clore, G. L., Gasper, K., & Garvin, E. (2001). Affect as information. In J. P. Forgas (Ed.), *Handbook of affect and social cognition* (pp. 121–144). Mahwah, NJ: Erlbaum.

Custers, R., & Aarts, H. (2005). Positive affect as implicit motivator: On the nonconscious operation of behavioral goals. *Journal of Personality and Social Psychology, 89*, 129–142.

Custers, R., & Aarts, H. (2010). The unconscious will: How the pursuit of goals operates outside of conscious awareness. *Science, 329*, 47–50.

Deci, E. L., & Ryan, R. M. (1985). *Intrinsic motivation and self-determination in human behavior.* New York: Plenum.

DeWall, C. N., Twenge, J. M., Koole, S. L., Baumeister, R. F., Marquez, A., & Reid, M. W. (2011). Automatic emotion regulation after social exclusion: Tuning to positivity. *Emotion, 11*, 623–636.

Diener, E. (2000). Subjective well-being: The science of happiness and a proposal for a national index. *American Psychologist, 55*, 34–43.

Eid, M., & Diener, E. (2001). Norms for experiencing emotions in different cultures: Inter- and intranational differences. *Journal of Personality and Social Psychology, 81*, 869–885.

Fishbach, A., & Ferguson, M. F. (2007). The goal construct in social psychology. In A. W. Kruglanski & E. T. Higgins (Eds.), *Social psychology: Handbook of basic principles* (pp. 490–515). New York: Guilford Press.

Fishbach, A., Friedman, R. S., & Kruglanski, A. W. (2003). Leading us not unto temptation: Momentary allurements elicit overriding goal activation. *Journal of Personality and Social Psychology, 84*, 296–309.

Fitzsimons, G. M., & Bargh, J. A. (2004). Automatic self-regulation. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 151–170). New York: Guilford Press.

Ford, B. Q., Shallcross, A. J., Mauss, I. B., Floerke, V. A., & Gruber, J. (under review). *Desperately seeking happiness: Valuing happiness is associated with symptoms and diagnosis of depression.*

Fredrickson, B. L. (2001). The role of positive emotions in positive psychology: The broaden-and-build theory of positive emotions. *American Psychologist, 56*, 218–226.

Freud, A. (1936). *The ego and the mechanisms of defense.* New York: International Universities Press.

Frijda, N. H. (1986). *The emotions.* New York: Cambridge University Press.

Garnefski, N., & Kraaij, V. (2006). Relationships between cognitive emotion regulation strate-

gies and depressive symptoms: A comparative study of five specific samples. *Personality and Individual Differences, 40*, 1659–1669.

Gollwitzer, P. M. (1990). Action phases and mind-sets. In E. T. Higgins & R. M. Sorrentino (Eds.), *Handbook of motivation and cognition: Foundations of social behavior* (Vol. 2, pp. 53–92). New York: Guilford Press.

Gollwitzer, P. M. (1999). Implementation intentions: Strong effects of simple plans. *American Psychologist, 54*, 493–503.

Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. K. (1998). Measuring individual differences in implicit cognition: The Implicit Association Test. *Journal of Personality and Social Psychology, 74*, 1464–1480.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*, 348–362.

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York: Guilford Press.

Gyurak, A., & Etkin, A. (this volume, 2014). A neurobiological model of implicit and explicit emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 76–90). New York: Guilford Press.

Gyurak, A., Gross, J. J., & Etkin, A. (2011). Explicit and implicit emotion regulation: A dual-process framework. *Cognition and Emotion, 25*, 400–412.

Harmon-Jones, E., Harmon-Jones, C., Amodio, D. M., & Gable, P. A. (2011). Attitudes toward emotions. *Journal of Personality and Social Psychology, 101*, 1332–1350.

Hopp, H., Troy, A. S., & Mauss, I. B. (2011). The unconscious pursuit of emotion regulation: Implications for psychological health. *Cognition and Emotion, 25*, 532–545.

Kashdan, T. B., Barrios, V., Forsyth, J. P., & Steger, M. F. (2006). Experiential avoidance as a generalized psychological vulnerability: Comparisons with coping and emotion regulation strategies. *Behaviour Research and Therapy, 44*, 1301–1320.

Kashdan, T. B., & Rottenberg, J. (2010). Psychological flexibility as a fundamental aspect of health. *Clinical Psychology Review, 30*, 865–878.

Keltner, D., & Haidt, J. (1999). Social functions of emotions at four levels of analysis. *Cognition and Emotion, 13*, 505–521.

Kitayama, S., Mesquita, B., & Karasawa, M. (2006). Cultural affordances and emotional experience: Socially engaging and disengaging emotions in Japan and the United States. *Journal of Personality and Social Psychology, 91*, 890–903.

Koole, S. L. (2009). The psychology of emotion regulation: An integrative review. *Cognition and Emotion, 23*, 4–41.

Koole, S. L., & Rothermund, K. (2011). "I feel better but I don't know why": The psychology of implicit emotion regulation. *Cognition and Emotion, 25*, 389–399.

Koole, S. L., van Dillen, L. F., & Sheppes, G. (2011). The self-regulation of emotion. In K. D. Vohs & R. F. Baumeister (Eds.), *Handbook of self-regulation: Research, theory, and applications* (2nd ed., pp. 22–40). New York: Guilford Press.

Kruglanski, A. W., Shah, J. Y., Fishbach, A., Friedman, R., Chun, W. Y., & Sleeth-Keppler, D. (2002). A theory of goal-systems. In M. P. Zanna (Ed.), *Advances in experimental psychology* (pp. 331–378). New York: Academic Press.

Mauss, I. B., Bunge, S. A., & Gross, J. J. (2007). Automatic emotion regulation. *Social and Personality Psychology Compass, 1*, 146–167.

Mauss, I. B., Cook, C. L., & Gross, J. J. (2007). Automatic emotion regulation during anger provocation. *Journal of Experimental Social Psychology, 43*, 698–711.

Mauss, I. B., Evers, C., Wilhelm, F. H., & Gross, J. J. (2006). How to bite your tongue without blowing your top: Implicit evaluation of emotion regulation predicts affective responding to anger provocation. *Personality and Social Psychology Bulletin, 32*, 589–602.

Mauss, I. B., Tamir, M., Anderson, C. L., & Savino, N. S. (2011). Can seeking happiness make people unhappy?: Paradoxical effects of valuing happiness. *Emotion, 11*, 807–815.

Mischel, W., Cantor, N., & Feldman, S. (1996). Principles of self-regulation: The nature of willpower and self-control. In E. T. Higgins & A. W. Kruglanski (Eds.), *Social psychology: Handbook of basic principles* (pp. 329–360). New York: Guilford Press.

Nesse, R. M., & Ellsworth, P. C. (2009). Evolution, emotions, and emotional disorders. *American Psychologist*, 129–139.

Parrott, W. G. (1993). Beyond hedonism: Motives for inhibiting good moods and for maintaining bad moods. In D. M. Wegner & J. W. Pennebaker (Eds.), *Handbook of mental control* (pp. 278–305). Upper Saddle River, NJ: Prentice Hall.

Riediger, M., Schmiedek, F., Wagner, G. G., & Lindenberger, U. (2009). Seeking pleasure and seeking pain: Differences in prohedonic and contra-hedonic motivation from adolescence to old age. *Psychological Science, 20*, 1529–1535.

Roemer, L., Salters, K., Raffa, S. D., & Orsillo, S. M. (2005). Fear and avoidance of internal experiences in GAD: Preliminary tests of a conceptual model. *Cognitive Therapy and Research, 29*, 71–88.

Rottenberg, J., & Johnson, S. L. (2007). *Emotion and psychopathology: Bridging affective and clinical science*. Washington, DC: American Psychological Association.

Rusting, C. L., & Larsen, R. J. (1995). Moods as sources of stimulation: Relationships between personality and desired mood states. *Personality and Individual Differences, 18*, 321–329.

Schooler, J. W., Ariely, D., & Loewenstein, G. (2003). The pursuit and monitoring of happiness can be self-defeating. In J. Carrillo & I. Brocas (Eds.), *Psychology and economics* (pp. 41–70). Oxford, UK: Oxford University Press.

Schweiger-Gallo, I. S., Keil, A., McCulloch, K. C., Rockstroh, B., & Gollwitzer, P. M. (2009). Strategic automation of emotion regulation. *Journal of Personality and Social Psychology, 96*, 11–31.

Shallcross, A. J., Troy, A. S., Boland, M., & Mauss, I. B. (2010). Let it be: Accepting negative emotional experiences predicts decreased negative affect and depressive symptoms. *Behaviour Research and Therapy, 48*, 921–929.

Srull, T. K., & Wyer, R. S. (1979). Role of category accessibility in the interpretation of information about persons—Some determinants and implications. *Journal of Personality and Social Psychology, 37*, 1660–1672.

Swann, W. B., & Schroeder, D. G. (1995). The search for beauty and truth—A framework for understanding reactions to evaluations. *Personality and Social Psychology Bulletin, 21*, 1307–1318.

Tamir, M. (2009). What do people want to feel and why?: Pleasure and utility in emotion regulation. *Current Directions in Psychological Science, 18*, 101–105.

Tamir, M., & Bigman, Y. (in press). When do people want to feel bad?: A taxonomy of motives. In W. G. Parrott (Ed.), *The positive side of negative emotions*. New York: Guilford Press.

Tamir, M., Bigman, Y., Rhodes, E., Salerno, J., & Schreier, J. (2013). *Utilitarian emotions: Expected usefulness of emotions shapes emotion regulation, experience, and decision making*. Manuscript under review.

Tamir, M., & Ford, B. Q. (2009). Choosing to be afraid: Preferences for fear as a function of goal pursuit. *Emotion, 9*, 488–497.

Tamir, M., & Ford, B. Q. (2012a). Should people pursue feelings that feel good or feelings that do good?: Emotional preferences and well-being. *Emotion, 12*, 1061–1070.

Tamir, M., & Ford, B. Q. (2012b). When feeling bad is expected to be good: Emotion regulation and outcome expectancies in social conflicts. *Emotion, 12*, 807–816.

Tamir, M., Ford, B. Q., & Ryan, E. (2013). Nonconscious goals can shape what people want to feel. *Journal of Experimental Social Psychology, 49*, 292–297.

Tamir, M., Mitchell, C., & Gross, J. J. (2008). Hedonic and instrumental motives in anger regulation. *Psychological Science, 19*, 324–328.

Teasdale, J. D., Segal, Z. V., Williams, J. M. G., Ridgeway, V. A., Soulsby, J. M., & Lau, M. A. (2000). Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *Journal of Consulting and Clinical Psychology, 68*, 615–623.

Thompson, R. A. (2011). Emotion and emotion regulation: Two sides of the developing coin. *Emotion Review, 3*, 53–61.

Tomaka, J., Blascovich, J., Kelsey, R. M., & Leitten, C. L. (1993). Subjective, physiological, and behavioral effects of threat and challenge appraisal. *Journal of Personality and Social Psychology, 65*, 248–260.

Totterdell, P., & Parkinson, B. (1999). Use and effectiveness of self-regulation strategies for improving mood in a group of trainee teachers. *Journal of Occupational Health Psychology, 4*, 219–232.

Troy, A. S., Shallcross, A. J., & Mauss, I. B. (in press). A person-by-situation approach to emotion regulation: Cognitive reappraisal can either hurt or help depending on the context. *Psychological Science*.

Troy, A. S., Wilhelm, F. H., Shallcross, A. J., & Mauss, I. B. (2010). Seeing the silver lining: Cognitive reappraisal ability moderates the relationship between stress and depression. *Emotion, 10*, 783–795.

Tsai, J. L., Knutson, B., & Fung, H. H. (2006). Cultural variation in affect valuation. *Journal of Personality and Social Psychology, 90*, 288–307.

Vaillant, G. (1977). *Adaptation to life*. Boston: Little, Brown.

Webb, T. L., Miles, E., & Sheeran, P. (2012). Dealing with feeling: A meta-analysis of the effectiveness of strategies derived from the process model of emotion regulation. *Psychological Bulletin, 138*, 775–808.

Webb, T. L., Schweiger Gallo, I., Miles, E., Gollwitzer, P. M., & Sheeran, P. (2012). Effective regulation of affect: An action control perspective on emotion regulation. *European Review of Social Psychology, 23*, 143–186.

Webb, T. L., & Sheeran, P. (2007). How do implementation intentions promote goal attainment?: A test of component processes. *Journal of Experimental Social Psychology, 43*, 295–302.

Weinberger, D. A. (1995). The construct validity of the repressive coping style. In J. L. Singer (Ed.), *Repression and dissociation: Implications for personality theory, psychopathology, and health* (pp. 337–386). Chicago: University of Chicago Press.

Westphal, M., Seivert, N. H., & Bonanno, G. A. (2010). Expressive flexibility. *Emotion, 10*, 92–100.

Williams, L. E., Bargh, J. A., Nocera, C. C., & Gray, J. R. (2009). The unconscious regulation of emotion: Nonconscious reappraisal goals modulate emotional reactivity. *Emotion, 9*, 847–854.

Wood, J. V., Heimpel, S. A., Manwell, L. A., & Whittington, E. J. (2009). This mood is familiar and I don't deserve to feel better anyway: Mechanisms underlying self-esteem differences in motivation to repair sad moods. *Journal of Personality and Social Psychology, 96*, 363–380.

Wrosch, C., Scheier, M. F., Miller, G. E., Schulz, R., & Carver, C. S. (2003). Adaptive self-regulation of unattainable goals: Goal disengagement, goal reengagement, and subjective well-being. *Personality and Social Psychology Bulletin, 29*, 1494–1508.

CHAPTER 23

Self-Awareness and Self-Relevant Thought in the Experience and Regulation of Emotion

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Dina Gohar

The evolution of human self-awareness constituted a seismic shift in mammalian psychology. Although some of our prehuman ancestors may have possessed rudimentary forms of self-awareness, much like modern chimpanzees and orangutans, evidence suggests that prehistoric people did not begin to think consciously about themselves until around 2 million years ago. Moreover, the archeological record indicates that people did not have the capacity to think about themselves in the abstract and symbolic ways that characterize modern human beings until culture began to appear between 40,000 and 60,000 years ago (Leary & Buttermore, 2003). The evolution of self-awareness had important implications for human beings' emotional lives, because human emotions often arise from the ways in which people think about themselves and the events that happen to them. Self-awareness and self-relevant thought have implications for the stimuli that trigger emotional states, the specific emotions that people experience, and people's efforts to manage and regulate their emotions.

Evidence suggests that most animals lack the ability to think consciously about themselves. Yet animals nonetheless experience a variety of emotions (Veissier, Boissy, Désiré, & Greiveldinger, 2009), and people sometimes experience emotions even when they are not self-aware. Thus, conscious self-awareness is not necessary for emotional experience. The emotional reactions of animals that lack self-awareness and those of human beings when they are not self-aware are controlled by two primary processes that occur without conscious awareness. First, many stimuli naturally and automatically evoke emotions. For example, most species instinctively react to certain threat cues without conscious thought, such as when the silhouette of a hawk in flight elicits fear in many birds, or when human beings react with fear to an object that approaches them suddenly. Other emotions, such as anger, sadness, and joy, appear to be similarly hard-wired.

Second, animals may learn to experience emotions in response to previously neutral stimuli through classical conditioning. For example, human and nonhuman animals may come to respond with negative emotions to otherwise neutral stimuli that have been repeatedly paired with aversive stimuli, or to respond positively to a neutral stimulus that has been associated with rewarding events. Indeed, clinical psychologists have used conditioning techniques such as systematic desensitization and counterconditioning to change people's emotional responses and

improve their emotion regulation in the treatment of anxiety problems such as phobias, panic attacks, and posttraumatic stress disorder (Foa, Huppert, & Cahill, 2006).

Although emotions can occur without self-awareness, people's capacity for self-awareness and self-relevant thought renders their emotional lives far more complex than those of animals that lack self-awareness. In this chapter, we discuss four basic ways in which self-awareness affects emotional experience and examine how people intentionally capitalize on these processes to manage their emotions. Specifically, due to their ability to think consciously about themselves, people's emotions are affected when they (1) compare themselves to their personal standards, (2) think about themselves in the past and future, (3) evaluate their personal characteristics, and (4) think about how they are perceived by other people. Each of these processes requires the ability to self-reflect.[1]

Comparisons to Personal Standards

Emotions often result when people assess the implications of a particular situation, event, or experience for their personal concerns (Frijda, 1986). Generally, these assessments involve a comparison of real or imagined outcomes to some goal or standard. Decades of research have shown that people's goals and standards influence their reactions to events, particularly when they are thinking consciously about them at the time, which requires self-awareness.

Self-Awareness and Personal Standards

According to various theories of self-awareness (Carver & Scheier, 1981; Duval & Wicklund, 1972), being self-aware induces an evaluative process in which people compare themselves to whatever standards are salient at that particular moment. For example, becoming self-aware while taking a test may lead people to evaluate themselves with regard to standards involving intellectual ability, whereas being self-aware while trying on new clothes may lead people to compare themselves to standards of physical attractiveness. Self-awareness is not necessary for goal pursuit (Bargh, Gollwitzer, & Lee-Chai, 2001); nevertheless, becoming self-aware seems to cause people to compare themselves and their behaviors to salient goals.

Becoming self-aware induces an evaluative process that evokes positive emotions when conditions meet or exceed one's standards and negative emotions when they do not. Because discrepancies between people's standards and their behavior are unpleasant, people who are self-aware try to avoid violating their personal standards and reduce such discrepancies when they occur (Duval & Wicklund, 1972; Macrae, Bodenhausen, & Milne, 1998; Scheier, Fenigstein, & Buss, 1974). In brief, many emotional experiences involve a comparison of situations, events, or experiences to goals and standards, and self-awareness is intimately involved in people's emotional reactions to events that have implications for their self-relevant goals.

According to self-discrepancy theory (Higgins, 1987), the specific emotions that people experience when they fail to achieve their goals depend on the nature of the discrepancy involved. When people perceive a discrepancy between how they think they are and how they would like to be (i.e., between their actual and ideal selves), dejection-related emotions such as disappointment, sadness, and depression predominate. In contrast, a discrepancy between how people think they are and how they think they ought to be (i.e., between actual and ought selves) leads to agitation-related emotions such as guilt, fear, and anxiety. Research has generally supported the notion that self-discrepancies influence emotion, but specific discrepancies do not always predict distinct emotions precisely as self-discrepancy theory predicts (Phillips & Silvia, 2010; Tangney, Niedenthal, Covert, & Barlow, 1998). For example, Phillips and Silvia (2010) found that although actual–ought discrepancies uniquely predicted anxiety, both actual–ideal and actual–ought discrepancies predicted depressed affect. When people think that they are close to becoming their feared selves, these actual–feared discrepancies predict agitation-related emotions better than do actual–ought discrepancies (i.e., how people think they ought to be), which more strongly predict emotions when people think they are far from assuming a feared identity (Carver, Lawrence, & Scheier, 1999).

In any case, discrepancies between how people think they are and how they want (and do not want) to be evoke negative emotions and lower subjective well-being (Brown & McConnell, 2011; Philips, Silvia, & Paradise, 2007; Tangney et al., 1998). When people make internal attributions for these discrepancies, they are especially likely to experience strong negative emotions (Petrocelli & Smith, 2005). For example, attributing a negative outcome to one's own bad decisions or incompetent behavior would likely result in different emotions than attributing the same outcome to another person's malevolent actions. Only when people believe that they played some role in undesired outcomes do self-discrepancies come into play. In addition, increased self-awareness (e.g., induced by seeing one's reflection in a mirror; Phillips & Silvia, 2005) and dispositional private self-consciousness (Fromson, 2006) both strengthen the relationship between self-discrepancies and negative emotions. These findings suggest that people can reduce the emotional impact of self-relevant discrepancies both by making external attributions for them and by reducing self-awareness.

Minimizing Self-Awareness

One implication of the link between self-awareness and emotion is that people can sometimes regulate their emotions by managing their self-awareness. Self-awareness prolongs and intensifies people's emotional states (Bryant & Veroff, 2007; Silvia, 2002). People who have experienced undesired events often find it difficult to stop thinking about them and their self-relevant implications, even though doing so usually makes them feel worse (Kross & Ayduk, 2008). In fact, trying not to think about disturbing events can increase the frequency of such thoughts and lead to a mood that is the opposite of the one intended (Wegner, Erber, & Zanakos, 1993). Therefore, rather than trying to stop thinking about themselves by force of will, people sometimes try to reduce their self-awareness more generally, thereby diminishing any unpleasant feelings that are sustained by self-thought. Evidence suggests that people sometimes try to attenuate their negative emotions by engaging in activities that lower their self-awareness (Baumeister, 1991). Although some of these tactics can be problematic (e.g., frequent substance abuse), others (e.g., mindfulness and meditation practices) can help people respond to negative events with greater equanimity.

One common way that people attenuate self-awareness is through distraction. People report that they watch television, socialize, read, engage in hobbies, exercise, shop, and distract themselves in other ways to take their mind off of unpleasant events. Research suggests that such distraction can, in fact, reduce negative feelings (Joormann, Siemer, & Gotlib, 2007; Kross & Ayduk, 2008). The effects of distraction on emotion may be mediated by a number of processes (e.g., many distracting activities are inherently pleasurable), but the reduction of self-thought is likely involved. Not only does thinking about one's experiences help to maintain one's emotional reactions to them, but self-awareness also intensifies affective states (Field, Joudy, & Hart, 2010; Scheier & Carver, 1977). Therefore, anything that lowers self-awareness should reduce emotion.

People also try to escape self-awareness and the aversive emotions that it can elicit in ways that may be maladaptive, for example, using alcohol and drugs, binge eating, and even masochism (Baumeister, 1991). Not all of the ways that people try to escape self-awareness are maladaptive, however. For example, people have used meditation to reduce their internal self-talk since ancient times. Although meditative practices are often promoted within spiritual traditions, their primary function is to reduce the amount of time that the practitioner engages in self-reflection (Leary, 2004). By taming what Hindus call the "chattering monkey mind," people are less likely to respond emotionally to self-related thoughts and more likely to be relaxed and present-focused. Indeed, clinical and counseling psychologists have successfully reduced problematic emotions in their clients by helping them to quiet their negative thoughts and to focus on the present moment, with practices that include features of mindfulness or meditation (e.g., acceptance and commitment therapy: Hayes, Strosahl, & Wilson, 1999; dialectical behavior therapy: Linehan, 1993; mindfulness-based stress reduction: Kabat-Zinn, 2003).

Reflecting on the Past and the Future

Unlike most other animals, people can reflect consciously on themselves over time—that is, to remember themselves in the past and imagine themselves in the future. Through the "eyes" of their cognitive analogue-I (Jaynes, 1976; Leary, Estrada, & Allen, 2009), people are able not only to remember or imagine themselves in other situations but also to plan intentionally for the future, to consider behavioral options, to imagine retrospectively how events might have turned out differently, and to learn from their mistakes. This ability to think consciously about the past and future also leads people to react emotionally to events that exist only in their minds. Because remembered, anticipated, and fantasized experiences of the analogue-I often have the emotional potency of real events, they can similarly impact people's emotions.

Reliving the Past and Imagining the Future

Reflecting about oneself in the past and future has a strong influence on people's emotions. Indeed, we suspect that a far greater proportion of people's emotional experiences arise from thinking about themselves in the past or future than from events that are occurring at the present time, and that people often compromise the positivity of their current emotional states by unnecessarily reflecting on the past and future.

First, people often experience strong emotions by reliving past experiences in their minds. For example, ruminating about past mistakes or misfortunes can elicit sadness, regret, or guilt. Autobiographical memories involving unfulfilled desires for competence, autonomy, and relatedness seem to affect negative emotions particularly strongly (Philippe, Koestner, Lecours, Beaulieu-Pelletier, & Bois, 2011). Conversely, recalling positive memories from the past plays an important role in maintaining positive moods (Joormann et al., 2007).

Similarly, many emotions, both pleasant and unpleasant, can be triggered by self-relevant thoughts about the future. Of these, anxiety is perhaps the most common. Of course, anticipating future threats is often adaptive, because it motivates people to take steps to avoid problems or plan how to cope effectively with future threats. However, people typically imagine far more frequent and more serious threats than actually occur. And even when dreaded events do arise, anticipatory anxiety typically does little more than to cause suffering in advance. In fact, many people experience chronic anxiety because they consistently overestimate the probability of negative events occurring or the adverse consequences of those events (Marshall et al., 2007; Miranda & Mennin, 2007). Moreover, believing that negative events are likely to occur (and that positive events are not likely to occur) can produce hopelessness and depression (Joiner, 2001). Thus, the human capacity to worry about things that have not yet happened—and that might, in fact, never even happen—takes a heavy emotional toll (Roemer & Borkovec, 1993).

If anxiety about the future is so often unnecessary, if not maladaptive, then why are people so prone to experiencing anxiety? Martin (1999) speculated that self-generated anxiety about the future became prevalent only with the emergence of agriculture around 10,000 years ago. Although our prehistoric ancestors were self-aware before this time (Leary & Buttermore, 2003), their hunting–gathering–scavenging lifestyle did not evoke much rumination about the distant future. With the emergence of agriculture, however, people moved to a delayed-return lifestyle in which many important outcomes—both good and bad—lay weeks or months in the future. The agricultural revolution led people to settle in communities, accumulate possessions, and plan for planting and harvesting of crops, and, thus, to fret about future outcomes that lay months or years ahead. Modern industrialized societies are profoundly delayed-return environments in which people devote considerable effort now for uncertain outcomes in the future. As a result, we are plagued by far more self-generated anxiety than were our hunter–gatherer ancestors.

When people imagine future events, they often think about how they are likely to feel when those events occur, which requires reflection about oneself in the future. Although people's affective forecasts are often inaccurate (Wilson & Gilbert, 2003),

imagining how they will feel in the future often helps people behave in ways that lead to desired emotional outcomes (e.g., "I will regret it later if I don't do something now"). Moreover, people's expectations about how they will feel when an event occurs can influence their emotional reactions to the event when it actually transpires (Wilson & Gilbert, 2003).

How people feel when an event occurs also depends on how they think about the alternatives. Concocting imaginary scenarios about how events might have turned out differently can make people feel better or worse about their lot depending on whether the alternatives they imagine are worse or better than what actually happened. Medvec, Madey, and Gilovich's (1995) finding that Olympic bronze medal winners (i.e., third-place finishers) appeared happier than silver medal winners (i.e., second-place finishers) is a good example of this effect. Presumably, bronze medalists compare finishing third to not getting a medal at all, whereas silver medalists compare their second-place finish to the imagined alternative of winning the gold. Similarly, people may become discontent even when current circumstances are good if they think that being somewhere else would be even better. Therefore, people can regulate their emotional responses to events by managing the ways in which they construe alternative outcomes.

In addition, cognitive representations of desired future states can motivate and guide people's behaviors (Emmons, 1986; Markus & Nurius, 1986). The capacity for self-thought again enters the picture, because thinking about progress toward one's goals evokes positive emotions, while lack of progress (and particularly movement away from those goals) evokes negative emotions (Higgins, 1987; Markus & Nurius, 1986). Thus, emotions are experienced not only because the current state of affairs is favorable or unfavorable to one's well-being but also because events reflect movement toward or away from one's goals.

Predictive Control

As noted, some tactics for improving people's emotion regulation involve teaching them to remain focused on the present situation as much as possible, turning to thoughts about the past and future only as needed to deal with practical issues. People can also regulate their emotional reactions to future events by managing their expectations. People's emotional reactions to events depend in part on what they expect to happen. For instance, expected negative outcomes tend to evoke less distress than unexpected ones, and events that fail to occur as anticipated may evoke stronger reactions than if they had not been expected. Self-awareness is not necessary for animals to develop expectations or to react emotionally to fulfilled and unfulfilled expectancies (e.g., the family pet may become distressed when daily routines are disrupted). However, the ability to self-reflect allows people to manage their feelings about upcoming events by exerting deliberate "predictive control" (Rothbaum, Weisz, & Snyder, 1982) in which they tell themselves what to expect.

For example, people can use positive self-talk to allay their anxiety about future outcomes. By telling themselves that things will turn out OK, people can sometimes positively influence how they feel. Such optimism is sometimes costly, though. Sweeny and Shepperd (2010) found that after controlling for actual exam performance, students who expected higher exam scores did not feel any better before receiving feedback and actually felt worse after learning their scores.

At other times, people manage their expectations to avoid being disappointed. For example, people try to soften the blow of failures, bad news, disappointments, and other aversive events by lowering their expectations and telling themselves that bad outcomes are likely to occur. This may not be an optimal tactic for maximizing positive affect, however, because the costs of negative expectations can outweigh the benefits for many people (Golub, Gilbert, & Wilson, 2009). On average, participants felt worse when they expected a negative rather than a positive outcome; moreover, their pessimistic expectations did not serve as a buffer against negative emotions once the event occurred. However, anticipating negative outcomes appears to lower anxiety among dispositionally anxious people who use this strategy regularly (i.e., defensive pessimists; Norem & Illingworth, 1993). In fact, preventing defensive pessimists from thinking

about anticipated negative events typically increases their negative emotions. Thus, defensive pessimism may be a useful means of emotional self-regulation for some people but not for others.

Evaluation of One's Personal Characteristics

People's self-relevant beliefs and evaluations often evoke emotional responses and are also involved in emotion regulation. Human infants develop a conception of themselves slowly over the first 2 years of life (Kagan, 1998) and characterize themselves in increasingly complex and abstract ways in a developmental progression that continues into late adolescence (Harter, 2012). As their self-concepts develop, children begin to experience myriad emotions by thinking about their own characteristics and behavior. Once people develop the capacity to label, characterize, and evaluate themselves, they react emotionally to these self-representations, as well as to events and information that validate or threaten them.

Self-Evaluations and Emotion

People react negatively to events that threaten their self-views and positively to events that affirm them, even when such events have no real consequences for their well-being. Thus, by virtue of being self-aware, people become emotionally invested in whether reality conforms to their desired self-views. Two kinds of symbolic satisfactions and threats to people's self-construals have received the most attention from researchers—self-enhancement and self-verification.

People prefer to evaluate themselves positively rather than negatively, and a great deal of research has examined the ways in which people try to maintain views of being good and effective individuals. Thus, events that support a favorable view of oneself tend to evoke positive emotions, such as pride, happiness, and satisfaction; events that contradict one's favorable self-view, on the other hand, are associated with negative emotions, such as anxiety, despondency, frustration, shame, and rage (Baumeister, 1998). Because people's identities include significant others in their lives, people also feel good or bad because of the successes or failures of people with whom they are associated.

Although many theorists have assumed that people desire positive feedback about themselves because they have a need to maintain or enhance their self-esteem, self-enhancement may be more parsimoniously viewed as a way to regulate emotion. People seek positive information about themselves, because being capable, attractive, socially skilled, or likable elicits positive emotions by increasing the perceived likelihood of desired outcomes. Conversely, receiving negative self-relevant feedback typically indicates a lower likelihood of desired outcomes and a greater likelihood of undesired ones.

Sociometer theory carries this notion a step further by suggesting that people's affective reactions to self-enhancing and self-depreciating events serve an important interpersonal function by providing feedback about the degree to which they are valued and accepted by others (for a review, see Leary, 2006). Events that threaten a person's positive self-image evoke negative emotions, because they cast that person in an undesirable light in other people's eyes, thereby undermining his or her relationships with them (Leary, Tambor, Terdal, & Downs, 1995). From this perspective, the feelings that arise from positive and negative self-relevant events are part of a monitoring system that keeps people apprised of their relational value and motivates behaviors to help them avoid social rejection.

Several perspectives suggest that people want to maintain not only a positive view of themselves but also a stable and consistent self-view (Swann & Buhrmester, 2012). As a result, information suggesting that people are not who or what they think they are evokes negative feelings. Cognitive dissonance theory was among the first perspectives to describe the emotional effects of having inconsistent beliefs. Any pair of contradictory beliefs can potentially induce the unpleasant affective state of dissonance, but inconsistent beliefs involving aspects of oneself seem to be particularly unpleasant (Aronson, 1968).

Swann and his colleagues have specifically examined people's responses to events that verify or disconfirm their self-views (for a review, see Swann & Buhrmester, 2012). People tend to be troubled by information

that discredits their self-perceptions, presumably because such information undermines their certainty in their beliefs about themselves and lowers their sense of predictability and control (Swann, Stein-Seroussi, & Giesler, 1992). As a result, receiving information that is inconsistent with one's self-concept is often troubling, even if it is favorable (Kwang & Swann, 2010).

Although self-verification and self-enhancement have often been pitted against one another as theoretical perspectives on motivated self-cognition, evidence shows that both processes occur, and research has begun to uncover the conditions under which each effect is obtained (Kwang, & Swann, 2010). Importantly, the possibility exists that both processes serve the goal of promoting one's social acceptance. That is, people often increase their chances of social acceptance by either portraying themselves in positive ways or seeking to interact with people who see them as they see themselves.

Regulating Emotion by Managing Self-Evaluations

Because self-evaluations influence emotions, people can regulate their emotions by managing how they perceive and evaluate themselves. Two of the most widely researched tactics for doing so involve social comparison and attribution.

Social Comparison

People's self-evaluations—and their emotions—are affected by how they compare to others, particularly in domains where no objective standard for self-evaluation exists (e.g., judgments of physical attractiveness or popularity). Therefore, people sometimes regulate their emotions through their choices of social comparison targets (Suls & Wheeler, 2012). Although people sometimes use comparison targets that provide an accurate indication of their own attributes, their selections more often appear designed to promote positive feelings (Buunk, Collins, Taylor, VanYperen, & Dakof, 1990; Morrell et al., 2012). At times, people seek downward comparisons to feel better about themselves by contrast; at other times, they seek upward comparisons for inspiration or hope.

Theorists once assumed that comparing ourselves to those who are worse off than we are (i.e., downward social comparison) generally leads to more positive emotions than comparing ourselves to people who are better off (upward social comparison). However, whether downward or upward comparisons lead to positive or negative affect depends on how people interpret the comparative information. When downward comparisons make people feel better or more fortunate than others, they induce positive emotions. In contrast, when downward social comparisons raise the specter that one might also fare poorly (e.g., when a newly diagnosed cancer patient compares him- or herself to a patient in later stages of the illness), they lead to negative emotions (Buunk et al., 1990; Morrell et al., 2012). Similarly, comparing oneself to outstanding role models evokes positive affect if the superstar's accomplishments seem personally attainable but negative affect when they do not (Lockwood & Kunda, 1997). The effects of upward and downward social comparison are quite complex and are also moderated by variables such as self-esteem, mood, neuroticism, and optimism (Lyubomirsky & Ross, 1997). Nevertheless, people often choose and interpret social comparisons in ways that help them regulate their emotions.

Attributions

In the same vein, people can regulate their emotions through their attributions for events that occur. People's beliefs about why particular emotion-inducing events occurred in the first place (e.g., "Why was I passed over for the promotion?") and why they reacted as they did to those events (e.g., "Why did I lose my temper?") influence both their self-evaluations and their emotions. Since Schachter and Singer's (1962) initial demonstration that people's interpretations of arousing events affect their emotional experiences, research has shown that attributions have a strong effect on emotion. The same event can evoke drastically different emotions depending on the person's attributions for it (Siemer, Mauss, & Gross, 2007), and such attributions necessarily involve self-focused thought.

Weiner's (1985) attribution theory of emotion most fully describes the relation-

ship between patterns of attributions and specific emotions. From this perspective, people's emotional responses to positive and negative events depend on three aspects of the attributions that they make—*causal locus* (whether the cause of the event was internal or external to the individual), *stability* (whether the cause is short-lived or long-lasting), and *controllability* (whether the cause is under the person's control). For example, if people attribute success to their own efforts (an internal, stable, and controllable cause), they experience positive self-agency emotions such as pride, satisfaction, and confidence, whereas attributing that same success to other people (an external, stable, uncontrollable cause) results in positive other-agency emotions such as gratitude, obligation, and appreciation toward those who helped. And, if the success is attributed to impersonal circumstances (an external, unstable, and uncontrollable causes), people feel lucky and happily surprised (positive situation-agency emotions).

The attribution of agency has proven to be particularly important in distinguishing among negative emotions, such as anger (other-agency), guilt (self-agency), and sorrow (circumstance-agency; Ellsworth & Smith, 1988). Additionally, agency attributions, regardless of their accuracy, influence people's appraisals of their ability to deal with negative events and their consequences (Ellsworth & Scherer, 2003). The emotional effects of attributions are also mediated by people's appraisals of the implications of those attributions for their well-being (David, Schnur, & Belloiu, 2002).

Research on both student and clinical samples demonstrates that people who characteristically explain negative events in terms of internal, stable, and global causes (i.e., a pessimistic explanatory style) are more likely to develop symptoms of depression (in particular, the subtype "hopeless depression") than those who tend to make external, unstable, and specific attributions for negative outcomes (Abramson, Metalsky, & Alloy, 1989; Alloy et al., 2000). In addition, attributing positive events to unstable and specific sources is associated with depression, albeit less strongly than attributions for negative events (Ahrens & Haaga, 1993). In fact, negative attributional styles contribute to a variety of common emotional difficulties, including depression, anxiety, and frequent anger and guilt (Hilt, 2004).

In light of the link between people's attributions and their emotions, interventions and psychotherapy techniques have been developed that focus on attributional reframing to ameliorate emotional distress. Such approaches involve teaching people to identify and challenge their inaccurate causal attributions and to develop more accurate, positive ones. For example, when people engage in excessive self-blame about negative events (i.e., unrealistically attributing negative events to a stable internal factor), an approach known as attributional retraining has been used with beneficial effects on depression, anxiety, and guilt. *Attributional retraining* involves reviewing the negative event that led to the attribution, searching for alternative explanations that are at least equally plausible, and alleviating some or all of the person's responsibility for the negative outcome. Conversely, attributional retraining can also involve fostering internal attributions for positive outcomes when appropriate, such as learning to attribute one's successes to effort and skill rather than luck, in order to increase positive emotions and self-efficacy (Hilt, 2004).

Techniques to help clients reframe their attributions are often used within the context of cognitive-behavioral therapy, which makes it difficult to assess their effectiveness as a unique mechanism for change. Even so, research generally supports the efficacy of attributional reframing for reducing emotional distress (Hilt, 2004). For example, decreased negative attributions in couples mediated increases in relationship satisfaction (Hrapczynski, Epstein, Werlinich, & LaTaillade, 2012), and a reduction in patients' negative inferences lowered their depression and anxiety (DeFronzo Dobkin et al., 2007). In addition, an integral component of treatment for panic disorder involves modifying patients' attributions for their physiological symptoms of anxiety (e.g., learning that they are not harmful) in order to reduce anxiety and the frequency of panic attacks.

Attributional retraining has also been used successfully in nonclinical settings, such as schools and the workplace, as both a method for preventing emotional distress and an intervention for emotional problems.

For instance, a study that focused on changing employees' negative attributional styles resulted in significant increases in job satisfaction and subjective well-being (Proudfoot, Corr, Guest, & Dunn, 2009). Overall, research suggests that attributional reframing can be an effective technique for improving emotional outcomes in both clinical and nonclinical populations. And, again, all of these effects are mediated by people's self-relevant thoughts.

Awareness of Other People's Perceptions and Evaluations

A great deal of human emotion arises from people's beliefs about how they are perceived and evaluated by other people. Other people's judgments of us have important implications for our social, occupational, romantic, financial, and other outcomes, so we understandably react differently when we are viewed favorably rather than unfavorably by other people. Self-awareness is needed to infer other people's thoughts, and some theorists have even proposed that the ability to reflect on one's private experiences evolved to help people infer what others might be thinking about them (Gallup, 1997). In fact, in young children the ability to imagine other people's perspectives emerges at about the same time as self-awareness (Focquaert, Braeckman, & Platek, 2008).

Self-Awareness and Social Emotions

Around this time, children also begin to experience self-conscious emotions—such as embarrassment, pride, shame, and guilt—in response to the real or imagined evaluations of other people (Miller, 1996). This particular subset of emotions is often labeled "self-conscious" because these emotions typically involve acute feelings of self-consciousness, as people think a great deal about themselves and what other people might be thinking about them (Tangney & Tracy, 2012). Yet the central feature of these emotions is that they occur when people believe that others have formed, or might form, particular positive or negative impressions of them. These emotions inherently involve reactions to social-evaluative events or perceived violations of social standards (see Dickerson, Grunewald, & Kemeny, 2004), even if those standards have been internalized and people are no longer consciously aware of their origin (Baldwin & Baccus, 2004).

People's perceptions of how others view and evaluate them (often called *reflected appraisals*) can elicit a variety of emotions. For example, people experience social anxiety when they think that others will not form desired impressions of them, even if they know that they possess positive attributes. Research has shown that people who score high in social anxiety tend to be highly self-focused and think about themselves negatively during social interactions. In fact, negative self-relevant imagery is causally involved in both trait social anxiety and the experience of state social anxiety in people who are not typically anxious (Makkar & Grisham, 2011). Similarly, people become embarrassed when they believe that other people have formed undesired impressions of them (Miller, 1996) and experience hurt feelings when they think that others do not sufficiently value them (Leary, Springer, Negel, Ansell, & Evans, 1998). In each of these cases, self-reflection is needed to infer what others might be thinking and to elicit the emotional response.

Guilt and shame also involve concerns about other people's evaluations. *Guilt* involves a negative evaluation of a specific behavior, whereas *shame* involves a negative evaluation of oneself (Tangney & Tracy, 2012). People often feel guilty or ashamed simply from knowing that others regard their behavior or character unfavorably, even if they personally believe that they did nothing wrong. Of course, people also experience positive emotions from imagining other people's reactions to them. People may experience pride when they think that others admire them, and joy when they think that others love them deeply, even in the absence of explicit indications of admiration or love.

Furthermore, people's beliefs about how others expect them to feel also affect their emotions. For example, when people feel sad but believe that others think they should be happy, they experience stronger negative emotions and lower subjective well-being. In fact, perceived social expectancies predict people's subsequent emotions and well-being more consistently than—and independently of—their personal expectancies. Research

has shown this is the case because perceived social expectancies (e.g., to be happy) promote negative self-evaluations in people when their emotions (e.g., sadness) violate these expectations (Bastian et al., 2012).

Regulating Social Emotions

Given the role of self-relevant thought in social emotions, another way that people can regulate their emotions is by managing their thoughts about what others might be thinking about them. Such strategies fall into two broad categories.

First, people sometimes reduce undesired social emotions by reevaluating the importance of other people's impressions and evaluations of them. Although people tend to react rather automatically to indications that others are judging them unfavorably, they may later decide that others' judgments are not actually important or consequential in a particular instance (Leary, 2006). Reducing the motivation to make a desired impression or the importance of being evaluated in a particular way can attenuate or minimize emotions such as social anxiety, embarrassment, guilt, and hurt feelings.

Second, to the extent that unpleasant social emotions arise from believing that one is being viewed unfavorably, people can reevaluate others' likely evaluations of them. For example, people who frequently experience social anxiety can be taught that other people's evaluations of them are usually not as negative as they imagine. Similarly, people whose feelings are easily hurt can learn that others' seemingly hurtful actions—such as minor slights, snubs, and signs of disinterest—are usually more benign than they might first appear.

Conclusion

Our focus in this chapter has been on the ways in which the human ability to self-reflect affects emotional experience and regulation. As we have seen, self-awareness allows people to experience emotions by comparing themselves to their personal standards, thinking about themselves in the past and future, assessing their personal characteristics, and thinking about how they are perceived by other people. By virtue of being self-aware, human beings live in an emotional world that differs greatly from that of other animals. Indeed, it is difficult to imagine what human subjective experience would be like without self-awareness and the internal self-talk that underlies so much human emotion.

Self-awareness was perhaps the pivotal psychological adaptation that put human beings on such a distinctly different path than that of other animals. From the standpoint of emotion, however, the capacity to think consciously about oneself is both a blessing and a curse (Leary, 2004). Self-awareness and self-relevant thought are beneficial in planning for the future in ways that maximize gains and minimize losses, and the emotions that arise when people look forward and backward are often useful in informing judicious decisions and effective behavior. Yet, as we have seen, people suffer far more from temporal emotions such as anxiety and regret than is necessary for successful self-regulation. Similarly, people's ability to compare themselves to standards and evaluate themselves accordingly provides important input into their behavioral choices. But people are often distressed by unfavorable self-evaluations even when they do not matter in a practical sense. Likewise, people could not interact successfully with others without an ability to infer what others are thinking about them, and the self-conscious emotions that sometimes arise are often beneficial in steering their behavior in ways that are socially advantageous. However, people are frequently more concerned about others' evaluations than they need to be, resulting in unnecessary social anxiety, embarrassment, guilt, and shame that can sometimes interfere with effective behavior and self-regulation.

People obviously prefer to feel positive emotions more than negative emotions, and they can change how they feel by changing how they think about themselves. As we have seen, people regulate their emotions in many ways, including attenuating self-awareness, predictive control, social comparison, and attributional reframing, among others. But even this hedonically desirable ability has both good and bad effects. Minimizing unwanted emotions certainly promotes subjective well-being, and managing one's emotions often facilitates effective behavior

and better self-regulation. However, when unpleasant emotions serve an important purpose in keeping people attuned to threats to their well-being or motivating them to engage in needed behavioral change, the use of self-talk to dampen such emotions might be counterproductive. We hope that future research will result in a better understanding of the conditions under which regulating emotions via self-relevant thought is beneficial rather than detrimental to people's well-being.

Note

1. Although our focus is on the implications of self-awareness for emotion, we should note that affective experiences can also influence the operation of self-related processes. For example, emotions can affect people's self-perceptions, self-efficacy, and ability to self-regulate (DeSteno & Salovey, 1997; Leith & Baumeister, 1996). Emotional experiences also influence where people focus their attention (Fredrickson & Branigan, 2005) and how they make decisions (Kugler, Connolly, & Ordóñez, 2012). Thus, not only is self-awareness intimately involved in the experience and regulation of emotion, but emotions also reciprocally affect self-related attention and thought.

References

Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., et al. (2000). The Temple–Wisconsin Cognitive Vulnerability to Depression (CVD) Project: Lifetime history of Axis I psychopathology in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology, 109*, 403–418.

Abramson, L. Y., Metalsky, F. I., & Alloy, L. B. (1989). Hopelessness depression: A theory based subtype of depression. *Psychological Review, 96*, 358–372.

Ahrens, A. H., & Haaga, D. A. F. (1993). The specificity of attributional style and expectations to positive and negative affectivity, depression, and anxiety. *Cognitive Therapy and Research, 17*, 83–98.

Aronson, E. (1968). Dissonance theory: Progress and problems. In R. P. Abelson, E. Aronson, W. J. McGuire, T. M. Newcomb, M. J. Rosenberg, & P. H. Tannenbaum (Eds.), *Theories of cognitive consistency: A sourcebook* (pp. 5–27). Chicago: Rand McNally.

Baldwin, M. W., & Baccus, J. R. (2004). Maintaining a focus on the social goals underlying self-conscious emotions. *Psychological Inquiry, 15*, 139–144.

Bargh, J. A., Gollwitzer, P. M., & Lee-Chai, A. (2001). The automated will: Nonconscious activation and pursuit of behavioral goals. *Journal of Personality and Social Psychology, 81*, 1014–1027.

Bastian, B., Kuppens, P., Hornsey, M. J., Park, J., Koval, P., & Uchida, Y. (2012). Feeling bad about being sad: The role of social expectancies in amplifying negative mood. *Emotion, 12*, 69–80.

Baumeister, R. F. (1991). *Escaping the self.* New York: Basic Books.

Baumeister, R. F. (1998). The self. In D. T. Gilbert, S. T. Fiske, & G. Lindzey (Eds.), *Handbook of social psychology* (4th ed., Vol. 2, pp. 680–740). Boston: McGraw-Hill.

Brown, C. M., & McConnell, A. R. (2011). Discrepancy-based and anticipated emotions in behavioral self-regulation. *Emotion, 11*, 1091–1095.

Buunk, B. P., Collins, R. L., Taylor, S. E., VanYperen, N. W., & Dakof, G. A. (1990). The affective consequences of social comparison: Either direction has its ups and downs. *Journal of Personality and Social Psychology, 59*, 1238–1249.

Bryant, F. B., & Veroff, J. (2007). *Savoring: A new model of positive experience.* Mahwah, NJ: Erlbaum.

Carver, C. S., Lawrence, J. W., & Scheier, M. F. (1999). Self-discrepancies and affect: Incorporating the role of fear selves. *Personality and Social Psychology Bulletin, 25*, 783–792.

Carver, C. S., & Scheier, M. F. (1981). *Attention and self-regulation: A control theory approach to human behavior.* New York: Springer-Verlag.

David, D., Schnur, J., & Belloiu, A. (2002). Another search for the "hot" cognitions: Appraisal, irrational beliefs, attributions, and their relation to emotion. *Journal of Rational-Emotive and Cognitive-Behavior Therapy, 20*, 93–132.

DeFronzo Dobkin, R., Allen, L. A., Alloy, L. B., Menza, M., Gara, M. A., & Panzarella, C.

(2007). Adaptive inferential feedback partner training for depression: A pilot study. *Cognitive and Behavioral Practice, 14*(4), 350–363.

DeSteno, D. A., & Salovey, P. (1997). The effects of mood on the structure of the self-concept. *Cognition and Emotion, 11*, 351–372.

Dickerson, S. S., Grunewald, T. L., & Kemeny, M. E. (2004). When the social self is threatened: Shame, physiology, and health. *Journal of Personality, 72*, 1189–1216.

Duval, S., & Wicklund, R. A. (1972). *A theory of objective self awareness*. New York: Academic Press.

Ellsworth, P. C., & Scherer, K. R. (2003). Appraisal processes in emotion. In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 575–595). New York: Oxford University Press.

Ellsworth, P. C., & Smith, C. A. (1988a). From appraisal to emotion: Differentiating among unpleasant feelings. *Motivation and Emotion, 12*, 271–302.

Emmons, R. A. (1986). Personal strivings: An approach to personality and subjective well-being. *Journal of Personality and Social Psychology, 51*, 1058–1068.

Field, N. P., Joudy, R., & Hart, D. (2010). The moderating effect of self-concept valence on the relationship between self-focused attention and mood: An experience sampling study. *Journal of Research in Personality, 44*, 70–77.

Foa, E. B., Huppert, J. D., & Cahill, S. P. (2006). Emotional processing theory: An update. In B. O. Rothbaum (Ed.), *Pathological anxiety: Emotional processing in etiology and treatment* (pp. 3–24). New York: Guilford Press.

Focquaert, F., Braeckman, J., & Platek, S. M. (2008). An evolutionary cognitive neuroscience perspective on human self-awareness and theory of mind. *Philosophical Psychology, 21*, 47–68.

Fredrickson, B. L., & Branigan, C. (2005). Positive emotions broaden the scope of attention and thought–action repertoires. *Cognition and Emotion, 19*, 313–332.

Fromson, P. M. (2006). Self-discrepancies and negative affect: The moderating roles of private and public self-consciousness. *Social Behavior and Personality, 34*, 333–350.

Frijda, N. H. (1986). *The emotions*. London: Cambridge University Press.

Gallup, G. G., Jr. (1997). On the rise and fall of self-conception in primates. In J. G. Snodgrass & R. L. Thompson (Eds.), *The self across psychology* (pp. 78–82). New York: New York Academy of Sciences.

Golub, S. A., Gilbert, D. T., & Wilson, T. D. (2009). Anticipating one's troubles: The costs and benefits of negative expectations. *Emotion, 9*, 277–281.

Harter, S. (2012). *The construction of the self: A developmental perspective* (2nd ed.). New York: Guilford Press.

Hayes, S. C., Strosahl, K., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York: Guilford Press.

Higgins, E. T. (1987). Self-discrepancy: A theory relating self and affect. *Psychological Review, 94*, 319–340.

Hilt, L. M. (2004). Attribution retraining for therapeutic change: Theory, practice, and future directions. *Imagination, Cognition and Personality, 23*(4), 289–307.

Hrapczynski, K. M., Epstein, N. B., Werlinich, C. A., & LaTaillade, J. J. (2012). Changes in negative attributions during couple therapy for abusive behavior: Relations to changes in satisfaction and behavior. *Journal of Marital and Family Therapy, 38*(1), 117–132.

Jaynes, J. (1976). *The origin of consciousness in the breakdown of the bicameral mind*. Boston: Houghton-Mifflin.

Joiner, T. E. (2001). Negative attributional style, hopelessness depression and endogenous depression. *Behaviour Therapy and Research, 39*, 139–149.

Joormann, J., Siemer, M., & Gotlib, I. H. (2007). Mood regulation in depression: Differential effects of distraction and recall of happy memories on sad mood. *Journal of Abnormal Psychology, 116*, 484–490.

Kabat-Zinn, J. (2003). Mindfulness-based stress reduction (MBSR). *Constructivism in the Human Sciences, 8*, 73–107.

Kagan, J. (1998). Is there a self in infancy? In M. Ferrari & R. J. Sternberg (Eds.), *Self-awareness: Its nature and development* (pp. 137–147). New York: Guilford Press.

Kross, E., & Ayduk, O. (2008). Facilitating adaptive emotional analysis: Distinguishing distanced-analysis of depressive experiences from immersed-analysis and distraction. *Personality and Social Psychology Bulletin, 34*, 924–938.

Kugler, T., Connolly, T., & Ordóñez, L. D.

(2012). Emotion, decision, and risk: Betting on gambles versus betting on people. *Journal of Behavioral Decision Making, 25*, 123–134.

Kwang, T., & Swann, W. B. (2010). Do people embrace praise even when they feel unworthy?: A review of critical tests of self-enhancement versus self-verification. *Personality and Social Psychology Review, 14*, 263–280.

Leary, M. R. (2004). *The curse of the self: Self-awareness, egotism, and the quality of human life*. New York: Oxford University Press.

Leary, M. R. (2006). Sociometer theory and the pursuit of relational value: Getting to the root of self-esteem. *European Review of Social Psychology, 16*, 75–111.

Leary, M. R., & Buttermore, N. E. (2003). Evolution of the human self: Tracing the natural history of self-awareness. *Journal for the Theory of Social Behaviour, 33*, 365–404.

Leary, M. R., Estrada, M. J., & Allen, A. B. (2009). The analogue-I and the analogue-me: The avatars of the self. *Self and Identity, 8*, 147–161.

Leary, M. R., Springer, C., Negel, L., Ansell, E., & Evans, K. (1998). The causes, phenomenology, and consequences of hurt feelings. *Journal of Personality and Social Psychology, 74*, 1225–1237.

Leary, M. R., Tambor, E. S., Terdal, S. K., & Downs, D. L. (1995). Self-esteem as an interpersonal monitor: The sociometer hypothesis. *Journal of Personality and Social Psychology, 68*, 518–530.

Leith, K. P., & Baumeister, R. F. (1996). Why do bad moods increase self-defeating behavior?: Emotion, risk-taking, and self-regulation. *Journal of Personality and Social Psychology, 71*, 1250–1267.

Linehan, M. M. (1993). *Cognitive behavioral treatment of borderline personality disorder*. New York: Guilford Press.

Lockwood, P., & Kunda, Z. (1997). Superstars and me: Predicting the impact of role models on the self. *Journal of Personality and Social Psychology, 73*, 91–103.

Lyubomirsky, S., & Ross, L. (1997). Hedonic consequences of social comparison: A contrast of happy and unhappy people. *Journal of Personality and Social Psychology, 73*, 1141–1157.

Macrae, C. N., Bodenhausen, G. V., & Milne, A. B. (1998). Saying no to unwanted thoughts: Self-focus and the regulation of mental life. *Journal of Personality and Social Psychology, 75*, 578–589.

Makkar, S. R., & Grisham, J. R. (2011). Social anxiety and the effects of negative self-imagery on emotion, cognition, and post-event processing. *Behaviour Research and Therapy, 49*, 654–664.

Markus, H., & Nurius, P. (1986). Possible selves. *American Psychologist, 41*, 954–969.

Marshall, R. D., Bryant, R. A., Amsel, L., Suh, E., Cook, J. M., & Neria, Y. (2007). The psychology of ongoing threat: Relative risk appraisal, the September 11 attacks, and terrorism-related fears. *American Psychologist, 62*, 304–316.

Martin, L. (1999). I-D compensation theory: Some implications of trying to satisfy immediate-return needs in a delayed-return culture. *Psychological Inquiry, 10*, 195–208.

Medvec, V. H., Madey, S. E., & Gilovich, T. (1995). When less is more: Counterfactual thinking and satisfaction among Olympic medalists. *Journal of Personality and Social Psychology, 69*, 603–610.

Miller, R. S. (1996). *Embarrassment: Poise and peril in everyday life*. New York: Guilford Press.

Miranda, R., & Mennin, D. S. (2007). *Cognitive Therapy and Research, 31*, 71–82.

Morrell, B., Jordens, C. C., Kerridge, I. H., Harnett, P., Hobbs, K., & Mason, C. (2012). The perils of a vanishing cohort: A study of social comparisons by women with advanced ovarian cancer. *Psycho-Oncology, 21*, 382–391.

Norem, J. K., & Illingworth, K. S. S. (1993). Strategy-dependent effects of reflecting on self and tasks: Some implications of optimism and defensive pessimism. *Journal of Personality and Social Psychology, 65*, 822–835.

Petrocelli, J. V., & Smith, E. R. (2005). Who I am, who we are, and why: Links between emotions and causal attributions for self- and group discrepancies. *Personality and Social Psychology Bulletin, 31*, 1628–1642.

Phillips, A. G., & Silvia, P. J. (2005). Self-awareness and the emotional consequences of self-discrepancies. *Personality and Social Psychology Bulletin, 31*, 703–713.

Phillips, A. G., & Silvia, P. J. (2010). Individual differences in self-discrepancies and emotional experience: Do distinct discrepancies predict distinct emotions? *Personality and Individual Differences, 49*, 148–151.

Phillips, A. G., Silvia, P. J., & Paradise, M. J. (2007). The undesired self and emotional experience: A latent variable analysis. *Journal of Social and Clinical Psychology, 26*, 1035–1047.

Philippe, F. L., Koestner, R., Lecours, S., Beaulieu-Pelletier, G., & Bois, K. (2011). The role of autobiographical memory networks in the experience of negative emotions: How our remembered past elicits our current feelings. *Emotion, 11*, 1279–1290.

Proudfoot, J. G., Corr, P. J., Guest, D. E., & Dunn, G. (2009). Cognitive-behavioural training to change attributional style improves employee well-being, job satisfaction, productivity, and turnover. *Personality and Individual Differences, 46*(2), 147–153.

Roemer, L., & Borkovec, T. D. (1993). Worry: Unwanted cognitive activity that controls unwanted somatic experience. In D. M. Wegner & J. W. Pennebaker (Eds.), *Handbook of mental control* (pp. 220–238). Englewood Cliffs, NJ: Prentice Hall.

Rothbaum, F., Weisz, J. R., & Snyder, S. S. (1982). Changing the world and changing the self: A two-process model of perceived control. *Journal of Personality and Social Psychology, 42*, 5–37.

Schachter, S., & Singer, J. E. (1962). Cognitive, social, and physiological determinants of emotional state. *Psychological Review, 69*, 379–399.

Scheier, M. F., & Carver, C. S. (1977). Self-focused attention and the experience of emotion: Attraction, repulsion, elation, and depression. *Journal of Personality and Social Psychology, 35*, 625–636.

Siemer, M., Mauss, I., & Gross, J. J. (2007). Same situation—different emotions: How appraisals shape our emotions. *Emotion, 7*, 592–600.

Silvia, P. J. (2002). Self-awareness and emotional intensity. *Cognition and Emotion, 16*, 195–216.

Scheier, M. F., Fenigstein, A., & Buss, A. H. (1974). Self-awareness and physical aggression. *Journal of Experimental Social Psychology, 10*, 264–273.

Suls, J., & Wheeler, L. (2012). Social comparison theory. In P. M. Van Lange, A. W. Kruglanski, & E. Higgins (Eds.), *Handbook of theories of social psychology* (Vol. 1, pp. 460–482). Thousand Oaks, CA: Sage.

Swann, W. B., Jr., & Buhrmester, M. D. (2012). Self-verification: The search for coherence. In M. R. Leary & J. P. Tangney (Eds.), *Handbook of self and identity* (2nd ed., pp. 405–424). New York: Guilford Press.

Swann, W. B., Jr., Stein-Seroussi, A., & Giesler, R. B. (1992). Why people self-verify. *Journal of Personality and Social Psychology, 62*, 392–401.

Sweeny, K., & Shepperd, J. A. (2010). The costs of optimism and the benefits of pessimism. *Emotion, 10*, 750–753.

Tangney, J. P., Niedenthal, P. M., Covert, M. V., & Barlow, D. H. (1998). Are shame and guilt related to distinct self-discrepancies?: A test of Higgins's (1987) hypotheses. *Journal of Personality and Social Psychology, 75*, 256–268.

Tangney, J. P., & Tracy, J. L. (2012). Self-conscious emotions. In M. Leary & J. P. Tangney (Eds.), *Handbook of self and identity* (2nd ed., pp. 446–478). New York: Guilford Press.

Veissier, I. I., Boissy, A. A., Désiré, L. L., & Greiveldinger, L. L. (2009). Animals' emotions: Studies in sheep using appraisal theories. *Animal Welfare, 18*, 347–354.

Wegner, D. M., Erber, R., & Zanakos, S. (1993). Ironic processes in the mental control of mood and mood-related thought. *Journal of Personality and Social Psychology, 65*, 1093–1104.

Weiner, B. (1985). An attributional theory of achievement motivation and emotion. *Psychological Review, 92*, 548–573.

Wilson, T. D., & Gilbert, D. T. (2003). Affective forecasting. In M. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 35, pp. 345–411). New York: Elsevier.

PART VII

PSYCHOPATHOLOGY

CHAPTER 24

Emotion Regulation in Anxiety Disorders

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The relationship between emotion regulation (ER) and anxiety disorders has received considerable attention since the publication of the first edition of this handbook. ER features more prominently in conceptual accounts of anxiety disorders, and empirical investigations that focus on the intersection of ER and anxiety have increased dramatically. It is no longer justified to assert that the role of emotion and ER is underrecognized in conceptualizations and treatments for anxiety disorders. Nor is it necessary to rely on extrapolation of findings from healthy samples to posit relationships between ER and anxiety disorders: In an exciting development, we are now able to review behavioral and neurobiological literatures that elucidate the role of ER in the development, phenomenology, and treatment of anxiety disorders.

This recent acceleration of research has generated an array of potentially important observations regarding the relationship of ER to anxiety disorders, which we aim to integrate in this chapter. One area of particular interest is the use of specific ER strategies (e.g., suppression, cognitive reappraisal) by individuals with anxiety disorders: how frequently they use certain strategies, how successful they are at implementing them, and—most importantly—how this may relate to the development and maintenance of anxiety disorder symptoms. We contend that the use of specific ER strategies by anxious individuals is best understood within the broader context of the basic emotional processing that characterizes anxiety disorders. The first section of the chapter therefore reviews evidence supporting the view that ER in anxiety disorders derives in large part from biological and psychological vulnerabilities that confer increased emotional reactivity, attentional biases toward threat and other negative information, and global tendencies to experience emotions as aversive and to engage in avoidant processing and behavior. These features of anxiety disorders influence anxious individuals' use of the specific ER strategies that constitute the focus of the latter portion of the chapter. In discussing these strategies, we consider several potential pathways to the ER difficulties reported by individuals with anxiety disorders, including overreliance on maladaptive ER, less frequent use or compromised effectiveness of adaptive ER, and dysfunction of neural systems supporting ER.

Defining Our Terms

Before we turn to our main topics, it will be useful to clarify the definitions of terms used throughout this chapter. Our definitions of

emotion and *emotion regulation* closely parallel those articulated by Gross (this volume). We consider emotions to be multimodal phenomena that involve changes in subjective experience, physiology, and action tendencies. Emotions occur in response to internal or external stimuli that are meaningful to the organism's survival, well-being, or other goals. In many situations, the subjective experience, physiological changes, or behavioral tendencies associated with an emotion increase the chances of attaining a goal (e.g., sympathetic arousal and the impulse to flee help achieve the goal of survival in a life-threatening situation). However, by definition, the emotions that characterize anxiety disorders are excessive, often causing the individual both subjective distress (in the form of intense emotions, catastrophic thoughts, and/or uncomfortable physical sensations) and interference with adaptive functioning (e.g., when patterns of extreme avoidance develop).

When using the term *emotion regulation*, we refer to processes that influence the occurrence, intensity, duration, and expression of emotion. These processes may support up-regulation or down-regulation of positive or negative emotions, and can be placed on a continuum of automatic to effortful (Gross, this volume). While all forms of ER are likely relevant to anxiety disorders, much of this chapter focuses on effortful down-regulation of negative emotion. This choice is partly based on typical anxiety disorder presentations, which are largely characterized by excessive negative emotions such as fear and anxiety. It also reflects the predominant focus of empirical work to date, because most research has focused on a limited number of effortful ER strategies.

Throughout the chapter we also refer to maladaptive and adaptive ER. By *maladaptive* we mean that an ER strategy is either unsuccessful in reducing the unwanted emotional response (as reflected by subjective report, physiological arousal, behavioral data, or brain imaging data) or associated with costs that likely outweigh any benefits of short-term reduction of acute emotion. In contrast, "adaptive" strategies promote (1) decreased subjective distress, physiological arousal, maladaptive behavior, and/or activation of emotion-generating brain regions; and (2) maintenance of abilities to pursue short- and long-term goals that are important to the individual. Effective ER likely involves a combination of selecting "good" strategies and being able to apply these methods flexibly depending on contextual demands (e.g., Kashdan & Rottenberg, 2010).

A Conceptual Framework for Examining ER in Anxiety Disorders

Consideration of the biological and psychological factors that underlie anxiety disorders provides an important framework for understanding ER in these disorders. Barlow (1988, 2002) proposed that anxiety disorders emerge out of a "triple vulnerability," wherein generalized biological vulnerabilities toward greater emotional reactivity, and a generalized psychological vulnerability arising from early developmental events that engender a sense of the world as uncontrollable or unpredictable (hence, threatening), represent an etiological diathesis toward psychopathology. When combined with life stress and learned experiences (specific vulnerabilities), symptoms associated with anxiety and related emotional disorders are likely to result. Specific diagnostic presentations (e.g., social anxiety disorder [SAD], panic disorder [PD]) are determined by learning experiences that focus the diathesis on particular contexts (e.g., social evaluation, bodily sensations; Barlow, 2002; Suárez, Bennett, Goldstein, & Barlow, 2009). In this way, the DSM anxiety diagnoses represent the endpoint of the convergence of the triple vulnerabilities. However, at the core of each specific disorder lies a common tendency to experience intense emotions that are viewed as threatening or unwanted, often prompting efforts to diminish or avoid the aversive emotional experience (Barlow, 1988, 2002).

Various lines of research lend support to the different components of this model. Neurobiological findings are broadly consistent with the notion of a generalized biological vulnerability to increased emotional reactivity in anxiety and related emotional disorders. Specific genetic polymorphisms have been associated with anxiety-related traits (e.g., Montag, Fiebach, Kirsch, &

Reuter, 2011) and shown to interact with stressors to predict anxiety sensitivity (an established risk factor for PD; Stein, Schork, & Gelernter, 2008) and onset of symptoms of emotional disorders (e.g., Caspi et al., 2003). Genetic findings have been linked to neural models of anxiety disorders, in that associations have been observed between these polymorphisms and both activation of neural substrates of emotion generation (e.g., Drabant et al., 2012; Munafo, Brown, & Hariri, 2008) and functional connectivity between emotion-generating regions and structures implicated in their inhibitory control (Pezawas et al., 2005). Importantly, hyperactivation of limbic structures implicated in emotion generation (e.g., amygdala, insula) coupled with reduced inhibitory cortical control of these structures has been found across anxiety disorders (e.g., Etkin & Wager, 2007; Shin & Liberzon, 2010).

Empirical support for a generalized psychological vulnerability to anxiety disorders is also evident. Anxious individuals consistently display attentional biases toward negative or threatening cues, as well as difficulty disengaging from negative stimuli (Beck & Clark, 1997; MacLeod, Mathews, & Tata, 1986; Olantunji & Wolitzky-Taylor, 2009; Pineles & Mineka, 2005). These cognitive biases toward negative information have been shown to predict elevated anxiety symptoms in response to stress (e.g., MacLeod & Hagan, 1992; van den Hout, Tenney, Huygens, Merckelbach, & Kindt, 1995). Additionally, traits reflecting diminished tolerance for distress, physical sensations, and other types of discomfort (e.g., uncertainty) are associated with a range of anxiety and mood disorders (Boswell, Thompson-Holland, Farchione, & Barlow, 2013; Keough, Riccardi, Timpano, Mitchell, & Schmidt, 2010; McEvoy & Mahoney, 2012; Olatunji & Wolitzky-Taylor, 2009) and in some cases have been shown to predict onset of anxiety disorders following stress (Schmidt, Lerew, & Jackson, 1999) or to mediate change in anxiety symptoms over time (Dugas, Laugesen, & Bukowski, 2012).

Implications for Emotion Regulation in Anxiety Disorders

The underlying features of anxiety disorders described earlier (i.e., heightened emotional reactivity, hypersensitivity to threat, and tendencies to experience emotions as aversive) likely have a considerable impact on the ER of anxious individuals. These influences may occur at multiple points along the temporal continuum of ER (see Gross, this volume), beginning at the earliest stages when automatic processes related to attention and awareness are engaged. Attention and automatic responses to threat occur within milliseconds and influence further information processing almost immediately (e.g., Garner, Mogg, & Bradley, 2006). As noted earlier, an extensive literature suggests that attentional biases toward threat and other negative information are key features of anxiety disorders (Beck & Clark, 1997; MacLeod et al., 1986; Olantunji & Wolitzky-Taylor, 2009; Pineles & Mineka, 2005). Such biases in the early processing of emotional stimuli may provoke a range of subsequent responses, including behavioral efforts to dampen responding (e.g., gaze aversion; Garner et al., 2006) and attempts to escape threatening information via cognitive control (e.g., Leutgeb, Schäfer, & Schienle, 2009; Paquette et al., 2003).

According to the "hypervigilance–avoidance" model, the early attentional biases toward threat that characterize anxiety disorders are paradoxically followed by *reduced* processing of threat (e.g., via cognitive avoidance) and increased avoidant responding. This model converges with behavioral and neuroimaging findings (e.g., Bishop, Duncan, Brett, & Lawrence, 2004; Koster, Crombez, Verschuere, Van Damme, & Wiersema, 2006; see Hofmann, Ellard, & Siegle, 2012, for a review), and with the clinical consensus that cognitive and behavioral avoidance are integral to the psychopathology of anxiety disorders. Examples from clinical observation include avoidance of situational and interoceptive (i.e., bodily sensation) cues in PD; avoidance of eye contact and social interactions in SAD; avoidance of contamination through washing, doubting thoughts through checking, or intrusive images through repetitive phrases in obsessive–compulsive disorder (OCD); avoidance of trauma reminders in posttraumatic stress disorder (PTSD); and avoidance of anxious arousal and uncertainty through worry and reassurance seeking in generalized anxiety disorder (GAD).

Tendencies toward cognitive and behavioral avoidance likely have broad impacts on anxious individuals' ER. Available evidence suggests that engagement with emotionally salient stimuli generally promotes more effective ER than avoidance or disengagement. For instance, a study that employed eye-tracking demonstrated that regardless of the specific ER strategy used (cognitive reappraisal or suppression), subjects who focused visual attention more on the emotionally salient aspects of a stimulus were more likely to regulate emotions successfully (Bebko, Franconeri, Ochsner, & Chiao, 2011). In addition, simply labeling emotions (which involves awareness and acknowledgment of emotions) has been shown to reduce distress as effectively as "active" ER strategies such as cognitive reappraisal and distraction (Lieberman, Inagaki, Tabibnia, & Crockett, 2011). Anxious subjects' tendencies to avoid deeper processing of emotional stimuli may preclude them from experiencing the benefits of engagement-oriented responses to emotional stimuli.

Relationship of Anxiety Disorders to ER: General or Specific?

Thus far we have discussed anxiety as a global trait, without distinguishing among the anxiety disorders (cf. Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2013). As we move toward discussion of specific ER strategies, we will for the most part continue to refer to anxiety disorders collectively. We should, however, note that there has been particular interest in the relationship of ER to GAD (e.g., McLaughlin, Mennin, & Farach, 2007; Mennin, Heimberg, Turk, & Fresco, 2005; Mennin, McLaughlin, & Flanagan, 2009; Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006; Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). The emotion dysregulation model of GAD posits that this disorder results from a convergence of high levels of emotional reactivity and intensity, dysfunctional meta-emotion (e.g., decreased clarity and understanding of emotions), and maladaptive efforts to regulate emotions (Mennin et al., 2005; Mennin & Fresco, this volume). GAD has been conceptualized as the "basic" anxiety disorder given that its defining features (e.g., anxious apprehension) reflect processes that contribute to the manifestation of symptoms in all anxiety disorders (Barlow, 1988; Brown, Barlow, & Liebowitz, 1994). Thus, findings related to emotion dysregulation in GAD may reflect processes found in anxiety disorders more generally.

In support of this view, several analogous findings emerge when considering emotional experience and ER across specific anxiety disorders. For instance, decreased emotional clarity has been associated with symptoms of GAD, PD, PTSD, and SAD (Baker, Holloway, Thomas, Thomas, & Owens, 2004; McLaughlin et al., 2007; Tull & Roemer, 2007; Weiss et al., 2012). Decreased acceptance of emotions has been found in GAD, PD, and PTSD (McLaughlin et al., 2007; Tull & Roemer, 2007; Weiss et al., 2012), and in one study individuals with SAD were not clearly differentiated from individuals with GAD on this variable (Mennin et al., 2009). Individuals with symptoms of PTSD (e.g., Ehring & Quack, 2010; Weiss et al., 2012), GAD (Mennin et al., 2005, 2009; Salters-Pedneault et al., 2006), and SAD (Mennin et al., 2009; Turk et al., 2005) also endorse difficulties with repairing negative mood and accessing effective ER strategies when distressed (these dimensions of ER were not assessed in available studies of PD).

Recent meta-analytic work also supports the view that maladaptive ER represents a transdiagnostic feature of anxiety and related emotional disorders. An analysis of 114 studies suggested comparable relationships of ER strategies to anxiety and mood disorder symptoms, which tended to display stronger relationships to ER than did eating and substance use disorder symptoms (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Avoidance, rumination, and suppression were positively related to anxiety and depression, whereas problem solving and cognitive reappraisal were negatively associated with both types of symptoms (though the reappraisal–anxiety relationship was only marginally significant, it was similar in magnitude to the reappraisal–depression relationship). Another investigation found that a latent "cognitive ER" factor (defined by cognitive reappraisal, problem solving, suppression, and rumination) was significantly associated with symptoms of three classes of disorder: anxiety, mood, and eating disorders (Aldao & Nolen-Hoeksema, 2010). The strongest association of the cognitive ER factor was with depressive symp-

toms, followed by anxiety symptoms, then eating symptoms.

Summary

Anxiety disorders are understood to emerge from a combination of biological vulnerabilities, psychological vulnerabilities, and stress. The vulnerabilities that underlie anxiety disorders have numerous potential impacts on the ER process. At the earliest stages, hypersensitivity to threat constrains attention, thereby limiting perceptual information and increasing threat salience. This appears to set the stage for attempts to regulate away from the threat either through cognitive control strategies or behavioral strategies that limit ongoing contact with the threatening stimuli. These types of avoidant responses may preclude effective ER that derives from engagement with emotional stimuli and recognition of emotions. Available evidence suggests that these basic emotion-processing and ER tendencies are a common factor across anxiety disorders.

Investigating Use of Specific ER Strategies in Anxiety Disorders

Having provided an overview of our conceptual framework for considering ER in relation to anxiety disorders, we now turn to discussion of specific ER strategies that have been the focus of most empirical work on this topic. We have chosen to highlight three ER strategies: cognitive reappraisal, suppression, and acceptance. These strategies have been strongly represented in recent empirical work and in development of treatments for anxiety disorders. For our purposes in this chapter, we examine acceptance alongside the "straightforward" ER strategies (cognitive reappraisal and suppression), while recognizing that acceptance is perhaps more precisely characterized as an "orientation" or "attitude" toward emotions rather than a regulation strategy *per se* (i.e., by definition, acceptance involves a relinquishing of efforts to modify emotional experience). We also should note that many other ER strategies are potentially relevant to anxiety; some of these (e.g., rumination, distraction) have been more extensively studied in relation to mood disorders and are covered by Joormann and Siemer (this volume).

Suppression

Suppression is a response-focused ER strategy that occurs late in the temporal unfolding of the emotional response (Gross, this volume). Suppression can be directed specifically toward inhibition of the behavioral expression of emotion (*expressive suppression*) and/or toward the dampening of subjective feelings (*emotion suppression*). Studies of healthy subjects indicate that expressive suppression (relative to passive viewing of emotional stimuli) effectively reduces expressive behavior; however, it also increases sympathetic arousal and fails to reduce the intensity of the subjective experience of emotion (Gross & Levenson, 1993, 1997). Neuroimaging work further suggests that expressive suppression increases activation of emotion-generating brain regions (e.g., amygdala and insula; Goldin, McRae, Ramel, & Gross, 2008).

Investigations of suppression with anxiety-prone subjects report similar results. Several studies have employed biological challenges involving inhalation of carbon dioxide–enriched air, which can provoke panic symptoms. In these studies, instructions to suppress emotions did not lead to reduced subjective distress in subjects with high anxiety sensitivity (Eifert & Heffner, 2003) or in patients diagnosed with PD (Levitt, Brown, Orsillo, & Barlow, 2004). A study using a mixed anxiety and mood disorder sample also found that instructions to suppress emotions during an emotion-provoking film did not reduce the subjective experience of emotion, and were associated with increased heart rate when compared to instructions to accept emotions during the film (Campbell-Sills, Barlow, Brown, & Hofmann, 2006a). This study also measured recovery of emotional responses, and showed that subjects instructed to suppress emotions during the film reported higher levels of distress during the postfilm recovery period than did subjects who had accepted emotions. This latter finding raises the possibility that another detrimental effect of suppression could be interference with recovery of emotions following termination of an emotion-provoking stimulus.

While these results suggest that suppression is generally an ineffective ER strategy for both healthy and anxious individuals, it should be noted that suppression is likely

beneficial under certain circumstances. For example, suppression after exposure to traumatic scenes was shown to reduce both subjective distress during exposure and later intrusions (Dunn, Billotti, Murphy, & Dalgleish, 2009). It may not be the engagement of suppression *per se*, but the rigid or habitual use of this ER strategy, that is maladaptive (cf. Bonanno, Papa, Lalande, Westphal, & Coifman, 2004).

Cognitive Reappraisal

Cognitive reappraisal entails thinking about stimuli in a way that diminishes the intensity of emotions. For example, while viewing mourners at a funeral, a person using cognitive reappraisal might think that the deceased person is at peace and that the mourners will recover with time. This strategy is conceptualized as an *antecedent-focused* strategy (i.e., one that is deployed before the full-scale emotional response has occurred; Gross, this volume). Use of cognitive reappraisal to manage negative emotions is associated with a range of beneficial effects in healthy subjects. These include reduced intensity of the subjective experience of emotion (Bebko et al., 2011; Gross, 1998; Hofmann, Heering, Sawyer, & Asnaani, 2009), reduced expressive behavior (Gross, 1998), attenuation of startle (Jackson, Malmstadt, Larson, & Davidson, 2000), enhanced parasympathetic tone (Aldao & Mennin, 2012), reduced behavioral avoidance (Wolgast, Lundh, & Viborg, 2011), and reduced amygdala activation (e.g., Goldin et al., 2008; Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner, Ray, et al., 2004).

Several of these outcomes have not been measured in studies of cognitive reappraisal that included anxious subjects. However, the most frequently reported benefit of cognitive reappraisal—reduced intensity of the subjective experience of emotion—has been demonstrated in individuals high in trait anxiety (Campbell-Sills et al., 2011) and in subjects diagnosed with anxiety disorders (Aldao & Mennin, 2012; Ball, Ramsawh, Campbell-Sills, Paulus, & Stein, 2012). Available evidence suggests divergent effects of cognitive reappraisal on the physiological responses and brain function of anxious and nonanxious individuals; these findings are discussed in detail below ("Pathways to Ineffective ER in Anxiety Disorders").

Acceptance

Acceptance entails allowing oneself to experience emotions without attempting to alter or suppress them. Acceptance-based interventions promote accepting attitudes toward emotions (e.g., emotions have a natural trajectory and will dissipate if allowed to run their course) and explicitly discourage suppression. The concept of acceptance shares features with the concept of mindfulness, which promotes nonjudgmental, present-moment awareness of emotions and other internal experiences. Acceptance and mindfulness are intended to increase subjects' awareness of their emotions (including habitual impulses to suppress or avoid discomfort), which may provide them greater opportunity to respond to emotions adaptively (Roemer, Orsillo, & Salters-Pedneault, 2008; Hill & Updegraff, 2012).

Experimental studies of healthy and anxious samples have shown that instructions to accept emotions are associated with reductions in subjective distress and behavioral avoidance (Eifert & Heffner, 2003; Levitt et al., 2004; Wolgast et al., 2011). For example, studies employing carbon dioxide challenges have shown that anxious subjects who accepted emotions reported less intense fear, fewer catastrophic thoughts, and greater willingness to complete another biological challenge when compared to subjects who either suppressed emotions or received no instructions (Eifert & Heffner, 2003; Levitt et al., 2004). Another study of a mixed anxiety and mood disorder sample showed that, compared to suppression, engaging in acceptance resulted in reduced heart rate during emotion-provoking films and faster recovery of subjective distress (Campbell-Sills et al., 2006a).

Summary

Results of experiments instructing subjects to use specific ER strategies support the view that reappraisal and acceptance are generally adaptive strategies, whereas suppression is adaptive under a more restricted range of circumstances (and can often be maladaptive). The available literature (though still limited) suggests that, like healthy subjects, individuals with anxiety disorders derive benefits from cognitive reappraisal and acceptance (e.g., reduced subjective distress) and experi-

ence detrimental effects of suppression (e.g., increased physiological arousal).

Pathways to Ineffective ER in Anxiety Disorders

Individuals with anxiety disorders report wide-ranging difficulties related to emotional experience and ER (Baker et al., 2004; Ehring & Quack, 2010; McLaughlin et al., 2007; Mennin et al., 2005, 2009; Salters-Pedneault et al., 2006; Tull & Roemer, 2007; Turk et al., 2005; Weiss et al., 2012). Consideration of the literature focused on specific ER strategies such as suppression, cognitive reappraisal, and acceptance raises a number of possible explanations for these ER difficulties. In this section, we consider the following potential pathways to ineffective ER in anxiety disorders:

1. *Strategy selection.* Anxious individuals might select maladaptive strategies more often, or adaptive strategies less often, than nonanxious individuals.
2. *ER ability.* Anxious individuals may be less competent in applying ER strategies.
3. *Divergent effects of ER strategies.* The same ER strategy, applied with a similar level of competence, may have different effects for individuals with and without anxiety disorders.
4. *Neurobiological differences.* Functioning of neural substrates supporting specific ER strategies may be compromised in anxious individuals.

Strategy Selection

Neurobiology, temperament, learning history, and other factors likely interact to determine individual differences in propensities to use specific ER strategies (e.g., see, in this volume, John & Eng; Rothbart, Sheese, & Posner; and Thompson). Consistent with experimental results, questionnaire studies suggest that habitual use of reappraisal to manage emotions is associated with a range of positive outcomes, including higher levels of overall positive affect and well-being, lower levels of overall negative affect, better interpersonal functioning, and fewer symptoms of anxiety and mood disorders (Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010; Eftekhari, Zoellner, & Vigil, 2009; Gross & John, 2003). In contrast, habitual use of suppression is related to lower levels of overall positive affect and well-being; higher levels of overall negative affect; poorer interpersonal functioning; and more symptoms of anxiety, mood, and eating disorders (Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010; Gross & John, 2003). Though effects of habitual use of acceptance have not been investigated, habitual mindfulness has been associated with lower levels of daily anxiety and depression, reduced emotional reactivity to laboratory stressors, improved abilities to differentiate emotions, decreased emotional lability, and fewer self-reported ER difficulties (Arch & Craske, 2010; Hill & Updegraff, 2012).

When selecting strategies to regulate emotional experiences, there is evidence that individuals with anxiety disorders rely more on maladaptive strategies, such as suppression, and less on adaptive strategies, such as reappraisal and acceptance. For instance, studies using both survey-based and experimental methods have found that individuals with anxiety disorders utilize suppression more than do nonanxious individuals (Baker et al., 2004; Campbell-Sills et al., 2006a; Levitt et al., 2004; Ball et al., 2012). With regard to adaptive strategy use, subjects with GAD have been found to endorse less frequent use of cognitive reappraisal (Ball et al., 2012) and acceptance (Salters-Pedneault et al., 2006). Children and adolescents diagnosed with anxiety disorders also report less frequent use of reappraisal in everyday life than do nonanxious subjects (Carthy, Horesh, Apter, Edge, & Gross, 2010).

A critical issue related to ER strategy selection in anxiety disorders is the role of emotional intensity. Empirical work has now shown that for healthy controls, ER strategy preference depends on situation intensity. Engagement of reappraisal is preferred in lower intensity situations, whereas disengagement/distraction is preferred in higher intensity situations (Sheppes, Scheibe, Suri, & Gross, 2011). Due to differences in baseline emotional state, emotional reactivity, and attentional biases, the same situation may be significantly more intense for anxious than for nonanxious individuals. The more intense emotions elicited by the situation may in turn create different conditions for selection of ER strategies; for instance, facilitating a disengagement-oriented strat-

egy (e.g., suppression) in a situation where the lower intensity emotions of healthy individuals would prompt an engagement-oriented strategy (e.g., cognitive reappraisal).

A variety of other factors may contribute to the observed differences between anxious and nonanxious subjects in the selection of ER strategies. With respect to reappraisal, an expanded awareness is necessary to allow for the processing of alternative interpretations. The narrowed attention fostered by attentional biases associated with anxiety disorders may preclude processing of alternate stimulus meanings, a necessary component of successful reappraisal. Similarly, the propensity toward hypervigilance followed by avoidant processing may preclude engagement of mindfulness and acceptance, both of which require openness toward all experience, including that which is uncomfortable. By contrast, suppression and behavioral avoidance naturally emanate from avoidant processing, and may therefore be more readily recruited by individuals with anxiety disorders.

ER strategy selection also may relate to individual differences in abilities to use executive control to mitigate the impact of emotional stimuli. Recent work suggests that individuals who habitually use cognitive reappraisal may have an enhanced ability to use executive control in the service of regulating emotions (Cohen, Henik, & Moyal, 2012; Gyurak, Goodkind, Kramer, Miller, & Levenson, 2012). By contrast, individuals with anxiety disorders may have compromised abilities in this regard. This view is consistent with findings of neuroimaging studies (discussed in more detail below) that show reduced prefrontal cortex (PFC) activation in individuals with anxiety disorders using reappraisal to regulate emotions (Goldin, Manber-Ball, Werner, Heimberg, & Gross, 2009; Goldin, Manber, Hakimi, Canli, & Gross, 2009; Ball et al., 2012; New et al., 2009), and the observation that nonclinical, anxiety-prone subjects require greater PFC resources than healthy controls to down-regulate negative emotions with reappraisal (Campbell-Sills et al., 2011). Diminished abilities to recruit executive control networks early in the processing of emotional stimuli could also lead to over-reliance on suppression (a strategy deployed later in the temporal continuum of ER) to manage negative emotions.

Another factor relevant to ER strategy selection is subjects' "meta-experience" of emotion, which includes dimensions such as attitudes toward emotions, understanding of emotions, emotional clarity, and acceptability of emotions. As noted earlier, individuals with anxiety disorders exhibit higher levels of traits reflecting diminished tolerance for emotional distress and other types of discomfort, as well as decreased clarity and acceptability of emotions. While studies directly examining the relationships between meta-experiences of emotion and selection of ER strategies have been sparse, one study using a mixed anxiety and mood disorder sample showed that less accepting attitudes toward current emotions mediated the relationship between negative emotion intensity and spontaneous use of suppression (Campbell-Sills et al., 2006a). Thus, more intense negative emotion prompted subjects to engage in suppression only when the emotions were experienced as less acceptable. This finding suggests that meta-experience of emotion may influence strategy selection in individuals with anxiety disorders.

ER Ability

Individual differences in abilities to implement ER strategies undoubtedly exist and may be relevant to the ER difficulties reported by anxious subjects. However, very few studies have evaluated the levels of competence that subjects have in applying specific ER strategies. This research aim requires methods for assessing the quality of the *process* by which individuals engage in ER strategies. One exception is Carthy et al.'s (2010) analysis of cognitive reappraisal abilities demonstrated by children with and without anxiety disorders during a laboratory ER task. In this study, subjects recited their reappraisals out loud during the task (which utilized negative images) and their responses were coded as reappraisal successes or failures. While children with anxiety disorders were able to reappraise the majority of stimuli successfully (87%), they demonstrated slightly but significantly lower reappraisal abilities than healthy controls (93%). One other study reported on the process of training subjects to use reappraisal to down-regulate emotions elicited by negative images (Campbell-Sills et al., 2011). Anxious and healthy control groups did not differ in

their need for extra training in reappraisal prior to performing the experimental task. More work is needed to elucidate whether there are important differences in abilities of anxious and nonanxious individuals to apply cognitive reappraisal and other ER strategies.

Divergent Effects of ER Strategies

Even when the same ER strategy is selected and applied with a similar level of competence, its effects may vary for anxious and nonanxious individuals. In particular, the beneficial effects of adaptive strategies could be attenuated, or the negative effects of maladaptive strategies could be amplified, leading to greater ER difficulties in individuals with anxiety disorders.

Two studies suggest that the costs of suppression may be higher for anxious individuals. In one study, the effects of suppression and no instructions were compared in subjects high and low in trait negative affect (a trait strongly associated with anxiety and mood disorders; Barlow, 2002; Brown & Barlow, 2009). Individuals with high negative affect who engaged in suppression showed a paradoxical increase in negative emotions when writing about a negative personal memory, whereas those with low negative affect did not (Dalgleish, Yiend, Schweizer, & Dunn, 2009). Another study found that instructions to suppress emotions were associated with increased subjective distress in subjects with elevated scores on a measure of experiential avoidance (a trait also associated with anxiety disorders; Feldner, Zvolensky, Eifert, & Spira, 2003). The increased sympathetic arousal found to accompany suppression may render this strategy particularly ineffective for anxiety-prone individuals, who are sensitive to and often fear physical symptoms.

Research suggests that under controlled experimental conditions (i.e., when subjects are trained to reappraise standardized emotional stimuli), anxious subjects can achieve reductions in subjective distress that are comparable to those observed in healthy subjects (Campbell-Sills et al., 2011; Ball et al., 2012). However, another recent study suggests that even when the subjective outcomes of cognitive reappraisal are equivalent, the physiological benefits of this strategy may be compromised for anxious individuals. In this study, subjects with GAD and healthy controls were assigned to cognitive reappraisal, acceptance, or passive viewing conditions (Aldao & Mennin, 2012). Their subjective experience and heart rate variability (HRV) were measured while they viewed films that elicited anxiety, sadness, and disgust. HRV, an index of parasympathetic influence on the heart, reflects the capacity of the autonomic nervous system to respond adaptively to changing environmental conditions. HRV tends to decrease under conditions of threat and to increase under conditions of flexible and adaptive regulation (Friedman, 2007).

Results revealed that subjects who used cognitive reappraisal reported lower subjective negative affect in response to the films compared to subjects who used acceptance or who engaged in passive viewing (which did not differ). This pattern was consistent across the GAD and healthy control groups. With respect to physiological effects, subjects with GAD displayed significantly lower HRV than healthy controls across all film types and regulation conditions. These findings were consistent with previous reports of low parasympathetic tone in GAD (Thayer, Friedman, & Borkovec, 1996). Moreover, during recovery from anxiety- and disgust-provoking films, subjects with GAD showed an HRV pattern that was opposite to that displayed by healthy controls. Healthy subjects who used reappraisal and acceptance showed higher HRV than those who had received no instructions, suggesting enhanced parasympathetic tone with adaptive ER. In contrast, subjects with GAD who used reappraisal and acceptance showed lower HRV than those who received no instructions. This pattern of parasympathetic response may indicate diminished efficacy of reappraisal and acceptance in GAD that is only observed at the physiological level.

Aldao and Mennin's (2012) HRV findings converge with those of another study of reappraisal in healthy individuals classified as high or low on neuroticism, a personality trait strongly related to anxiety and mood disorders (e.g., Brown, Chorpita, & Barlow, 1998; Barlow et al., 2013; Wilamowska et al., 2010). In this study (Di Simplicio et al., 2012), "low neuroticism" subjects showed the expected pattern of higher HRV when regulating emotions (vs. maintaining emo-

tions), while "high neuroticism" subjects showed no significant difference in HRV across the two conditions. These results signify low parasympathetic tone in the high neuroticism group, as well as reduced physiological benefits of cognitive reappraisal for anxiety-prone individuals.

Finally, a recent ecological study of anxious and healthy children found differences in the efficacy of ER strategies that were related to anxiety disorder status (Tan et al., 2012). This study assessed emotional reactions to negative events in the past hour, use of ER strategies (distraction, reappraisal, problem solving, acceptance, avoidance, and rumination), and current emotional experience. Interestingly, children with anxiety disorders endorsed using the various ER strategies with the same frequency as nonanxious youth. Examination of strategy use in relation to peak and current emotional intensity ratings revealed that some adaptive strategies (e.g., reappraisal) were equally effective in down-regulating subjective distress in children with and without anxiety disorders. However, the data suggested that acceptance was only effective for decreasing subjective distress in nonanxious children.

Neurobiological Differences

Another factor that may contribute to ineffective ER in anxiety disorders is dysfunction of neural systems supporting ER. The majority of research on this topic has focused on cognitive reappraisal, and these studies have revealed several potentially important neural correlates of anxious subjects' use of this strategy.

One pattern that has now been observed in participants diagnosed with several distinct anxiety disorders (GAD, SAD, PD, and PTSD) is hypoactivation of PFC regions implicated in "top-down" control of emotions during reappraisal of emotional stimuli (Ball et al., 2012; Goldin, Manber, et al., 2009; Goldin, Manber-Ball, et al., 2009; New et al., 2009). This suggests that less robust engagement of PFC may contribute to ER difficulties across anxiety disorders. One recent study (Ball et al., 2012) showed that subjects with GAD and PD who were trained to use cognitive reappraisal achieved similar reductions in subjective distress compared to healthy controls who received the same training. However, the subjects with GAD and PD displayed decreased dorsomedial and dorsolateral PFC activation while implementing this strategy. Moreover, PFC activation during reappraisal was inversely related to anxiety severity (e.g., subjects with the highest levels of anxiety severity displayed the lowest levels of left dorsolateral PFC activation), providing additional evidence of a significant relationship between anxiety and PFC function during ER.

Functional abnormalities also were observed when subjects with SAD were instructed to use cognitive reappraisal to reduce emotions elicited by personally relevant statements (Goldin, Manber-Ball, et al., 2009). In this study, potentiated responses in regions such as the medial PFC and amygdala delayed normal cognitive processing in the dorsolateral PFC, which only occurred after a temporal lag relative to that in healthy controls. One possible interpretation of this pattern is that anxious individuals must overcome an initial increased aversive response (reflected in medial PFC and amygdala hyperactivity) before implementing reappraisal (reflected in delayed dorsolateral PFC processing).

Dysfunction of neural circuitry supporting cognitive reappraisal (and other forms of ER) may be context dependent. In particular, dysfunction may be exacerbated in the presence of emotional cues that represent the focus of the individual's anxiety disorder. A study of individuals with SAD found differences in neural activation that were related to stimulus type (Goldin, Manber, et al., 2009). When regulating responses to images depicting physical threat, no differences emerged between subjects with SAD and healthy controls. However, when regulating reactions to social threat, subjects with SAD displayed reduced activation in brain regions implicated in cognitive control (dorsolateral PFC, dorsal anterior cingulate cortex), visual attention (medial cuneus, posterior cingulate), attention (bilateral dorsal parietal), and visual feature detection (bilateral fusiform, superior temporal gyrus). These findings suggest that in SAD the coordination of cognitive control circuitry may be selectively compromised during the regulation of social threat.

Few neuroimaging studies have examined anxious subjects' use of ER strategies

other than reappraisal. An exception, a recent investigation of subjects with GAD, compared the effects of acceptance, suppression, and worry on subjective and neural responses to personally relevant worry statements (Ellard, 2012). Both acceptance and suppression were associated with less subjective distress and amygdala activation than worry. However, engagement of PFC structures to meet these regulatory goals differed for the two conditions. Suppression was associated with increased activation of insula and of right lateral PFC regions implicated in working memory and the generation of inner speech, which suggested paradoxically greater engagement of brain regions implicated in somatic and linguistic representations of emotional stimuli. On the other hand, acceptance was associated with reduced insula activation and greater left dorsomedial PFC activation. The dorsomedial PFC has been implicated in representations of meta-states of self-awareness, such as making inferences about moment-to-moment feelings (Ochsner, Knierim, et al., 2004), intentions for action (Lau, Rogers, Haggard, & Passingham, 2004), and tactical response selection (Matsuzaka, Akiyamaa, Tanjib, & Mushiake, 2012). Additionally, left lateralization of PFC activation has been associated with approach motivation, whereas right lateralized activation has been associated with avoidance (Speilberg et al., 2011). Importantly, acceptance also engaged ventromedial PFC regions implicated in extinction learning, which may relate to behavioral data suggesting greater willingness to reengage with aversive stimuli following acceptance (Eifert & Heffner, 2003; Levitt et al., 2004).

Studies of functional connectivity measured during resting states also contribute to our understanding of ER in anxiety disorders. Deviations in functional connectivity between subregions of the amygdala and a broad network of somatosensory association cortices, motor regions, memory regions, and regions associated with self-referential processing have been observed in individuals with GAD (Etkin, Prater, Schatzberg, Menon, & Greicius, 2009). Increased amygdala–dorsolateral PFC connectivity was also found in patients with GAD, whereas no such functional connectivity was found in healthy controls. This finding is intriguing given the consistent implication of dorsolateral PFC in down-regulation of negative emotions via cognitive reappraisal (e.g., Goldin et al., 2008; Ochsner et al., 2002; Ochsner, Ray, et al., 2004). The greater amygdala–dorsolateral PFC coupling in GAD may represent a compensatory mechanism that functions as an attempt to dampen emotional responding through cognitive control (cf. Borkovec, Alcaine, & Behar, 2004). Such a compensatory mechanism may in fact be counterproductive for anxious individuals given the observation that worry is associated with increased amygdala activation in subjects with GAD (Ellard, 2012).

Finally, it should be noted that more complex and fine-grained analyses are needed to further elucidate the neural bases of ER difficulties in anxiety disorders. A recent pathway mapping analysis identified subcortical regions that mediated the relationship between PFC activation and reappraisal success in healthy subjects (Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008). Two distinct pathways—one through the amygdala (associated with reduced reappraisal success) and one through the nucleus accumbens and ventral striatum (associated with greater reappraisal success)—were identified. This implies that successful reappraisal may involve both the dampening of amygdala responses by the PFC and an increase in nucleus accumbens/ventral striatum activation, which may facilitate approach toward the emotion-provoking stimulus. Extrapolating to the case of ER in anxiety disorders, these results raise the possibility that dysfunction may extend beyond regions implicated in cognition and emotion to include regions implicated in processing sensory information and choosing an adaptive behavioral response. Specifically, ER difficulties across anxiety disorders may be characterized by not only deficits in cortical control of affective responses but also a failure to bring appropriate approach- and reward-related behaviors online (e.g., Etkin, Prater, Hoeft, Menon, & Schatzberg, 2010). These findings also suggest that cortical control of limbic responses represents only one aspect of widespread processing necessary in order to achieve regulatory goals through reappraisal.

Summary

The available evidence suggests that the problems with ER reported by individuals with anxiety disorders may be partly due to strategy selection, because anxious individuals appear to select the maladaptive strategy of suppression more frequently and adaptive strategies such as cognitive reappraisal and acceptance less frequently. This may be attributable to the more intense emotions experienced by anxiety-prone individuals or to their more negative meta-experiences of emotions, both of which may favor avoidant and less cognitively demanding methods for regulating emotions (e.g., suppression). Biases against use of reappraisal may relate to diminished abilities of anxious individuals to use executive control to modulate emotions, or to narrowed (i.e., threat-focused) attention that does not allow for processing of alternative appraisals. Overreliance on suppression may be particularly costly for anxious individuals given evidence that they are more susceptible to paradoxical increases in distress when using suppression to manage emotions.

More research is needed to determine whether individuals with anxiety disorders are less competent in applying adaptive strategies (e.g., cognitive reappraisal) once they have been prompted to use them. Available evidence suggests that when trained to use reappraisal, anxious and nonanxious subjects achieve similar reductions in subjective distress. However, reappraisal may be less effective in promoting enhanced parasympathetic tone in anxious individuals. The growing neuroimaging literature on ER and anxiety disorders further suggests a failure to recruit PFC effectively and efficiently during cognitive reappraisal. Dysfunction of neural substrates of ER likely constitutes another basis of the regulatory difficulties reported by individuals with anxiety disorders.

Maladaptive ER: Risk Factor or Epiphenomenon of Anxiety Disorders?

An important issue we have yet to address is the directionality of relationships between anxiety- and ER-related constructs. Although the reviewed associations between ER strategies and anxiety disorders are compelling, it is not possible to ascertain causal relationships due to the cross-sectional nature of virtually all available studies. For instance, the findings that anxious subjects use suppression more than healthy controls could be explained by habitual suppression being a risk factor for anxiety disorders. However, it is also plausible that the intense emotions that characterize anxiety disorders could overwhelm the subject to the extent that more cognitively demanding ER strategies (e.g., reappraisal) are less available and suppression becomes the chosen strategy by default.

Fortunately, two recent longitudinal studies provide data that permit discrimination between the "risk" and "epiphenomenon" hypotheses. The data so far suggest that use of maladaptive ER conveys risk for psychopathology. In a prospective study of adolescents, structural equation modeling was used to evaluate the relationship between ER (a latent factor defined by indicators of emotional understanding, dysregulated expression of sadness and anger, and ruminative responses to distress) and symptoms of anxiety, depression, aggression, and eating pathology (McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011). Adolescents were assessed at baseline and again seven months later. Analyses showed that after researchers controlled for baseline symptoms, poor ER at baseline predicted increased symptoms of anxiety, aggression, and eating pathology (but not depression) measured 7 months later. Importantly, psychopathology symptoms measured at baseline did not predict worse ER at the follow-up.

A large (N > 1,000), prospective, community-based study also investigated the relationship between use of adaptive and maladaptive ER strategies and psychopathology (Aldao & Nolen-Hoeksema, 2012). Composite ER scores were used, such that maladaptive ER reflected use of behavioral disengagement, denial, suppression, and the "brooding" facet of rumination, and adaptive ER reflected use of positive reframing (similar to reappraisal) and acceptance. The overall index of psychopathology that was used included anxiety symptoms, depressive symptoms, and alcohol use. Regression

analyses controlling for baseline symptoms showed that self-reported use of maladaptive strategies at baseline predicted global psychopathology 1 year later. The authors noted that the same results emerged when only internalizing symptoms (depression and anxiety) were considered, though results pertaining to anxiety symptoms alone were not reported. Although measurement of anxiety was not optimal in this study (composite of a single clinician rating and questionnaire that mainly assesses autonomic arousal), the results contribute to the evidence that use of maladaptive strategies is not simply an epiphenomenon or consequence of anxiety disorders or other psychopathology.

If overreliance on maladaptive ER strategies constitutes a risk factor for anxiety and related emotional disorders, it is plausible that habitual use of adaptive strategies could impart resilience to psychopathology. However, the only available longitudinal data do not support this hypothesis. In Aldao and Nolen-Hoeksema's (2012) longitudinal study, self-reported use of adaptive strategies (cognitive reappraisal and acceptance) at baseline did not significantly predict psychopathology symptoms 1 year later. Nor did the interaction of adaptive and maladaptive strategies at baseline predict psychopathology at follow-up. The lack of a significant longitudinal relationship between adaptive ER and psychopathology converges with cross-sectional research showing that maladaptive strategies have stronger associations with symptoms of anxiety and mood disorders than adaptive strategies (e.g., Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010; Moore, Zoellner, & Mollenholt, 2008). For example, a cross-sectional investigation of ER in trauma-exposed women found that expressive suppression was strongly positively associated with stress-related symptoms, whereas cognitive reappraisal was only weakly negatively associated with these symptoms (Moore et al., 2008).

Despite these negative findings, it is still possible that adaptive ER relates in important ways to anxiety disorders and other psychopathology. For example, one longitudinal study of an undergraduate sample reported divergent outcomes for individuals high in neuroticism who endorsed low and high levels of "emotional repair," a construct related to use of adaptive ER (Auerbach, Abela, & Ringo Ho, 2007). Highly neurotic individuals with low levels of emotional repair endorsed increased engagement in risky behaviors following increases in anxiety and depression symptoms. In contrast, highly neurotic individuals who endorsed high levels of emotional repair displayed decreased engagement in risky behaviors following increases in anxiety and depression. These results suggest that adaptive ER may curtail emotional responses that could lead to clinical worsening or complications.

Cross-sectional evidence also suggests that adaptive ER strategies may have a palliative effect on anxiety disorder symptoms. For example, a study of male veterans with PTSD found that the combination of higher levels of emotional clarity and more frequent use of cognitive reappraisal was associated with lower overall PTSD severity and greater positive affect (Boden, Bonn-Miller, Kashdan, Alvarez, & Gross, 2012). Another study showed that trauma-exposed undergraduates with "probable PTSD" endorsed greater difficulties related to ER such as decreased emotional acceptance and difficulties engaging in goal-directed behavior, controlling impulsive behavior, and accessing adaptive ER strategies when distressed. In contrast, trauma-exposed subjects without PTSD had fewer difficulties controlling impulsive behaviors and greater access to adaptive ER strategies when distressed (relative to both trauma-exposed subjects with probable PTSD and those without trauma exposure; Weiss et al., 2012). Aldao and Nolen-Hoeksema (2012) also reported a significant interaction effect of adaptive and maladaptive ER strategies on psychopathology symptoms at the baseline assessment of their study. Level of adaptive strategy use was negatively correlated with psychopathology for subjects endorsing high usage of maladaptive strategies, suggesting that adaptive ER mitigated the detrimental effects of maladaptive ER.

It is also possible that aspects of adaptive ER that have not yet been examined in longitudinal investigations are important to resilience to psychopathology. Although frequent use of adaptive ER strategies was not shown to predict psychopathology symptoms (Aldao & Nolen-Hoeksema, 2012), frequent *and competent* use may do so. Though cross-sectional, one community-

based study showed that cognitive reappraisal ability (measured as success in reducing subjective and physiological indicators of emotion on a standardized task) was associated with lower levels of depressive symptoms in women experiencing high levels of stress (Troy, Wilhelm, Shallcross, & Mauss, 2010).

Finally, it is noteworthy that treatment-related changes in ER are associated with reductions in anxiety disorder symptoms. For example, a recent study showed that changes in reappraisal self-efficacy mediated the effects of cognitive-behavioral therapy for SAD (Goldin et al., 2012). Initial efficacy data from several treatments for anxiety disorders that directly target ER further suggest that facilitating adaptive ER can lead to resolution of anxiety disorder symptoms. In addition to ER-focused treatments for specific anxiety disorders (e.g., emotion regulation therapy for GAD: Mennin & Fresco, this volume; acceptance-based behavior therapy for GAD: Roemer et al., 2008), our group has developed and tested a unified protocol for transdiagnostic treatment of emotional disorders, designed to reduce maladaptive emotional processing, promote increased engagement of adaptive ER, and facilitate extinction of distressing reactions to intense negative emotions (Barlow et al., 2011). Results of the initial randomized controlled trial demonstrated that this intervention led to significant reductions in anxiety focused on emotional experience, anxiety sensitivity, and emotional avoidance strategies (Ellard et al., 2011), while another study showed large treatment effect sizes across disorders (Farchione et al., 2012). Empirical support for the efficacy of the unified protocol and other ER-focused treatments for anxiety disorders reinforces the notion that ER is an important factor that may contribute to the onset and/or maintenance of anxiety disorders.

Summary

The results of two recent longitudinal investigations suggest that maladaptive ER may temporally precede and increase risk for psychopathology, including anxiety disorders, consistent with the etiological model to which we have referred in this chapter. While adaptive ER does not appear to confer protection against later anxiety or related symptoms, preliminary research suggests that adaptive ER is associated with decreased severity of anxiety disorder symptoms and may reduce clinical complications (which often result from maladaptive/avoidant ER). Interventions designed to facilitate adaptive ER also have shown promising results. More longitudinal research is needed to determine whether competent use of adaptive strategies protects individuals from developing anxiety and related emotional disorders when exposed to stressors or in the presence of other risk factors for disorder.

Conclusions and Future Directions

A dramatic increase in research over the last decade has led to important observations regarding the relationship of ER to the etiology, phenomenology, and treatment of anxiety disorders. We have attempted to synthesize these important data and to consider them within the broader context of basic features of anxiety disorders that influence ER.

Individuals with anxiety disorders report wide-ranging difficulties related to emotional experience and ER. We have posited that these difficulties emanate from underlying features of anxiety disorders that include heightened emotional reactivity, hypersensitivity to threat, and increased tendencies toward avoidant processing and behavior. These basic characteristics of anxiety disorders may contribute to problematic ER strategy selection, because the emotional processing of anxious individuals appears to facilitate maladaptive strategies, such as suppression, and impede adaptive strategies, such as cognitive reappraisal and acceptance. ER-related difficulties also may be exacerbated by differences in consequences of ER strategies for anxious individuals (e.g., negative effects of suppression may be potentiated; physiological benefits of adaptive strategies may be attenuated). Furthermore, mounting evidence of dysfunction of neural systems supporting adaptive ER suggests that this may be a key factor underlying the difficulties reported by individuals with anxiety disorders.

Despite the substantial expansion of interest and research related to ER and anxiety

disorders, it is clear that much more empirical work is needed to refine our understanding of the topics addressed in this chapter. Basic questions related to measurement of ER and construct validity remain important areas for further study and, in general, replication of many of the findings summarized in this chapter is needed. Although we believe the evidence points to maladaptive ER being a transdiagnostic feature of anxiety and related emotional disorders, more empirical work is needed to confirm this view or, alternatively, to establish the specificity of relationships among ER-related constructs and distinct anxiety symptoms and disorders. Additionally, because longitudinal studies have been sparse, more of this type of research is needed to clarify the directionality of relationships between ER- and anxiety-related variables, and to identify the most optimal ER-related targets for treatment and prevention of anxiety disorders. The recent longitudinal work suggesting that maladaptive ER contributes to risk for anxiety disorders offers further support for the view that ER is an important factor to consider in etiological models of anxiety disorders, as well as in future treatment development and prevention efforts.

References

Aldao, A., & Mennin, D. S. (2012). Paradoxical cardiovascular effects of implementing adaptive emotion regulation strategies in generalized anxiety disorder. *Behaviour Research and Therapy, 50*, 122–130.

Aldao, A., & Nolen-Hoeksema, S. (2010). Specificity of cognitive emotion regulation strategies: A transdiagnostic examination. *Behaviour Research and Therapy, 48*, 974–983.

Aldao, A., & Nolen-Hoeksema, S. (2012). When are adaptive strategies most predictive of psychopathology? *Journal of Abnormal Psychology, 121*, 276–281.

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*, 217–237.

Arch, J. J., & Craske, M. G. (2010). Laboratory stressors in clinically anxious and non-anxious individuals: The moderating role of mindfulness. *Behaviour Research and Therapy, 48*, 495–505.

Auerbach, R. P., Abela, J. R., & Ringo Ho, M. H. (2007). Responding to symptoms of depression and anxiety: Emotion regulation, neuroticism, and engagement in risky behaviors. *Behaviour Research and Therapy, 45*, 2182–2191.

Baker, R., Holloway, J., Thomas, P. W., Thomas, S., & Owens, M. (2004). Emotional processing and panic. *Behaviour Research and Therapy, 42*, 1271–1287.

Ball, T. M., Ramsawh, H. J., Campbell-Sills, L., Paulus, M. P., & Stein, M. B. (2013). Prefrontal dysfunction during emotion regulation in generalized anxiety and panic disorder. *Psychological Medicine, 43*(7), 1475–1486.

Barlow, D. H. (1988). *Anxiety and its disorders: The nature and treatment of anxiety and panic* (1st ed.). New York: Guilford Press.

Barlow, D. H. (2002). *Anxiety and its disorders: The nature and treatment of anxiety and panic* (2nd ed.). New York: Guilford Press.

Barlow, D. H., Ellard, K. K., Fairholme, C. P., Farchione, T. J., Boisseau, C. L., Allen, L. B., et al. (2011). *Unified protocol for the transdiagnostic treatment of emotional disorders: Therapist guide*. New York: Oxford University Press.

Barlow, D. H., Sauer-Zavala, S. E., Carl, J. R., Bullis, J. R., & Ellard, K. K. (in press). The nature, diagnosis, and treatment of neuroticism: Back to the future. *Clinical Psychological Science*.

Bebko, G. M., Franconeri, S. L., Ochsner, K. N., & Chiao, J. Y. (2011). Look before you regulate: Differential perceptual strategies underlying expressive suppression and cognitive reappraisal. *Emotion, 11*, 732–742.

Beck, A. T., & Clark, D. A. (1997). An information processing model of anxiety: Automatic and strategic processes. *Behaviour Research and Therapy, 35*, 49–58.

Bishop, S., Duncan, J., Brett, M., & Lawrence, A. D. (2004). Prefrontal cortical function and anxiety: Controlling attention to threat-related stimuli. *Nature Neuroscience, 7*, 184–188.

Boden, M. T., Bonn-Miller, M. O., Kashdan, T. B., Alvarez, J., & Gross, J. J. (2012). The interactive effects of emotional clarity and cognitive reappraisal in posttraumatic stress disorder. *Journal of Anxiety Disorders, 26*, 233–238.

Bonanno, G. A., Papa, A., Lalande, K., Westphal, M., & Coifman, K. (2004). The importance of being flexible: The ability to both enhance and suppress emotional expression

predicts long-term adjustment. *Psychological Science, 15*, 482–487.

Borkovec, T. D., Alcaine, O., & Behar, E. (2004). Avoidance theory of worry and generalized anxiety disorder. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 77–108). New York: Guilford Press.

Boswell, J. F., Thompson-Holland, J., Farchione, T. J., & Barlow, D. H. (2013). Intolerance of uncertainty: A common change factor in the treatment of emotional disorders. *Journal of Clinical Psychology, 69*, 630–645.

Brown, T. A., & Barlow, D. H. (2009). A proposal for a dimensional classification system based on the shared features of the DSM-IV anxiety and mood disorders: Implications for assessment and treatment. *Psychological Assessment, 21*, 256–271.

Brown, T. A., Barlow, D. H., & Liebowitz, M. (1994). The empirical basis of GAD. *American Journal of Psychiatry, 151*, 1272–1280.

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology, 107*, 179–192.

Campbell-Sills, L., Barlow, D. H., Brown, T. A., & Hofmann, S. G. (2006a). Effects of suppression and acceptance on emotional responses of individuals with anxiety and mood disorders. *Behaviour Research and Therapy, 44*, 1251–1263.

Campbell-Sills, L., Barlow, D. H., Brown, T. A., & Hofmann, S. G. (2006b). Acceptability and suppression of negative emotion in anxiety and mood disorders. *Emotion, 6*, 587–595.

Campbell-Sills, L., Simmons, A. N., Lovero, K. L., Rochlin, A. A., Paulus, M. P., & Stein, M. B. (2011). Functioning of neural systems supporting emotion regulation in anxiety-prone individuals. *NeuroImage, 54*, 689–696.

Carthy, T., Horesh, N., Apter, A., Edge, M. D., & Gross, J. J. (2010). Emotional reactivity and cognitive regulation in anxious children. *Behaviour Research and Therapy, 48*, 384–393.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science, 301*, 386–389.

Cohen, N., Henik, A., & Moyal, N. (2012). Executive control attenuates emotional effects—for high reappraisers only? *Emotion, 12*, 970–979.

Dalgleish, T., Yiend, J., Schweizer, S., & Dunn, B. D. (2009). Ironic effects of emotion suppression when recounting distressing memories. *Emotion, 9*, 744–749.

Di Simplicio, M., Costoloni, G., Western, D., Hanson, B., Taggart, P., & Harmer, C. J. (2012). Decreased heart rate variability during emotion regulation in subjects at risk for psychopathology. *Psychological Medicine, 42*, 1775–1783.

Drabant, E. M., Ramel, W., Edge, M. D., Hyde, L. W., Kuo, J. R., Goldin, P. R., et al. (2012). Neural mechanisms underlying 5-HTTLPR-related sensitivity to acute stress. *American Journal of Psychiatry, 169*, 397–405.

Dugas, M. J., Laugesen, N., & Bukowski, W. M. (2012). Intolerance of uncertainty, fear of anxiety, and adolescent worry. *Journal of Abnormal Child Psychology, 40*, 863–870.

Dunn, B. D., Billotti, D., Murphy, V., & Dalgleish, T. (2009). The consequences of effortful emotion regulation when processing distressing material: A comparison of suppression and acceptance. *Behaviour Research and Therapy, 47*, 761–773.

Eftekhari, A., Zoellner, L. A., & Vigil, S. A. (2009). Patterns of emotion regulation and psychopathology. *Anxiety, Stress and Coping, 22*, 571–586.

Ehring, T., & Quack, D. (2010). Emotion regulation difficulties in trauma survivors: The role of trauma type and PTSD symptom severity. *Behavior Therapy, 41*, 587–598.

Eifert, G. H., & Heffner, M. (2003). The effects of acceptance versus control contexts on avoidance of panic-related symptoms. *Journal of Behavior Therapy and Experimental Psychiatry, 34*, 293–312.

Ellard, K. K. (2012). *An examination of the neural correlates of emotion acceptance versus worry in generalized anxiety disorder.* Unpublished doctoral dissertation, Boston University, Boston, MA.

Ellard, K. K., Fairholme, C. P., Thompson-Hollands, J., Carl, J. R., Farchione, T. J., & Barlow, D. H. (2011, November). *Early reductions in distress and anxiety related to emotional experiences in the Unified Protocol: Effect on treatment response.* Paper presented at the 45th annual meeting of the Association for Behavioral and Cognitive Therapies, Toronto, Canada.

Etkin, A., Prater, K. E., Hoeft, F., Menon, V.,

& Schatzberg, A. F. (2010). Failure of anterior cingulate activation and connectivity with the amygdala during implicit regulation of emotional processing in generalized anxiety disorder. *American Journal of Psychiatry, 167*, 545–554.

Etkin, A., Prater, K. E., Schatzberg, A. F., Menon, V., & Greicius, M. D. (2009). Disrupted amygdalar subregion functional connectivity and evidence of a compensatory network in generalized anxiety disorder. *Archives of General Psychiatry, 66*, 1361–1372.

Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry, 164*, 1476–1488.

Farchione, T. J., Fairholme, C. P., Ellard, K. K., Boisseau, C. L., Thompson-Hollands, J., Carl, J. R., et al. (2012). Unified protocol for transdiagnostic treatment of emotional disorders: A randomized controlled trial. *Behavior Therapy, 43*, 666–678.

Feldner, M. T., Zvolensky, M. J., Eifert, G. H., & Spira, A. P. (2003). Emotional avoidance: An experimental test of individual differences and response suppression using biological challenge. *Behaviour Research and Therapy, 41*, 403–411.

Friedman, B. H. (2007). An autonomic flexibility–neurovisceral integration model of anxiety and cardiac vagal tone. *Biological Psychology, 74*, 185–199.

Garner, M., Mogg, K., & Bradley, B. P. (2006). Orienting and maintenance of gaze to facial expressions in social anxiety. *Journal of Abnormal Psychology, 115*, 760–770.

Goldin, P. R., Manber, T., Hakimi, S., Canli, T., & Gross, J. J. (2009). Neural bases of social anxiety disorder: Emotional reactivity and cognitive regulation during social and physical threat. *Archives of General Psychiatry, 66*, 170–180.

Goldin, P. R., Manber-Ball, T., Werner, K., Heimberg, R., & Gross, J. J. (2009). Neural mechanisms of cognitive reappraisal of negative self-beliefs in social anxiety disorder. *Biological Psychiatry, 66*, 1091–1099.

Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The neural bases of emotion regulation: Reappraisal and suppression of negative emotion. *Biological Psychiatry, 63*, 577–586.

Goldin, P. R., Ziv, M., Jazaieri, H., Werner, K., Kraemer, H., Heimberg, R. G., et al. (2012). Cognitive reappraisal self-efficacy mediates the effects of individual cognitive-behavioral therapy for social anxiety disorder. *Journal of Consulting and Clinical Psychology, 80*(6), 1034–1040.

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224–237.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*, 348–362.

Gross, J. J., & Levenson, R. W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64*, 970–986.

Gross, J. J., & Levenson, R. W. (1997). Hiding feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology, 106*, 95–103.

Gyurak, A., Goodkind, M. S., Kramer, J. H., Miller, B. L., & Levenson, R. W. (2012). Executive functions and the down-regulation and up-regulation of emotion. *Cognition and Emotion, 26*, 103–118.

Hill, C. L., & Updegraff, J. A. (2012). Mindfulness and its relationship to emotional regulation. *Emotion, 12*, 81–90.

Hofmann, S. G., Ellard, K. K., & Siegle, G. J. (2012). Neurobiological correlates of cognitions in fear and anxiety: A cognitive–neurobiological information-processing model. *Cognition and Emotion, 26*, 282–299.

Hofmann, S. G., Heering, S., Sawyer, A. T., & Asnaani, A. (2009). How to handle anxiety: The effects of reappraisal, acceptance, and suppression strategies on anxious arousal. *Behaviour Research and Therapy, 47*, 389–394.

Jackson, D. C., Malmstadt, J. R., Larson, C. L., & Davidson, R. J. (2000). Suppression and enhancement of emotional responses to unpleasant pictures. *Psychophysiology, 37*, 515–522.

John, O. P., & Eng, J. (this volume, 2014). Three approaches to individual differences in affect regulation: Conceptualization, measures, and findings. In J. J. Gross (Ed.), *Handbook of*

emotion regulation (2nd ed., pp. 321–345). New York: Guilford Press.

Joormann, J., & Siemer, M. (this volume, 2014). Emotion regulation in mood disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 413–427). New York: Guilford Press.

Kashdan, T. B., & Rottenberg, J. (2010). Psychological flexibility as a fundamental aspect of health. *Clinical Psychology Review, 30*, 865–878.

Keough, M. E., Riccardi, C. J., Timpano, K. R., Mitchell, M. A., & Schmidt, N. B. (2010). Anxiety symptomatology: The association with distress tolerance and anxiety sensitivity. *Behavior Therapy, 41*, 567–574.

Koster, E. H., Crombez, G., Verschuere, B., Van Damme, S., & Wiersema, J. R. (2006). Components of attentional bias to threat in high trait anxiety: Facilitated engagement, impaired disengagement, and attentional avoidance. *Behaviour Research and Therapy, 44*, 1757–1771.

Lau, H. C., Rogers, R. D., Haggard, P., & Passingham, R. E. (2004). Attention to intention. *Science, 303*, 1208–1210.

Leutgeb, V., Schäfer, A., & Schienle, A. (2009). An event-related potential study on exposure therapy for patients suffering from spider phobia. *Biological Psychology, 82*, 293–300.

Levitt, J. T., Brown, T. A., Orsillo, S. M., & Barlow, D. H. (2004). The effects of acceptance versus suppression of emotion on subjective and psychophysiological response to carbon dioxide challenge in patients with panic disorder. *Behavior Therapy, 35*, 747–766.

Lieberman, M. D., Inagaki, T. K., Tabibnia, G., & Crockett, M. J. (2011). Subjective responses to emotional stimuli during labeling, reappraisal, and distraction. *Emotion, 11*, 468–480.

MacLeod, C., & Hagan, R. (1992). Individual differences in the selective processing of threatening information, and emotional responses to a stressful life event. *Behaviour Research and Therapy, 30*, 151–161.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95*, 15–20.

Matsuzaka, Y., Akiyama, T., Tanji, J., & Mushiake, H. (2012). Neuronal activity in the primate dorsomedial prefrontal cortex contributes to strategic selection of response tactics. *Proceedings of the National Academy of Sciences, 109*, 4633–4638.

McEvoy, P. M., & Mahoney, A. E. (2012). To be sure, to be sure: Intolerance of uncertainty mediates symptoms of various anxiety disorders and depression. *Behavior Therapy, 43*, 533–545.

McLaughlin, K. A., Hatzenbuehler, M. L., Mennin, D. S., & Nolen-Hoeksema, S. (2011). Emotion dysregulation and adolescent psychopathology: A prospective study. *Behaviour Research and Therapy, 49*, 544–554.

McLaughlin, K. A., Mennin, D. S., & Farach, F. J. (2007). The contributory role of worry in emotion generation and dysregulation in generalized anxiety disorder. *Behaviour Research and Therapy*, 45, 1735–1752.

Mennin, D. S., & Fresco, D. M. (this volume, 2014). Emotion regulation therapy. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 469–490). New York: Guilford Press.

Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2005). Preliminary evidence for an emotion dysregulation model of generalized anxiety disorder. *Behaviour Research and Therapy, 43*, 1281–1310.

Mennin, D. S., McLaughlin, K. A., & Flanagan, T. J. (2009). Emotion regulation deficits in generalized anxiety disorder, social anxiety disorder, and their co-occurrence. *Journal of Anxiety Disorders, 23*, 866–871.

Montag, C., Fiebach, C. J., Kirsch, P., & Reuter, M. (2011). Interaction of 5-HTTLPR and a variation on the oxytocin receptor gene influences negative emotionality. *Biological Psychiatry, 69*, 601–603.

Moore, S. A., Zoellner, L. A., & Mollenholt, N. (2008). Are expressive suppression and cognitive reappraisal associated with stress-related symptoms? *Behaviour Research and Therapy, 46*, 993–1000.

Munafo, M. R., Brown, S. M., & Hariri, A. R. (2008). Serotonin transporter (5-HTTLPR) genotype and amygdala activation: A meta-analysis. *Biological Psychiatry, 63*, 852–857.

New, A. S., Fan, J., Murrough, J. W., Liu, X., Liebman, R. E., Guise, K. G., et al. (2009). A functional magnetic resonance imaging study of deliberate emotion regulation in resilience and posttraumatic stress disorder. *Biological Psychiatry, 66*, 656–664.

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. (2002). Rethinking feelings: An FMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14*, 1215–1229.

Ochsner, K. N., Knierim, K., Ludlow, D. H.,

Hanelin, J., Ramachandran, T., Glover, G., & Mackey, S. C. (2004). Reflecting upon feelings: An fMRI study of neural systems supporting the attribution of emotion to self or other. *Journal of Cognitive Neuroscience, 16*, 1746–1772.

Ochsner, K. N., Ray, R. D., Cooper, J. C., Robertson, E. R., Chopra, S., Gabrieli, J. D., et al. (2004). For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *NeuroImage, 23*, 483–499.

Olatunji, B. O., & Wolitzky-Taylor, K. B. (2009). Anxiety sensitivity and the anxiety disorders: A meta-analytic review and synthesis. *Psychological Bulletin, 135*, 974–999.

Paquette, V., Lévesque, J., Mensour, B., Leroux, J. M., Beaudoin, G., Bourgouin, P., et al. (2003). "Change the mind and you change the brain": Effects of cognitive-behavioral therapy on the neural corrleates of spider phobia. *NeuroImage, 18*, 401–409.

Pezawas, L., Meyer-Lindenberg, A., Drabant, E. M., Verchinski, B. A., Munoz, K. E., Kolachana, B. S., et al. (2005). 5-HTTLPR polymorphism impacts human cingulate–amygdala interactions: A genetic susceptibility mechanism for depression. *Nature Neuroscience, 8*, 828–834.

Pineles, S. L., & Mineka, S. (2005). Attentional biases to internal and external sources of potential threat in social anxiety. *Journal of Abnormal Psychology, 114*, 314–318.

Roemer, L., Orsillo, S. M., & Salters-Pedneault, K. (2008). Efficacy of an acceptance-based behavior therapy for generalized anxiety disorder: Evaluation in a randomized controlled trial. *Journal of Consulting and Clinical Psychology, 76*, 1083–1089.

Rothbart, M. K., Sheese, B. E., & Posner, M. I. (this volume, 2014). Temperament and emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 305–320). New York: Guilford Press.

Salters-Pedneault, K., Roemer, L., Tull, M. T., Rucker, L., & Mennin, D. S. (2006). Evidence of broad deficits in emotion regulation associated with chronic worry and generalized anxiety disorder. *Cognitive Therapy and Research, 30*, 469–480.

Schmidt, N. B., Lerew, D. R., & Jackson, R. J. (1999). Prospective evaluation of anxiety sensitivity in the pathogenesis of panic: Replication and extension. *Journal of Abnormal Psychology, 108*, 532–537.

Sheppes, G., Scheibe, S., Suri, G., & Gross, J. J. (2011). Emotion regulation choice. *Psychological Science, 22*, 1391–1396.

Shin, L. M., & Liberzon, I. (2010). The neurocircuitry of fear, stress, and anxiety disorders. *Neuropsychopharmacology, 35*, 169–191.

Speilberg, J. M., Miller, G. A., Engels, A. S., Herrington, J. D., Sutton, B. P., Banich, M. T., et al. (2011). Trait approach and avoidance motivation: Lateralized neural activity associated with executive function. *NeuroImage, 54*, 661–670.

Stein, M. B., Shorck, N. J., & Gelernter, J. (2008). Gene-by-environment (serotonin transporter and childhood maltreatment) interaction for anxiety sensitivity, an intermediate phenotype for anxiety disorders. *Neuropsychopharmacology, 33*, 312–319.

Straube, T., Glauer, M., Dilger, S., Mentzel, H. J., & Miltner, W. H. (2006). Effects of cognitive-behavioral therapy on brain activation in specific phobia. *NeuroImage, 29*, 125–135.

Suárez, L. M., Bennett, S. M., Goldstein, C. R., & Barlow, D. H. (2009). Understanding anxiety disorders from a "triple vulnerability" framework. In M. M. Antony & M. B. Stein (Eds.), *Oxford handbook of anxiety and related disorders* (pp. 153–172). New York: Oxford University Press.

Tan, P. Z., Forbes, E. E., Dahl, R. E., Ryan, N. D., Siegle, G. J., Ladouceur, C. D., et al. (2012). Emotional reactivity and regulation in anxious and nonanxious youth: A cell-phone ecological momentary assessment study. *Journal of Child Psychology and Psychiatry, 53*, 197–206.

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry, 39*, 255–266.

Thompson, R. A. (this volume, 2014). Socialization of emotion and emotion regulation in the family. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 173–186). New York: Guilford Press.

Tolin, D. F., Abramowitz, J. S., Brigidi, B. D., & Foa, E. B. (2003). Intolerance of uncertainty in obsessive–compulsive disorder. *Journal of Anxiety Disorders, 17*, 233–242.

Troy, A. S., Wilhelm, F. H., Shallcross, A. J., & Mauss, I. B. (2010). Seeing the silver lining: Cognitive reappraisal ability moderates the relationship between stress and depressive symptoms. *Emotion, 10*, 783–795.

Tull, M. T., & Roemer, L. (2007). Emotion regu-

lation difficulties associated with the experience of uncued panic attacks: Evidence of experiential avoidance, emotional nonacceptance, and decreased emotional clarity. *Behavior Therapy, 38*, 378–391.

Turk, C. L., Heimberg, R. G., Luterek, J. A., Mennin, D. S., & Fresco, D. M. (2005). Emotion dysregulation in generalized anxiety disorder: A comparison with social anxiety disorder. *Cognitive Therapy and Research, 29*, 89–106.

van den Hout, M., Tenney, N., Huygens, K., Merckelbach, H., & Kindt, M. (1995). Responding to subliminal threat cues is related to trait anxiety and emotional vulnerability: A successful replication of Macleod and Hagan (1992). *Behaviour Research and Therapy, 33*, 451–454.

Wager, T. D., Davidson, M. L., Hughes, B. L., Lindquist, M. A., & Ochsner, K. N. (2008). Prefrontal-subcortical pathways mediating successful emotion regulation. *Neuron, 59*, 1037–1050.

Weiss, N. H., Tull, M. T., Davis, L. T., Dehon, E. E., Fulton, J. J., & Gratz, K. L. (2012). Examining the association between emotion regulation difficulties and probable posttraumatic stress disorder within a sample of African Americans. *Cognitive Behavior Therapy, 41*, 5–14.

Wilamowska, Z. A., Thompson-Hollands, J., Fairholme, C. P., Ellard, K. K., Farchione, T. J., & Barlow, D. H. (2010). Conceptual background, development, and preliminary data from the unified protocol for transdiagnostic treatment of emotional disorders. *Depression and Anxiety*, 27, 882–890.

Wolgast, M., Lundh, L. G., & Viborg, G. (2011). Cognitive reappraisal and acceptance: An experimental comparison of two emotion regulation strategies. *Behaviour Research and Therapy, 49*, 858–866.

CHAPTER 25

Emotion Regulation in Mood Disorders

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Imagine receiving a negative evaluation at work, having an argument with your partner, discovering you have forgotten to pay a bill and have incurred a late fee; and imagine experiencing all of this on a gray, rainy, winter day. Situations like these, which have the potential to induce lasting negative affect, are part of everyday life. Perhaps not surprisingly, researchers have found that people generally do not endure negative affect passively; rather, they actively use strategies to try to regulate their negative moods and emotions. It is important to note, however, that there is a wide range of individual differences in the ability to regulate affect: Whereas some individuals can successfully regulate their negative affect, others cannot, and still others may respond to their emotions in ways that further exacerbate their negative affect. Importantly, it is becoming increasingly clear that these individual differences in the regulation of negative moods and emotions play a significant role in the onset and maintenance of mood disorders. Less frequently discussed is the regulation of positive moods and emotions and of difficulties that may stem from an inability to respond effectively to positive affect. Excessive bathing in the glory of initial positive feedback, for example, may lead to an overestimation of the ability to complete the project in a timely manner, whereas an inability to savor a success and the use of strategies that down-regulate the ensuing positive mood may interfere with the motivation to start the next project. Therefore, both difficulties in the regulation of positive and negative affect have been implicated in mood disorders.

Mood disorders are among the most prevalent of all psychiatric disorders, with major depressive disorder (MDD) affecting almost 20% of Americans at some point in their lives (Kessler & Wang, 2009). Given the high prevalence and the substantial personal and societal costs of mood disorders, efforts to identify risk factors and underlying mechanisms, as well as effective intervention strategies, are particularly pressing. As implied by the label, the hallmark feature of mood disorders is disordered affect. MDD is defined by sustained negative affect and difficulties experiencing positive affect. Bipolar disorder (BD) is characterized by episodes of depressed but also elevated and/or irritable mood. Despite the fact that disordered affect is the central feature of mood disorders, theories of the onset, maintenance, and recurrence of these disorders have traditionally focused on cognition and

behavior but not as much on affect. Indeed, cognitive-behavioral interventions have proven successful in treating these disorders by focusing on the modification of maladaptive cognitions and behaviors (Beck, Rush, Shaw, & Emery, 1979). Despite these successes, many researchers have pointed out that there is room for improvement in our theoretical models and in our treatment approaches. Specifically, a closer look at the concept of emotion regulation and at mechanisms that allow us to understand individual differences in the important ability to regulate affective states may help us better understand vulnerability to affective disorders and thereby improve our treatment approaches.

Indeed, models of both depression and BD have emphasized the role of emotion regulation. It has been proposed, for example, that individuals who experience episodes of depression do not differ from their nondepressed counterparts in the degree to which they become sad but instead are characterized by an inability to repair or regulate their emotions, resulting in longer episodes of sadness and depressed mood for depression-prone individuals (Teasdale, 1988; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). In depression, preferential use of maladaptive strategies, such as rumination, as well as difficulties using adaptive strategies, such as reappraisal, may explain why negative emotions in response to life events quickly spiral into sustained negative mood (e.g., Nolen-Hoeksema et al., 2008). In BD, we find similar difficulties regulating negative affect. In addition, maladaptive responding to positive affect and events may explain how elevated mood leads into a manic episode (Gruber, 2011; Johnson, 2005). Indeed, Gruber (2011) points out that "BD involves a tendency to increase or amplify positive emotions" (p. 219). Likewise, Johnson (2005) has described how increases in positive affect after an initial success and individual differences in responding to positive mood are related to the development of mania. Even though these predictions seem plausible, however, empirical evidence is still largely missing, particularly when it comes to BD. A first goal of this chapter is therefore to summarize empirical findings that support the proposition that people who are diagnosed with affective disorders do indeed differ from their nondisordered counterparts in emotion regulation.

At least two different but related aspects of emotion regulation are crucial in this context. First, it is possible that people who are prone to experience affective disorders and their nondisordered counterparts differ in the selection of specific emotion regulation strategies because of wrong expectations about the usefulness of a strategy in a given situation, or because of acquired preferences for the selection of ineffective or maladaptive strategies. Indeed, numerous studies show that depression is associated with a tendency to ruminate when experiencing negative affect, and that the use of this strategy is perceived as helpful in coping with negative events even though it in fact interferes with recovery (Papageorgiou & Wells, 2001). In addition, it is possible that affective disorders are characterized by a more limited repertoire of strategies that may interfere with the flexible use of multiple strategies that fit the situation at hand. This flexibility has been proposed as particularly adaptive (Kashdan & Rottenberg, 2010). It is also possible, however, that depression-related difficulties in emotion regulation are due not to strategy selection but to problems implementing adaptive strategies such as reappraisal. Of course, these two aspects of emotion regulation in affective disorders may be related. Difficulties implementing an adaptive strategy, for example, may result in preferential selection of maladaptive strategies.

In this chapter, we review studies examining the first aspect of emotion regulation in affective disorders by summarizing research on the use of different emotion regulation strategies, then examine evidence for the second, that people with affective disorders have difficulties implementing adaptive strategies. We also examine underlying mechanisms of emotion dysregulation in affective disorders by focusing on basic cognitive processes that may help or hinder emotion regulation and discuss how these may be affected by mood disorders. Our discussion focuses on studies that have used clinically diagnosed samples, but we also review studies with analogue samples if they are particularly informative or if clinical studies are missing.

Emotion regulation is defined here as strategic and automatic processes that influ-

ence the occurrence, magnitude, duration, and expression of an emotional response (see Gross, this volume). The focus of most studies on emotion regulation is the down-regulation of negative affect. This aspect of emotion regulation is also the primary focus of this chapter when it comes to MDD. However, we also discuss how depression may be associated with problems savoring positive affect and that a risk factor for BD is rumination on positive events (e.g., in response to successful goal achievement), which may result in a failure to down-regulate positive affect that may then spiral into a full-blown manic episode (Johnson, 2005).

Strategy Selection in Mood Disorders

Recent research suggests that it is overly simplistic to differentiate adaptive from maladaptive strategies. Indeed, the adaptiveness of a particular strategy will likely depend on the context in which it is used, as well as the flexibility of its use (Bonnano, Papa, Lalande, Westphal, & Coifman, 2004). Still, previous research has identified specific strategies that are more likely to incur additional costs (e.g., increase in cognitive load or physiological arousal) and that seem less likely to regulate affect effectively (Gross & John, 2003). Emotion regulation is a broad concept that comprises a number of different processes, and it is beyond the scope of this chapter to provide an exhaustive review of the literature. We therefore focus on strategies that have consistently been linked to mood disorders and have been the focus of empirical studies on depression and/or BD.

Rumination

A particularly detrimental response to negative affect that has been associated with various emotional disorders is *rumination*, which is bringing an idea back to mind over and over again. Originally the word referred to the way cows and certain other animals eat, storing partially digested food in a stomach called a rumen, bringing that food up later to chew over more thoroughly. Even in the original Latin, however, it took on a vivid figurative meaning, describing the practice of bringing an idea back to mind to work over further. Many of us mull over important matters in this way, digesting them a little at a time. But in depression, rumination frequently features negative, self-deprecating statements and pessimistic ideas about the world and the future.

Depressive rumination has been shown in numerous studies to exacerbate and prolong depressed mood (see review by Nolen-Hoeksema et al., 2008). It may seem counterintuitive to think that someone would use rumination as an emotion regulation strategy given that numerous studies show that it increases negative affect. However, it is a frequently used response when people experience negative affect or encounter a negative event, and studies show that depressed individuals perceive many benefits of rumination, such as feelings of increased self-awareness and understanding (Papageorgiou & Wells, 2001). Lyubomirksy and Nolen-Hoeksema (1993), for example, reported that rumination in dysphoric participants (i.e., participants with subclinical levels of depressive symptoms usually assessed via self-report) was associated with an enhanced sense of insightfulness. Empirical evidence has shown that people who tend to engage in rumination when distressed are more likely to develop depressive disorders and tend to experience more prolonged periods of depression (e.g., Nolen-Hoeksema, 2000). Indeed, considerable evidence has linked higher trait rumination with the onset and maintenance of depression (see Nolen-Hoeksema et al., 2008, for a review). Nolen-Hoeksema (2000), for example, examined a sample of approximately 1,300 adults randomly selected from the community. Among nondepressed individuals, rumination scores at first assessment predicted the onset of new major depressive episodes over the following year. Researchers have also examined the effects of an experimental induction of rumination on people's mood and behaviors. Rumination in these studies is usually contrasted with distraction, another emotion regulation strategy that we discuss later. In general, compared with distraction, rumination leads to sustained negative mood, increases in negative cognitions and overgeneral autobiographical memory, and decreases in effective problem solving in depressed participants (e.g., Watkins & Moulds, 2005).

Although much less studied, individuals with BD have also been shown to ruminate about negative emotion. In fact, some studies indicate that people with BD ruminate even more than people with unipolar depression (Kim, Yu, Lee, & Kim, 2012). However, BD is associated with not only increased depressive rumination but also rumination about positive emotion. Indeed, *positive rumination*, defined as dwelling on the content, consequences, and causes of positive feelings (Feldman, Joormann, & Johnson, 2008), has been identified as an important risk factor for BD. Gruber (2011), for example, proposes that positive rumination may interfere with the processing of relevant external information that could help terminate the emotional state. Compared to control participants, college students diagnosed with BD or MDD endorsed heightened rumination in response to negative affect, but only those with BD endorsed elevated rumination in response to positive affect (Johnson, McKenzie, & McMurrich, 2008). Moreover, within BD, ruminative responses to negative affect were explained by depressive symptoms. Gruber, Eidelman, Johnson, Smith, and Harvey (2011) also examined rumination about positive and negative emotions: Compared to the control group, the BD group endorsed greater trait rumination for positive as well as negative emotions. Interestingly, participants with BD in this sample were not at the time experiencing episodes of depression or mania, which suggests that elevated rumination is not just a correlate of the depressive or manic state. Importantly, trait rumination about negative and positive emotion was associated with greater lifetime depression frequency, whereas trait rumination about positive emotion was associated with greater lifetime mania frequency. Similarly, Alloy et al. (2009) found that trait rumination about positive emotion was associated with greater lifetime mania frequency in their BD group. Although few rumination inductions have been used in the BD literature, researchers found heightened positive emotion in a ruminative compared with reflective (i.e., third-person perspective) induction in participants with BD compared with healthy controls (Gruber, Harvey, & Johnson, 2009). In contrast, Gruber et al. (2011) exposed their participants with interepisode BD and control participants to a rumination induction in response to the recall of a positive autobiographical memory and reported no group differences in self-reported affect or physiological responding.

It seems interesting that whereas positive rumination is associated with negative outcomes in BD, depressed participants may have difficulties savoring positive events. Thus, although rumination on negative content clearly seems present and maladaptive in both MDD and BD, the maladaptive response may be rumination on positive content in BD, whereas lack of savoring may be the maladaptive response in MDD. Raes, Smets, Nelis, and Schoofs (2012), for example, found in two nonclinical student samples that a self-reported dampening response to positive affect predicted depressive symptoms in 3- and 5-month follow-up assessments.

Reappraisal

In his fascinating book on depression, Andrew Solomon (2001) writes about one of his depressive episodes, "I started to fear, every time my dog left the room, that it was because he was not interested in me" (p. 86). The tendency to interpret emotion-eliciting situations in a negative way is a defining feature of depressive disorders. Altering these interpretations is obviously a powerful way to regulate affect and a main component of cognitive therapy for depression. *Reappraisal* involves changing a situation's meaning to alter one's emotional response to the situation (Gross, 1998; Gross & John, 2003). Reappraisal has been studied extensively in nonclinical populations and has been shown to reduce negative affect (John & Gross, 2004; Urry, 2009). In addition, reappraisal does not entail the social and cognitive costs associated with rumination and other less adaptive strategies (Richards & Gross, 2000). Furthermore, it is associated with reduced physiological activation in response to negative emotion (Ray, McRae, Ochsner, & Gross, 2010; Urry, 2009).

Not surprisingly, studies in clinical and nonclinical samples have tied less frequent habitual use of reappraisal to greater depression severity (Garnefski & Kraaij, 2006; Joormann & Gotlib, 2010). It should be noted, however, that most of these studies have used a cross-sectional design, which

makes it difficult to examine whether decreased reappraisal use is a symptom of depression or indeed a risk factor. Garnefski, Legerstee, Kraaij, van den Kommer, and Teerds (2002), for example, found reduced use of reappraisal in a clinical sample diagnosed with depression and anxiety. Few researchers have examined the habitual use of reappraisal in BD. One exception is Gruber, Harvey, and Gross (2012), who examined the spontaneous use of reappraisal in BD in response to emotional film clips. They found that individuals with BD were more likely than control individuals to use reappraisal in response to not only positive and negative but also neutral film clips. The authors interpreted this result as showing that BD is associated with more regulation effort.

It is important to note that habitual reappraisal is not beneficial in all contexts. Some studies have found habitual use of reappraisal to be unrelated to symptoms or correlated with poorer outcomes. Nezlek and Kuppens (2008), for example, who examined daily measures of affect and emotion regulation, found that daily use of reappraisal to upregulate positive emotions was associated with increases in positive affect and self-esteem, whereas no relation was found between the use of reappraisal to down-regulate negative affect and the experience of negative affect. In addition, in a recent meta-analysis, Aldao, Nolen-Hoeksema, and Schweizer (2010) reported that whereas greater reappraisal use is related to lower depression and anxiety symptoms, reappraisal is more inconsistently related to symptoms than other strategies (rumination, suppression). In a follow-up study, Aldao and Nolen-Hoeksema (2012) found that the degree to which reappraisal is adaptive depends on the frequency of maladaptive strategy use; reappraisal was related to lower depression symptoms but only for individuals who frequently use maladaptive strategies (rumination, suppression, etc.). The authors argued that reappraisal serves as a compensatory strategy to counteract the problems that come with greater maladaptive strategy use.

Suppression

Suppression has long been regarded as a particularly maladaptive regulation strategy. *Suppression* is conceptualized as an emotion regulation strategy by which an individual attempts to inhibit the effects of external cues on internal (e.g., physiological) and external (e.g., emotional expression) states. The inhibition of emotion expression is frequently referred to as *expressive suppression*. Research on emotion suppression indicates that habitual use of this strategy is largely ineffective in reducing negative emotions. Findings indicate that expressive suppression, for example, is associated with increased depression symptoms (Joormann & Gotlib, 2010). Moreover, emotion suppression is associated with increased use of rumination (Liverant, Kamholz, Sloan, & Brown, 2011) and decreased inhibitory control (Joormann & Gotlib, 2010). However, one study indicates that providing instructions on the use of expressive suppression in response to negative stimuli may be efficacious in reducing acute emotional responding among depressed participants (Liverant, Brown, Barlow, & Roemer, 2008). Though virtually no studies have examined the use of emotion suppression in individuals with BD, one recent study found that, compared to healthy controls, interepisode participants with BD demonstrate increased use of spontaneous expressive suppression in response to both positive and negative mood inductions (Gruber et al., 2012). In a clinical sample that included depressed and anxious participants, Campbell-Sills, Barlow, Brown, and Hofmann (2006) examined emotion suppression, using a mood induction film and the assessment of spontaneous use of emotion regulation strategies. They reported that clinical participants used more suppression, and that suppression was related to higher levels of negative affect.

Use of Multiple Strategies

Few studies have examined the use of more than one strategy in psychopathology. A noteworthy exception is the aforementioned meta-analysis by Aldao et al. (2010), which examined the relation between the habitual use of six emotion regulation strategies (rumination, reappraisal, suppression, acceptance, problem solving, avoidance) and four groups of symptoms of psychopathology (depression, anxiety, substance-related disorders, and eating disorders) in studies

that used clinical or normative samples. They obtained a large effect size for the association of depression with rumination and avoidance, a medium to large effect size for problem solving and suppression, and small effect sizes for acceptance and reappraisal. The authors summarized their findings by pointing out that, overall, adaptive emotion regulation strategies (reappraisal, acceptance) showed a weaker association with psychopathology than maladaptive strategies. In a follow-up study using a community sample, Aldao et al. (2010) further found an interaction between the use of adaptive and maladaptive strategies cross-sectionally. High use of adaptive strategies was associated with lower psychopathology only in participants who also reported high levels of use of maladaptive strategies. The authors further reported that the use of adaptive strategies did not predict psychopathology in a follow-up assessment, whereas the use of maladaptive strategies significantly predicted psychopathology 6 months later.

Studies that use clinical samples and examine the use of a range of different emotion regulation strategies are particularly informative. D'Avanzato, Joormann, Siemer, and Gotlib (2013), for example, compared habitual use of emotion regulation strategies in control participants and in samples of participants diagnosed with depression or social anxiety disorder. These authors reported that depressed participants were more likely to ruminate and less likely to use reappraisal than control participants, whereas participants with social anxiety disorder reported more expressive suppression compared to the two other groups. In addition, whereas elevated rumination was also found in previously depressed participants who were not currently depressed, healthy controls and the previously depressed group did not differ on use of reappraisal or expressive suppression. Interestingly, only use of reappraisal and rumination were related to symptom severity across all samples; expressive suppression use was unrelated to depressive or anxiety symptoms.

These results are in line with a recent study by Ehring, Tuschen-Caffier, Schnülle, Fischer, and Gross (2010) that compared formerly depressed to never-depressed participants. Like D'Avanzato et al. (2013), they reported that formerly depressed participants used more rumination but did not differ from never-depressed participants in reported use of reappraisal or suppression. In addition, the formerly depressed group reported more emotion nonacceptance. Interestingly, even though the groups did not differ on habitual use of suppression, the formerly depressed group reported more spontaneous use of suppression in response to a negative film clip. No group differences were found in reappraisal use in response to the film clip. The authors interpret this finding to suggest that depression may not be associated with suppression of all emotional experiences but rather with the specific suppression of sadness. Further studies are needed to examine this possibility more closely.

Few studies have examined the use of multiple strategies in BD. Green et al. (2011) showed that the BD group reported more frequent use of rumination and less frequent use of reappraisal in response to negative life events compared to unaffected relatives, but this study did not examine responses to positive events. Abnormal response styles to negative mood have previously been observed in students with hypomanic traits (Thomas & Bentall, 2002) and in patients with BD (Thomas, Knowles, Tsai, & Bentall, 2007). Thomas and Bentall (2002) found that hypomanic traits were associated with not only rumination but also increased distraction and risk taking. Thomas et al. (2007) found that patients with mania reported high levels of risk taking and active coping (distraction and problem solving), whereas patients with remitted BD reported high levels of rumination. Clearly more research is needed on the interplay of emotion regulation strategies in this disorder.

Summary of Strategy Selection

In summary, the results clearly show that depression is characterized by increased use of rumination and less use of reappraisal. There is some indication that rumination use may be a stable feature of depression risk even outside of acute episodes of the disorder, whereas reduced use of reappraisal is observed only in people who are currently depressed (see D'Avanzato et al., 2013; Ehring et al., 2010). This finding suggests

that rumination may be a risk factor for not only the recurrence but also the onset of depression, whereas changes in reappraisal use may be an epiphenomenon of experiencing a depressive episode. Results for expressive suppression and other forms of suppression are less clear-cut, as are findings for the use of acceptance in depression.

It is interesting to note that, in contrast to MDD, BD seems to be characterized by problems regulating negative and positive affect, and many studies now suggest an important role for positive rumination in the onset of manic episodes. Indeed, it is possible that acute mood state plays an important role here, with patients with BD in a depressive episode resembling patients with MDD in their use of rumination and reduced use of reappraisal. At the same time, positive rumination when experiencing hypomania may play a critical role in the onset of manic episodes (Johnson, 2005). Other studies have shown that participants with BD deliberately choose self-calming strategies during early phases of hypomania to try to prevent the emergence of manic symptoms (Lam & Wong, 1997). The use of other emotion regulation strategies in BD, however, has either not been studied or studies have led to inconclusive results. It should be noted that BD is also characterized by irritable affect, yet no studies have examined the relation of this aspect of the disorder to emotion regulation.

It is further important to note that most studies reviewed here examine individuals who are currently in episode. It is not clear, therefore, whether the use of maladaptive strategies precedes the onset of the disorder or is merely another symptom of being depressed or manic. Gaining more insight into whether the use of emotion regulation strategies is a vulnerability factor will be important for intervention and prevention programs. In addition, we should note that the majority of studies of habitual strategy use are cross-sectional and have relied on self-report only. This is problematic, because it is not clear to what extent people can accurately report on their use of strategies, and the self-report of emotion regulation may be affected by current mood state. More studies that examine strategy use in response to a mood manipulation (e.g., Ehring et al. 2010) are clearly needed. Importantly, it is not clear what underlies the preference and dispositional use of specific emotion regulation strategies in affective disorders. It is likely that difficulties implementing certain strategies also shape the selection of emotion regulation strategies. In the second part of this chapter, we therefore examine the implementation of emotion regulation strategies in mood disorders and try to identify cognitive processes that may underlie difficulties in emotion regulation.

Implementation of Adaptive Strategies

Whereas the majority of studies discussed in the previous section used self-report to examine the frequency of use of different emotion regulation strategies, other studies have focused on whether affective disorders are characterized by less effective implementation of adaptive strategies. These researchers typically instruct participants to use a strategy such as reappraisal during an emotion-eliciting event, then assess affect, as well as psychophysiological indicators of arousal, before and after the use of the strategy.

Distraction

Perhaps the best studied adaptive strategy in depression is distraction. Distraction is frequently used as a control condition in rumination studies, and many studies have shown that distraction seems to regulate affect effectively in depressed participants (see Nolen-Hoeksema et al., 2008, for a review). Whereas depressed participants instructed to ruminate show increased negative affect and recall of negative memories, those instructed to distract do not differ from the control group on these measures. For example, in a study that examined the recall of positive memories in depression, currently and formerly depressed participants did not differ from control participants in their affect recovery after a negative mood induction that was followed by a distraction induction (Joormann, Siemer & Gotlib, 2007). Thus, studies suggest that depressed participants are able to use distraction to regulate negative affect effectively. Research on the use of distraction in BD is currently nonexistent.

Reappraisal

Only a handful of studies has examined the effectiveness of reappraisal in affective disorders, and results are much more mixed. Indeed, to our knowledge, no study thus far has examined whether current MDD is associated with less effective reappraisal use. Troy, Wilhelm, Shallcross and Mauss (2010) recruited a nonclinical sample of individuals who had recently experienced a stressful life event and found that elevated depression symptoms were related to less effective reappraisal in response to an emotion-eliciting film clip. Similarly, McRae, Jacobs, Ray, John, and Gross (2012) reported that individuals with lower well-being (composite of lower positive affect, higher negative affect, and lower life satisfaction) exhibited reduced effectiveness of reappraisal in response to negative emotional pictures, and that performance on this task was related to reduced habitual use of reappraisal. Thus, this study suggests a relation between the effectiveness of strategies and habitual strategies use as proposed earlier. In contrast, Ehring et al. (2010) reported that even though formerly depressed participants were less likely to endorse the use of reappraisal on self-report, they did not differ from the control group in their ability to use reappraisal effectively when explicitly instructed to do so. Indeed, these authors summarized their results by saying that formerly depressed participants choose ineffective strategies but can use more functional ones if so instructed. Given that no studies have examined reappraisal effectiveness in current depression, however, it is unclear whether this result is specific to a previously depressed sample or would generalize to current depression. Very few studies have looked at the effectiveness of different emotion regulation strategies in BD. A noteworthy exception is the previously mentioned study by Gruber et al. (2012), who found that participants with BD reported more effort when using reappraisal and suppression but also less success. It should be noted, however, that effort and success were assessed using two self-report items; these results therefore clearly require replication using alternative operationalizations of these constructs.

Flexibility of use and the timing over the course of an emotional response are key variables that may influence the effectiveness of reappraisal. Reappraisal, initiated once an emotional response is underway (often referred to as *online reappraisal*), appears to be more difficult to implement and more cognitively taxing than anticipatory reappraisal or other strategies, such as distraction, implemented at early processing stages of an emotional episode (Sheppes & Meiran, 2007; Sheppes, Catran, & Meiran, 2009; Urry, 2009). Online reappraisal may be particularly important to examine in MDD. Given the persistent negative affect characterizing this disorder, individuals with depression are likely to need to implement emotion regulation strategies in response to an existing emotional state of relatively high intensity. If implemented online, reappraisal probably requires levels of cognitive control that may not be available to people with affective disorders. This may explain the mixed findings on the effectiveness of reappraisal in mood disorders. We discuss this point in more detail later, when we examine cognitive processes that underlie effective emotion regulation.

Mood-Incongruent Recall

Other studies have examined the recall of mood-incongruent material in depression (Joormann et al., 2007). In these studies, participants did not receive explicit instructions to regulate affect. Instead, they were informed that the project examined memory processes and were asked to recall mood-incongruent memories (i.e., recall of a positive autobiographical memory) immediately following a negative mood induction. Recall of mood-incongruent memories was compared to a distraction condition. The results showed that not only dysphoric but also currently depressed and formerly depressed participants compared to controls did not experience a recovery of their negative affect when recalling mood-incongruent memories (see also Werner-Seidler & Moulds, 2012). Distraction, however, repaired negative affect in all groups. These results suggest that depression is associated with an inability to focus on positive material to offset negative affect, which may be an important automatic mood repair mechanism. The results are well aligned with the aforementioned finding that depression is associated with reduced savoring after experiencing positive events.

Summary of Strategy Implementation

In summary, the studies presented so far suggest that affective disorders are associated with more frequent use of maladaptive strategies, as well as difficulties implementing adaptive strategies. Specifically, even though distraction seemed to work for depressed participants, recall of positive material worked well in nondisordered participants but did not repair negative affect in the clinical sample. Results of studies focusing on reappraisal are more mixed and it may be important to take the timing of reappraisal into account.

Why do depressed people show a preference for the use maladaptive strategies, and why do they find it difficult to use adaptive strategies? Cognitive theories of emotional disorders have identified mood-congruent biases in the processing of emotional information, and recent research has examined deficits in cognitive control that may interfere with emotion regulation (Joormann, 2010). These studies may improve our insight into mechanisms that underlie emotion and mood regulation difficulties and thereby provide critical information for interventions. However, given the scarcity of research on this topic in BD, in the following section we focus primarily on MDD.

Cognitive Processes and Difficulties in Emotion Regulation

Cognitive Biases

Individual differences in cognitive processes such as attention, memory, and interpretation may affect the attention toward emotion-eliciting aspects of a situation, the initial appraisal of a situation, and the ability to use regulation strategies such as reappraisal or distraction effectively (see Figure 25.1). Cognitive models of depression posit that depressed individuals exhibit mood-congruent biases in all aspects of information processing, including attention, memory, and interpretation (Mathews & MacLeod, 2005). Biased memory for negative, relative to positive, information represents perhaps the most robust cognitive finding associated with MDD and may be related to the difficulties using mood-incongruent recall to repair negative affect, as we discussed earlier. Other studies have reported interpretation biases in depression (Lawson, MacLeod, & Hammond, 2002). Indeed, a recent meta-analysis by Hallion and Ruscio (2012) showed that biased interpretation is one of the most consistently reported findings in the depression literature. Interpretation biases may lead

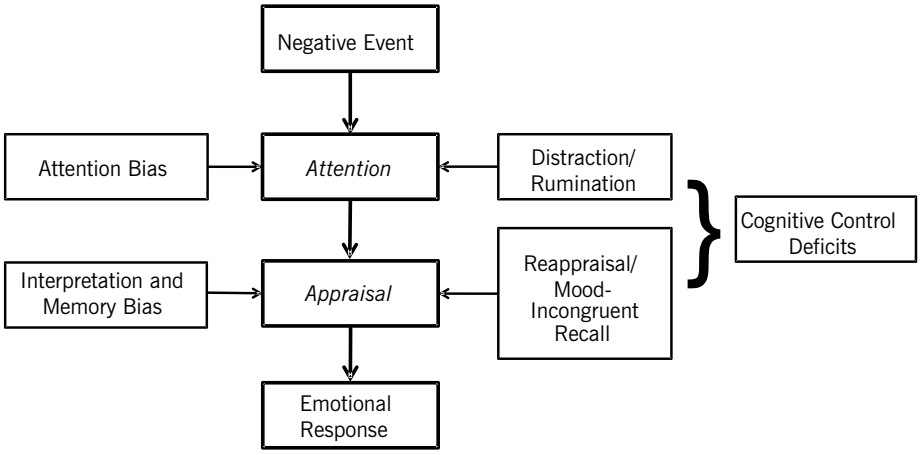

FIGURE 25.1. Cognitive processes and emotion dysregulation in depression. Cognitive biases such as difficulties disengaging from negative material and biases in interpretation and memory, affect both attention to emotion-eliciting aspects of events and appraisals of the event, which in turn affect the emotion response. Through their effect on attention and appraisal processes, cognitive biases also interfere with emotion regulation. In addition, cognitive control deficits further impair the effective implementation of emotion regulation strategies such as reappraisal, distraction, and mood-incongruent recall, increasing the likelihood of maladaptive responding, for example, in the form of rumination.

to inflexible, automatic, and unconscious appraisals that make it difficult to regulate emotions by using deliberate reappraisal of the situation (Siemer & Reisenzein, 2007). Whereas some studies have failed to find attentional biases in depression, others suggest that stimulus exposure duration is critical (e.g., Mogg, Bradley, Williams, & Mathews, 1993). Specifically, biases have been found when stimuli are presented for longer durations (see Mathews & MacLeod, 2005, for a review). Recent studies therefore suggest that depressed individuals do not direct their attention to negative information more frequently than do control participants, but once it captures their attention, they exhibit difficulties disengaging from it (e.g., Joormann & Gotlib, 2007). Difficulties disengaging from negative material may result in maintenance of attention on mood-congruent information, which may eventually lead to recurrent ruminative thoughts.

Cognitive Control

In addition to cognitive biases, depression-related deficits in cognitive control may affect emotion regulation (see Figure 25.1). Negative mood is generally associated with the activation of mood-congruent representations in working memory (Siemer, 2005). The ability to control the contents of working memory (WM) might therefore play an important role in recovery from negative affect. WM is a limited-capacity system that reflects the focus of attention and the temporary activation of representations that are the content of awareness. Cognitive inhibition is part of executive control processes that select and update WM content (Hasher, Zacks, & May, 1999). Deficits in cognitive inhibition may make it difficult to discard mood-congruent content from WM, keeping attention focused on the emotion-eliciting aspects and the initial appraisals of the event. The effectiveness of distraction and reappraisal may directly depend on the ability to inhibit mood-congruent content in WM, and difficulties with inhibition may result in rumination.

In a series of studies using a negative priming task, dysphoric participants, clinically depressed participants, and participants with a history of depressive episodes exhibited reduced inhibition of negative but not positive material (Joormann & Gotlib, 2010; Goeleven, De Raedt, Baert, & Koster, 2006). These findings suggest that depression involves difficulties keeping irrelevant emotional information from *entering* WM. Other studies suggest that depression is also associated with difficulties *removing* previously relevant negative material from WM (Joormann & Gotlib, 2008). Difficulties inhibiting the processing of negative material that was, but is no longer, relevant might explain why people respond to negative mood states and negative life events with recurring, uncontrollable, and unintentional negative thoughts.

It is likely that deficits in cognitive control not only affect people's ability to disengage attention from irrelevant material but also make it difficult for them intentionally to forget unwanted material. Investigators have consistently documented such memory biases in depression. Deficits in inhibition have been tested in directed forgetting tasks, in which participants are instructed to forget previously studied material at some point during the experiment. Later, however, recall is tested, of both material that was to be remembered and material that was to be forgotten. Using emotional material, Power, Dalgleish, Claudio, Tata, and Kentish (2000) reported that depressed participants showed less forgetting of negative material. Using a similar task with depressed participants, Joormann, Hertel, LeMoult, and Gotlib (2009) replicated these difficulties in the intentional forgetting of negative material. A subset of participants, however, was provided a strategy to improve suppression by using thought substitutes. In this condition depressed participants were able to inhibit and forget negative words. This finding has intriguing implications for interventions.

Deficits in disengaging attention from negative material and difficulties controlling the content of WM may affect emotion regulation in various ways. An inability to appropriately expel mood-congruent items from WM as they become irrelevant may result in rumination. Deficits in cognitive control may also lead to difficulties attending to and processing new information, thereby hindering the use of more adaptive strategies. Effective reappraisal, for example, depends on a person's ability to override (automatic) interpretation biases that lead to unwanted appraisals of the emotion-eliciting cues. Replacing automatic appraisals with alter-

native evaluations of the situation requires cognitive control. Finally, deficits in cognitive control can make it difficult to access mood-incongruent material. People frequently recruit pleasant memories to repair sad mood. Difficulties inhibiting salient but irrelevant thoughts could therefore reduce the use of more effective emotion regulation strategies and/or render these strategies less effective. Few studies so far have investigated the association between inhibition and the use and effectiveness of emotion regulation strategies such as reappraisal and distraction. Numerous findings, however, support the proposition that inhibition deficits are related to the use of increased rumination. For example, Joormann (2006) reported a correlation between rumination and deficits in cognitive inhibition, as assessed by negative priming, and Joormann and Gotlib (2008) found a correlation between rumination and the ability to remove irrelevant negative material from WM. Finally, Joormann and Gotlib (2010) reported that an inability to inhibit the processing of negative material was related to an increased likelihood of rumination and a decreased likelihood of using reappraisal, in both healthy and depressed participants.

Summary and Future Directions

Given that sustained affective states are the hallmark feature of mood disorders, basic research on the regulation of mood states and emotions provides important information for an improved understanding of risk factors for the development and maintenance of MDD and BD. Research on emotion regulation may thereby contribute to the development of improved prevention and intervention efforts. Our review of the literature provides evidence of the importance of both aspects of emotion regulation presented at the beginning of this chapter: individual differences in the habitual use of specific emotion regulation strategies and disorder-related difficulties in the implementation of adaptive strategies. Specifically, MDD is associated with frequent use of rumination and less use of reappraisal. BD is also related to increased rumination use, although, interestingly, in response to positive and negative events. Results for other emotion regulation strategies were either missing or inconsistent, so more work is needed to clarify further the role of suppression–avoidance and acceptance in both disorders, as well as the role of reappraisal in BD. Given methodological issues associated with the use of self-report in research on emotion regulation, future studies should include additional measures such as experience sampling or experimental manipulations of mood to understand strategy selection better in affective disorders.

Fewer studies have examined difficulties in the implementation of adaptive strategies. Initial findings suggest that whereas depressed participants can use distraction effectively, reappraisal is more challenging. Importantly, studies suggest that depression is associated with difficulties using positive material to offset negative affect. Whereas some studies suggest that these difficulties in strategy implementation do not just characterize currently depressed participants, more research is needed on the question of whether we find similar deficits prior to the first episode of the disorder, and whether these difficulties predict future episodes of depression or mania. Specifically, longitudinal studies in high-risk samples are needed to answer the important question of whether these difficulties in emotion regulation are indeed a risk factor for the onset of the disorder or simply an epiphenomenon of mood disorders.

We further examined cognitive processes that may underlie difficulties in emotion regulation. Biased processing in depression may affect the initial appraisal of an emotion-eliciting event, as well as the ability to reappraise. Cognitive control deficits may affect response-focused strategies such as distraction and online reappraisal (see Figure 25.1). Whereas both strategies require cognitive control, online reappraisal is frequently seen as the more effortful strategy, which may explain why distraction works but reappraisal may fail in depressed participants. Importantly, recent work by Sheppes, Scheibe, Suri, and Gross (2011) has shown that reappraisal becomes more difficult when affect is intense, and that non-disordered participants prefer distraction over reappraisal when dealing with intense emotions. As discussed, mood disorders are associated with deficits in cognitive control, and emotion regulation in these disorders frequently requires modifying intense affective states. Indeed, Sheppes and Gross

(2011) have pointed out that engaging emotion regulation strategies at a later point in the emotion episode typically results in the regulation of more intense affect. Mood disorders probably involve many attempts at regulation of high-intensity affect at later points in time, because they frequently require the regulation of global moods with unclear or unknown antecedents. It is therefore not surprising that reappraisal is difficult to implement in mood disorders. Future research should focus more on identifying differences between emotion and mood regulation, and on implications of these differences for affective disorders.

Most studies on emotion regulation have focused on the down-regulation of negative affect, but our review of the literature shows that the processing of mood-congruent material and the regulation of positive affect may also play an important role in mood disorders, specifically in BD. Difficulties savoring positive events and recalling positive material when feeling sad, however, may also play a role in MDD. Therefore, the down-regulation of negative affect seems a very narrow focus, and studies in BD and depression should pay more attention to the regulation of positive affect. In addition, at numerous points during the chapter, we have recommended flexibility in the use of emotion regulation strategies as an important topic for future studies. Very few studies examining affective disorders have assessed more than one strategy, and it is currently not known whether the primary problem in emotion regulation in these disorders is inflexibility or of habitual use of a specific strategy, or difficulties implementing a strategy.

Finally, there are some important treatment implications of research on emotion regulation in mood disorders. Cognitive-behavioral therapy has focused on the identification and modification of maladaptive cognitions, and recent additions to this intervention have started to focus more systematically on training reappraisal skills and preventing emotional avoidance (Campbell-Sills & Barlow, 2007). If inflexibility is a problem, it may be helpful to develop a broader repertoire of strategies and to identify habitual emotion regulation strategies. Given the earlier discussion of cognitive processes that underlie emotion regulation, recent studies on cognitive bias modification and cognitive control training seem particularly promising. Most of these studies have involved anxiety disorders, but at least two recent articles provide encouraging data to indicate that training to modify attention biases may also apply to depression (Wells & Beevers, 2010; Baert, Koster, & De Raedt, 2011). Other studies have focused on modifying memory and interpretation biases, but these trainings have not yet been tested with depressed participants (Raes, Williams, & Hermans, 2009). Of special importance is a recent study that showed improvement in cognitive control after training in executive control. Specifically, cognitive control training yielded transferable gains to improve control over affective stimuli (Schweizer, Hampshire, & Dalgleish, 2011). A similar training showed effects on thought control over intrusive memories (Bomyea & Amir, 2011). These findings are exciting and represent first steps toward a more comprehensive model of how cognitive factors facilitate or hinder emotion regulation, thereby affecting individuals' vulnerability to experience depressive episodes.

At this point, however, a lot of questions remain unanswered. Why do people diagnosed with affective disorders prefer certain emotion regulation strategies, and are they aware of these preferences? How do people learn to use these strategies, and how can we help them unlearn them? Are individual differences in attention, interpretation, memory, and cognitive control indeed related to the selection and effectiveness of regulation strategies? Does modifying cognitive biases and cognitive control affect emotional responding, stress reactivity, and emotion regulation? Are the outlined associations among depression, cognitive processes, and emotion regulation specific to depressive disorders or a feature of psychopathology in general? These and other questions should be the main focus of future studies in this exciting area of research. A deeper understanding of the ways that cognitive processes help and hinder emotion regulation may allow us to better address some of the difficulties depressed individuals encounter in developing new and more flexible ways of thinking, a central goal of cognitive therapy. Understanding the relation of cognitive processes and emotion regulation may also help us understand how we can get these new ways of thinking to "stick" and lead to

permanent changes in emotional response, facilitating the use of these new skills in the face of stress.

References

Aldao, A., & Nolen-Hoeksema, S. (2012). When are adaptive strategies most predictive of psychopathology? *Journal of Abnormal Psychology, 121*(1), 276–281.

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*(2), 217–237.

Alloy, L. B., Abramson, L. Y., Flynn, M., Liu, R. T., Grant, D. A., Jager-Hyman, S., et al. (2009). Self-focused cognitive styles and bipolar spectrum disorders: Concurrent and prospective associations. *International Journal of Cognitive Therapy, 2*(4), 354–372.

Baert, S., Koster, E. H. W., & De Raedt, R. (2011). Modification of information processing biases in emotional disorders: Clinically relevant developments in experimental psychopathology. *International Journal of Cognitive Therapy, 4*(2), 208–222.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression: A treatment manual*. New York: Guilford Press.

Bomyea, J., & Amir, N. (2011). The effect of an executive functioning training program on working memory capacity and intrusive thoughts. *Cognitive Therapy and Research, 35*(6), 529–535.

Bonanno, G. A., Papa, A., Lalande, K., Westphal, M., & Coifman, K. (2004). The importance of being flexible. *Psychological Science, 15*, 482–487.

Campbell-Sills, L., & Barlow, D. H. (2007). Incorporating emotion regulation into conceptualizations and treatments of anxiety and mood disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 542–559). New York: Guilford Press.

Campbell-Sills, L., Barlow, D. H., Brown, T. A., & Hofmann, S. G. (2006). Acceptability and suppression of negative emotion in anxiety and mood disorders. *Emotion, 6*(4), 587–595.

D'Avanzato, C., Joormann, J., Siemer, M., & Gotlib, I. H. (2013). Emotion regulation in depression and anxiety: Examining diagnostic specificity and stability. *Cognitive Therapy and Research*. [E-publication prior to print]

Ehring, T., Tuschen-Caffier, B., Schnülle, J., Fischer, S., & Gross, J. J. (2010). Emotion regulation and vulnerability to depression: Spontaneous versus instructed use of emotion suppression and reappraisal. *Emotion, 10*(4), 563–572.

Feldman, G. C., Joormann, J., & Johnson, S. L. (2008). Responses to positive affect: A self-report measure of rumination and dampening. *Cognitive Therapy and Research, 32*(4), 507–525.

Garnefski, N., & Kraaij, V. (2006). Cognitive Emotion Regulation Questionnaire—development of a short 18-item version (CERQ-short). *Personality and Individual Differences, 41*(6), 1045–1053.

Garnefski, N., Legerstee, J., Kraaij, V., van den Kommer, T., & Teerds, J. (2002). Cognitive coping strategies and symptoms of depression and anxiety: A comparison between adolescents and adults. *Journal of Adolescence, 25*(6), 603–611.

Goeleven, E., De Raedt, R., Baert, S., & Koster, E. H. W. (2006). Deficient inhibition of emotional information in depression. *Journal of Affective Disorders, 93*(1–3), 149–157.

Green, M. J., Lino, B. J., Hwang, E. J., Sparks, A., James, C., & Mitchell, P. B. (2011). Cognitive regulation of emotion in bipolar I disorder and unaffected biological relatives. *Acta Psychiatrica Scandinavica, 124*(4), 307–316.

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224–237.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*(2), 348–362.

Gruber, J. (2011). Can feeling too good be bad?: Positive emotion persistence (PEP) in bipolar disorder. *Current Directions in Psychological Science, 20*(4), 217–221.

Gruber, J., Eidelman, P., Johnson, S. L., Smith, B., & Harvey, A. G. (2011). Hooked on a feeling: Rumination about positive and negative emotion in inter-episode bipolar disorder. *Journal of Abnormal Psychology, 120*(4), 956–961.

Gruber, J., Harvey, A. G., & Gross, J. J. (2012). When trying is not enough: Emotion regula-

tion and the effort–success gap in bipolar disorder. *Emotion, 12*(5), 997–1003.

Gruber, J., Harvey, A. G., & Johnson, S. L. (2009). Reflective and ruminative processing of positive emotional memories in bipolar disorder and healthy controls. *Behaviour Research and Therapy, 47*(8), 697–704.

Hallion, L. S., & Ruscio, A. M. (2011). A meta-analysis of the effect of cognitive bias modification on anxiety and depression. *Psychological Bulletin, 137*(6), 940–958.

Hasher, L., Zacks, R. T., & May, C. P. (1999). Inhibitory control, circadian arousal, and age. In D. Gopher & A. Koriat (Eds.), *Attention and performance XVII: Cognitive regulation of performance: Interaction of theory and application* (pp. 653–675). Cambridge, MA: MIT Press.

John, O. P., & Gross, J. J. (2004). Healthy and unhealthy emotion regulation: Personality processes, individual differences, and life span development. *Journal of Personality, 72*(6), 1301–1333.

Johnson, S. L. (2005). Mania and dysregulation in goal pursuit: A review. *Clinical Psychology Review, 25*(2), 241–262.

Johnson, S. L., McKenzie, G., & McMurrich, S. (2008). Ruminative responses to negative and positive affect among students diagnosed with bipolar disorder and major depressive disorder. *Cognitive Therapy and Research, 32*(5), 702–713.

Joormann, J. (2006). Differential effects of rumination and dysphoria on the inhibition of irrelevant emotional material: Evidence from a negative priming task. *Cognitive Therapy and Research, 30*(2), 149–160.

Joormann, J. (2010). Cognitive inhibition and emotion regulation in depression. *Current Directions in Psychological Science, 19*(3), 161–166.

Joormann, J., & Gotlib, I. H. (2007). Selective attention to emotional faces following recovery from depression. *Journal of Abnormal Psychology, 116*(1), 80–85.

Joormann, J., & Gotlib, I. H. (2008). Updating the contents of working memory in depression: Interference from irrelevant negative material. *Journal of Abnormal Psychology, 117*(1), 182–192.

Joormann, J., & Gotlib, I. H. (2010). Emotion regulation in depression: Relation to cognitive inhibition. *Cognition and Emotion, 24*(2), 281–298.

Joormann, J., Hertel, P. T., LeMoult, J., & Gotlib, I. H. (2009). Training forgetting of negative material in depression. *Journal of Abnormal Psychology, 118*(1), 34–43.

Joormann, J., Siemer, M., & Gotlib, I. H. (2007). Mood regulation in depression: Differential effects of distraction and recall of happy memories on sad mood. *Journal of Abnormal Psychology, 116*(3), 484–490.

Kashdan, T. B., & Rottenberg, J. (2010). Psychological flexibility as a fundamental aspect of health. *Clinical Psychology Review, 30*(4), 467–480.

Kessler, R. C., & Wang, P. S. (2009). Epidemiology of depression. In I. H. Gotlib & C. L. Hammen (Eds.), *Handbook of depression* (2nd ed., pp. 5–22). New York: Guilford Press.

Kim, S., Yu, B. H., Lee, D. S., & Kim, J.-H. (2012). Ruminative response in clinical patients with major depressive disorder, bipolar disorder, and anxiety disorders. *Journal of Affective Disorders, 136*(1–2), e77–e81.

Lam, D., & Wong, G. (1997). Prodromes, coping strategies, insight and social functioning in bipolar affective disorders. *Psychological Medicine, 27*(5), 1091–1100.

Lawson, C., MacLeod, C., & Hammond, G. (2002). Interpretation revealed in the blink of an eye: Depressive bias in the resolution of ambiguity. *Journal of Abnormal Psychology, 111*(2), 321–328.

Liverant, G. I., Brown, T. A., Barlow, D. H., & Roemer, L. (2008). Emotion regulation in unipolar depression: The effects of acceptance and suppression of subjective emotional experience on the intensity and duration of sadness and negative affect. *Behaviour Research and Therapy, 46*(11), 1201–1209.

Liverant, G. I., Kamholz, B. W., Sloan, D. M., & Brown, T. A. (2011). Rumination in clinical depression: A type of emotional suppression? *Cognitive Therapy and Research, 35*(3), 253–265.

Lyubomirsky, S., & Nolen-Hoeksema, S. (1993). Self-perpetuating properties of dysphoric rumination. *Journal of Personality and Social Psychology, 65*(2), 339–349.

Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annual Review of Clinical Psychology, 1*(1), 167–195.

McRae, K., Jacobs, S. E., Ray, R. D., John, O. P., & Gross, J. J. (2012). Individual differences in reappraisal ability: Links to reappraisal frequency, well-being, and cognitive control. *Journal of Research in Personality, 46*(1), 2–7.

Mogg, K., Bradley, B. P., Williams, R., &

Mathews, A. (1993). Subliminal processing of emotional information in anxiety and depression. *Journal of Abnormal Psychology, 102*(2), 304–311.

Nezlek, J. B., & Kuppens, P. (2008). Regulating positive and negative emotions in daily life. *Journal of Personality, 76*(3), 561–579.

Nolen-Hoeksema, S. (2000). Further evidence for the role of psychosocial factors in depression chronicity. *Clinical Psychology: Science and Practice, 7*(2), 224–227.

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3*(5), 400–424.

Papageorgiou, C., & Wells, A. (2001). Positive beliefs about depressive rumination: Development and preliminary validation of a self-report scale. *Behavior Therapy, 32*(1), 13–26.

Power, M. J., Dalgleish, T., Claudio, V., Tata, P., & Kentish, J. (2000). The directed forgetting task: Application to emotionally valent material. *Journal of Affective Disorders, 57*(1–3), 147–157.

Raes, F., Smets, J., Nelis, S., & Schoofs, H. (2012). Dampening of positive affect prospectively predicts depressive symptoms in non-clinical samples. *Cognition and Emotion, 26*(1), 75–82.

Raes, F., Williams, J. M. G., & Hermans, D. (2009). Reducing cognitive vulnerability to depression: A preliminary investigation of MEmory Specificity Training (MEST) in inpatients with depressive symptomatology. *Journal of Behavior Therapy and Experimental Psychiatry, 40*(1), 24–38.

Ray, R. D., McRae, K., Ochsner, K. N., & Gross, J. J. (2010). Cognitive reappraisal of negative affect: Converging evidence from EMG and self-report. *Emotion, 10*(4), 587–592.

Richards, J. M., & Gross, J. J. (2000). Emotion regulation and memory: The cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology, 79*(3), 410–424.

Schweizer, S., Hampshire, A., & Dalgleish, T. (2011). Extending brain-training to the affective domain: Increasing cognitive and affective executive control through emotional working memory training. *PLoS ONE, 6*(9), e24372.

Sheppes, G., Catran, E., & Meiran, N. (2009). Reappraisal (but not distraction) is going to make you sweat: Physiological evidence for self-control effort. *International Journal of Psychophysiology, 71*(2), 91–96.

Sheppes, G., & Gross, J. J. (2011). Is timing everything? Temporal considerations in emotion regulation. *Personality and Social Psychology Review, 15*(4), 319–331.

Sheppes, G., & Meiran, N. (2007). Better late than never?: On the dynamics of online regulation of sadness using distraction and cognitive reappraisal. *Personality and Social Psychology Bulletin, 33*(11), 1518–1532.

Sheppes, G., Scheibe, S., Suri, G., & Gross, J. J. (2011). Emotion-regulation choice. *Psychological Science, 22*(11), 1391–1396.

Siemer, M. (2005). Mood-congruent cognitions constitute mood experience. *Emotion, 5*(3), 296–308.

Siemer, M., & Reisenzein, R. (2007). The process of emotion inference. *Emotion, 7*, 1–20.

Solomon, A. (2001). *The noonday demon*. New York: Scribner.

Teasdale, J. D. (1988). Cognitive vulnerability to persistent depression. *Cognition and Emotion, 2*(3), 247–274.

Thomas, J., & Bentall, R. P. (2002). Hypomanic traits and response styles to depression. *British Journal of Clinical Psychology, 41*(3), 309–313.

Thomas, J., Knowles, R., Tai, S., & Bentall, R. P. (2007). Response styles to depressed mood in bipolar affective disorder. *Journal of Affective Disorders, 100*(1–3), 249–252.

Troy, A. S., Wilhelm, F. H., Shallcross, A. J., & Mauss, I. B. (2010). Seeing the silver lining: Cognitive reappraisal ability moderates the relationship between stress and depressive symptoms. *Emotion, 10*(6), 783–795.

Urry, H. L. (2009). Using reappraisal to regulate unpleasant emotional episodes: Goals and timing matter. *Emotion, 9*(6), 782–797.

Watkins, E., & Moulds, M. (2005). Distinct modes of ruminative self-focus: Impact of abstract versus concrete rumination on problem solving in depression. *Emotion, 5*(3), 319–328.

Wells, T. T., & Beevers, C. G. (2010). Biased attention and dysphoria: Manipulating selective attention reduces subsequent depressive symptoms. *Cognition and Emotion, 24*(4), 719–728.

Werner-Seidler, A., & Moulds, M. L. (2012). Mood repair and processing mode in depression. *Emotion, 12*(3), 470–478.

CHAPTER 26

Emotion Regulation in Substance Use Disorders

Hedy Kober

Have you ever had coffee or tea? A glass of wine? Smoked even a single cigarette? Virtually all adults report consuming psychoactive drugs[1] at some point in their lives, suggesting that casual drug use is quite common (Substance Abuse and Mental Health Services Administration [SAMHSA], 2011). On the other end of the drug use spectrum, substance use disorders (SUDs; or addictions) are complex illnesses, encompassing a host of severe negative physical, economic, and social consequences, and contributing to worldwide disability. With a lifetime prevalence of 35.3% in the general population, individuals with SUDs constitute a relatively small proportion of casual drug users, yet they also represent the most prevalent and costly of psychiatric disorders (National Institute of Mental Health [NIMH], 2007; SAMHSA, 2011).

Defined as "a problematic pattern of drug use, leading to clinically significant impairment or distress" (American Psychiatric Association, 2013, p. 481), SUDs are both personally and socially devastating in that they are often chronic and can severely impair even basic life functioning. In the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5), SUDs are characterized by the presence of symptoms including tolerance, withdrawal, continued use despite wishes to stop, continued use despite known negative consequences, and importantly, a loss of regulatory control over drug cravings, as well as further drug use. As such, loss of regulatory control is a key feature of SUDs. The addition of *drug craving* (strong desire for drugs) as a diagnostic criterion for SUDs in DSM-5 emerged from a wealth of accumulated research over the last decade directly linking craving to drug use and *relapse* (return to drug use following abstinence; e.g., Shiffman et al., 2013; see later sections for additional discussion). This suggests that craving is also a key feature in SUDs, and that regulation of craving is a specific form of emotion regulation that can directly reduce drug use.

This chapter focuses on the crucial and complex role of emotion regulation in SUDs (see Figure 26.1 for a schematic summary). In the first section, I discuss the role of acute drug intoxication as a means of emotion regulation, arguing specifically that people use drugs in part to regulate their current emotional state. This may include increasing positive affect, ameliorating a preexisting negative state, or decreasing craving. In the next section, I explore the role of emotion dysregulation in SUDs, both as a possible cause for and a possible consequence of drug use. In this section, I make several specific arguments. First, I argue that emotion dysregulation in childhood and adolescence may be an early risk factor and/or distal causal factor in the later development of SUDs. Second, I argue that an inability to effectively regulate emotions in specific moments may

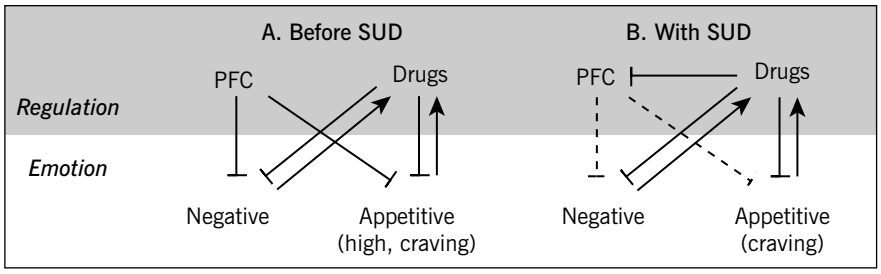

FIGURE 26.1. A simplified model of emotion regulation in SUDs. *Panel A:* Before SUDs. Prefrontal cortex (PFC) and drugs can both serve to regulate emotion. It is thought that PFC implements regulation over negative emotion and craving (indicated by downward blunted arrows). In turn, unregulated negative emotion and craving are associated with increased drug use (upward arrows). Here I propose that drugs can be seen as a form of emotion regulation as well (indicated by downward blunted arrows), increasing feelings of high, and decreasing negative emotion and craving. In this context, deficient emotion regulation or PFC control may serve as risk factors for SUDs. *Panel B:* After development of SUDs. Chronic drug use affects PFC (indicated by blunted arrow), diminishing its ability to regulate negative emotion, as well as drug craving (dashed downward blunted arrows). In turn, unregulated negative emotion and craving further lead to increased drug use (upward arrows). Drug use itself continues to regulate both negative emotion and drug craving (though perhaps less effectively). This results in a vicious cycle of reduced PFC-based emotion regulation, negative affect, craving, and increased drug use. Therefore, treatments for SUDs often focus on enhancing emotion regulation skills, especially regulation of craving, which has been linked to reduced drug use.

be a proximal causal factor for instances of drug use in individuals who are already suffering from SUDs. Third, I posit that SUDs are marked by deficits in regulation of a specific appetitive state, namely, drug craving, which is at the core of these disorders. I then review evidence that suggests differences in the structure and function of the prefrontal cortex (PFC) may be the neural mechanisms underlying emotion dysregulation in SUDs. This section further highlights that although some PFC abnormalities may precede drug use, the long-term effect of chronic drug use on PFC may further impair emotion regulation in SUDs. In this way, drug use may lead to further emotion dysregulation. The chapter concludes with a section on treatments for SUDs, many of which focus on increasing emotion regulation skills geared specifically toward regulation of craving as means of reducing substance use.

Drug Use as Emotion Regulation

Drugs can regulate emotion by pharmacologically altering one's current state. For example, although the exact pharmacological profiles of individual drugs differ, and these differences have both theoretical and neurobiological implications (e.g., Badiani, Belin, Epstein, Calu, & Shaham, 2011), many drugs are ultimately described as euphoric, increasing positive emotion (Jaffe & Jaffe, 1989). In human laboratory experiments, self-administration of drugs, including alcohol, methamphetamine, cocaine, and marijuana, significantly increase feelings of "high" and "good drug effects" (e.g., Hart, Ward, Haney, Foltin, & Fischman, 2001; see Figure 26.1A). Consistently, it is has been proposed that these positive effects of drugs lead to positive reinforcement and increase the likelihood of future drug use (Kober, Turza, & Hart, 2009). Furthermore, drug users often develop positive expectancies regarding drug use (e.g., "If I drink, I will feel good") that are associated with increased drug use and increased risk of developing SUDs (e.g., Jones, Corbin, & Fromme, 2001).

In addition to increasing positive emotion, various drugs are known to alleviate negative emotional states, including anxiety (e.g., alcohol, and anxiolytic medication such as Valium and Xanax), sadness and

depression (e.g., stimulants such as cocaine and amphetamines), and pain (e.g., heroin, morphine, and other synthetic prescription opiates such as Vicodin). Consistently, it has been proposed that these negativity-reducing effects of drugs lead to negative reinforcement, thus increasing the likelihood of future drug use (Koob & Le Moal, 2008). This idea was initially popularized by the "self-medication hypothesis" proposed by Khantzian (1985), which has two main components: (1) Unpleasant affective states predispose individuals to drug use, and (2) the choice of drug is not random; rather, it is the nature of the drug's effects in ameliorating the preexisting negative state that renders a particular drug more or less reinforcing. In other words, those with a particular predisposition to negative affect states are more likely to develop an SUD for a drug that reverses those particular affective states. To illustrate, Khantzian suggested that individuals with strong rage and aggression use opiate drugs to regulate these emotions. In contrast, individuals with preexisting depression and melancholy develop cocaine use disorders due to cocaine's ability to relieve these symptoms. The self-medication hypothesis is consistent with patients' reports that "they got hooked not because they had taken the drug, but because they were not normal before in such a way that the drugs were . . . not the problem but a solution" (Le Moal, 2009, p. 542). It is further consistent with the observation that the expectancy that drugs will alleviate negative affect (e.g., "Drinking will calm me down") is associated with increased drug use and increased risk for SUDs (Jones et al., 2001).

Although the self-medication hypothesis has been challenged, several lines of evidence support the hypothesis that drug use serves to regulate negative emotion. First, SUDs frequently co-occur with a number of other psychiatric disorders, especially mood and anxiety disorders. Moreover, preexisting psychiatric diagnoses increase the likelihood of an individual to subsequently develop an SUD (e.g., Kessler et al., 2005). This suggests that individuals who already experience difficult emotions are more likely to seek and use drugs, and to develop problematic habits of drug use that presumably ameliorate their affective symptoms. A related point is that treatment for such comorbid disorders frequently reduces drug use (Nunes & Levin, 2004). Second, and similarly, those with chronic pain are far more likely to develop SUDs relative to the general, pain-free population, especially to pain-reducing drugs such as opiates (Morasco et al., 2011).

Third, even normal-range trait levels of negative affect are related to drug use. For example, trait depression and neuroticism correlate negatively with time to relapse in cigarette smokers (Gilbert, Crauthers, Mooney, McClernon, & Jensen, 1999), while trait levels of anger and anxiety correlate with craving to drink in alcoholics (Litt, Cooney, & Morse, 2000). Fourth, negative affective states are known triggers for craving in the context of both casual and problematic substance use (e.g., Shiffman, Paty, Gnys, Kassel, & Hickcox, 1996). This phenomenon ranges from the common epithet "I had such a hard day, I need a beer or a stiff drink" to instances of relapse to drug use after experiencing a strong life stressor (e.g., death in family). Indeed, it has been well documented that both naturally occurring and experimentally induced negative affect and stress increase drug craving, drug use, and relapse (e.g., Sinha & Li, 2007).

Finally, drug use also serves to regulate the experience of craving, which is one of the most common motivators for drug use (Childress et al., 1993; Shiffman et al., 2013). That is, individuals with SUDs use drugs to temporarily alleviate their experience of craving, thus generating a vicious cycle of increasing craving and use. Taken together, the evidence reviewed in this section suggests that drug taking can be a form of emotion regulation. Specifically, the acute effects of drugs may regulate preexisting emotions in both casual and problem drug users, including increasing positive emotion, decreasing negative emotion, and decreasing craving for drugs themselves (see Figure 26.1A).

Emotion (dys)Regulation Is a Causal Factor in SUDs

Although many people casually use drugs and alcohol, only a small percentage develop SUDs, highlighting the need to identify risk and causal factors for the initiation, develop-

ment, and maintenance of these severe disorders. Of course, because SUDs are complex disorders, they are likely caused and maintained by an intricate combination of factors, including genetic, cognitive, behavioral, individual-difference, and environmental variables, that likely interact across multiple levels of analysis. At this time, emotion regulation abilities are already emerging as one important contributor in the etiology and maintenance of SUDs, although in the next decade it is likely that larger longitudinal studies will allow us to identify additional factors.

Emotion (dys)Regulation as an Early Risk Factor

As reviewed below, SUDs are frequently associated with emotion regulation deficits. The specific question here is: Do these deficits precede the development of the disorder so that they may be considered a risk factor? The answer appears to be yes. Beginning with the classic "marshmallow test" experiments in the 1960s by Mischel and colleagues, it has been proposed that the ability to delay gratification, and regulate emotions like desire, is crucial to children's developmental trajectories (for review, see Mischel, Ayduk, Berman, Casey, Gotlib, et al., 2011 and Luerssen & Ayduk, this volume). In these studies, preschool children were typically presented with a tasty treat and told that they could have it now, or alternatively, wait to receive two treats at a later time—if they could delay gratification. In his seminal work, Mischel reported that children vary in their ability to delay gratification, ranging from not being able to wait at all to waiting as long as the experimenter allowed (and using a variety of spontaneous strategies to facilitate delay). In his follow-up work, Mischel (2011) reported that those preschool children who were able to delay gratification the longest (by waiting for a larger treat rather than indulging immediately in a smaller treat) later achieved higher Standard Achievement Test (SAT) scores, had better social-cognitive and emotional coping in adolescence, and importantly, were least likely to use crack cocaine in adulthood. This body of work highlights how individual differences in emotion regulation (which manifest as early as preschool) may predate the development of SUDs and could therefore be conceptualized as a risk factor predicting illness onset.

In the years since this work was published, additional data has accumulated to further suggest that poor self-control[2] in childhood is indeed a risk factor for drug use and the development of SUDs. For example, Moffitt and colleagues (2011) followed 1,000 children from birth to age 32. In childhood, participants were assessed on various self-control measures related to emotion regulation, including emotional lability, frustration tolerance, and persistence. The authors report that individual differences in self-control were significantly predictive of adult health outcomes, including substance use and dependence, as much as 30 years later. Importantly, the contribution of self-control factors was distinct from the contribution of intelligence, social class, and other family-life variables. Strikingly, the highest and lowest one-fifth of the sample on measured self-control were associated with a prevalence of 3 and 10%, respectively, of polysubstance dependence in adulthood.

In addition, in childhood, the related construct of *trait impulsivity*—the tendency to act without thought or regard for consequences—has been repeatedly associated with the development of SUDs in later adolescence and adulthood (see Ivanov, Newcorn, Morton, & Tricamo, 2011; Verdejo-García, Lawrence, & Clark, 2008, for reviews). Furthermore, longitudinal studies suggest that children who suffer from childhood disorders such as attention-deficit/hyperactivity disorder and conduct disorder, which are associated with poor emotional/behavioral regulation and impulsivity, are far more likely to use drugs and to receive an SUD diagnosis by late adolescence or young adulthood (e.g., August et al., 2006). It has also been suggested that the association between childhood disruptive behavior and adolescent-onset substance use may be mediated by early deficits in emotion regulation and inhibitory control (Ivanov et al., 2011). A similar construct used by Tarter and colleagues (2003), termed *neurobehavioral disinhibition*, is indexed by measures of emotion regulation, executive cognitive functioning, and behavior control. This construct distinguishes between 10- to 12-year-old boys who are at

low vs. high risk for development of SUDs (determined by parental SUD diagnosis). In addition, this construct was found to predict substance use at age 16, as well as SUDs in early adulthood.

The mechanisms by which early emotion dysregulation leads to later SUDs are a target of current investigation. One prevailing hypothesis is that emotion regulation abilities (and cognitive control more generally) depend on the function of PFC regions (see Ochsner & Gross, this volume; Johnstone & Walter, this volume) that are not yet fully developed in children and adolescents (see Riediger & Klipker, this volume). Indeed, adolescence represents a period of both reduced emotion regulation abilities (Silvers et al., 2012) as well as substantial neural development (Giedd et al., 1999). Specifically, regions of lateral PFC have been found to be relatively hypoactive during emotion-related tasks in adolescents as compared to adults (e.g., Pfeifer, Lieberman, & Dapretto, 2007), with regulation-related activation in this area increasing with age (McRae et al., 2012).

Given this developmental trajectory, emotion dysregulation in adolescence may contribute to SUD risk via two parallel routes. First, immature emotion regulation capacities in adolescence may result in higher levels of stress and negative emotion, which has been shown to lead to the initiation of drug use in animal models (e.g., Haney, Maccari, Le Moal, Simon, & Vincenzo Piazza, 1995) and in human adults (see Sinha & Li, 2007, for review). Second, self-regulation failures in adolescence may underlie increased impulsivity and risk-taking behaviors that may also lead to initiation of drug use (e.g., Ivanov et al., 2011). Ultimately, the developmental trajectory of self-regulatory function suggests that at least some adolescents may be less able to recruit the neural circuitry needed to regulate their emotions optimally and to ultimately avoid substance use.

The idea that adolescents as a group may in fact be less able to recruit the necessary neurocircuitry to regulate emotions and avoid substance use—along with the observation that individual differences in the ability to do so are predictive of future substance use—is especially important, because adolescence is a period of heightened risk taking (SAMHSA, 2011) and peer influence (Steinberg, 2005), both of which expose adolescents to drugs. Thus, increased exposure to drugs, coupled with increased emotional reactivity and decreased regulatory capacities (rooted in ongoing brain development), make adolescence a particularly vulnerable period for substance use.

Indeed, drug use is most often initiated in adolescence. For example, 82.4% of first uses of alcohol occur in individuals under the age of 21 (the legal drinking age), and 58.8% of smokers had their first cigarette under the age of 18 (SAMHSA, 2011). These early use statistics are especially important, because earlier age of onset is associated with higher rates of SUDs and worse outcomes. For example, those who initiated alcohol use prior to age 14 are more than four times more likely to receive an SUD diagnosis in adulthood (16.2 vs. 3.8%; similar rates are reported for illicit drugs). Similarly, earlier age of smoking onset predicts a higher number of cigarettes smoked in adulthood (Taioli & Wynder, 1991). Taken together, these data support the notion that emotion (dys) regulation is an early risk factor for SUDs. Next I discuss how emotion regulation may operate as an ongoing causal factor that may contribute to and exacerbate existing SUDs.

Emotion (dys)Regulation in Current SUDs

Several models of SUDs directly implicate deficient regulation as a key and primary motive for ongoing drug use and relapse, including the *relapse prevention model* (Marlatt & Witkiewitz, 2005), the *affective processing model* (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004), and the aforementioned *self-medication hypothesis* (Khantzian, 1985), among others. Indeed, whether or not emotion regulation deficits are a pre-existing risk factor for SUDs (as proposed in the previous section), those who currently suffer from SUDs frequently display such deficits, which may contribute to the clinical course of the disorder. Several lines of evidence support this association (Figure 26.1B for schematic illustration).

First, self-reported emotion regulation skills are lower in individuals with SUDs than in healthy controls (e.g., Fox, Hong, & Sinha, 2008). In addition, greater difficulties in regulating emotion is associated

with more drug use (e.g., Berking et al., 2011) possibly as a means of emotion regulation (Bonn-Miller, Vujanovic, & Zvolensky, 2008). Second, less effective styles of emotion regulation (e.g., suppression vs. reappraisal) are related to increased drug use (Fucito, Juliano, & Toll, 2010). Third, individual differences in negative affect have been repeatedly associated with drug use and relapse in clinical (e.g., Gamble et al., 2010), as well as laboratory studies (e.g., Sinha & Li, 2007). Fourth, as reviewed earlier, SUDs are highly comorbid with affective disorders, such as depression, which feature impaired regulation of negative affect as a key diagnostic feature (American Psychiatric Association, 2013). Furthermore, those with co-occurring symptoms of SUDs and affective disorders show significantly higher rates of relapse to drug use after treatment (e.g., Bradizza, Stasiewicz, & Paas, 2006) offering additional support for the link between emotion regulation deficits and SUDs.

Additional evidence links constructs related to emotion regulation and SUDs. For example, *emotional intelligence*—defined as the ability to be aware of emotions, identify emotions correctly, interpret them appropriately, and regulate them effectively—is inversely associated with alcohol and drug-related problems (Riley & Schutte, 2003). Moreover, emotional intelligence moderated the association between negative emotion and alcohol craving in alcohol-dependent individuals (Cordovil de Sousa Uva et al., 2010). Furthermore, a recent meta-analysis of this construct suggests that not only is it inversely related to smoking, alcohol, and drug use, but also that individual differences in particular components, namely, "identification of emotion" and "regulation of emotion," are particularly related to SUDs (Kun & Demetrovics, 2010). Similarly, *distress tolerance*—the ability to persist in goal-directed activity when experiencing psychological distress—is related to emotion regulation and is inversely associated with substance use frequency and SUDs (Marshall-Berenz, Vujanovic, & MacPherson, 2011), as well as SUD treatment dropout and eventual relapse (e.g., Daughters et al., 2005). In addition, impulsivity is reportedly higher in those with SUDs (see Verdejo-García et al., 2008, for review). Finally, it has been suggested that those with SUDs exhibit relative deficits in nonaffective forms of self-regulation and executive function, including working memory and response inhibition, which may also relate to PFC function (Goldstein & Volkow, 2011).

Emotion (dys)Regulation in Current SUDs: Regulation of Craving

In the previous section, I reviewed evidence suggesting that those with SUDs have difficulties regulating emotions. Notably, the evidence overwhelmingly centers on regulation of negative emotions. However, in the context of SUDs, it is critical to consider not only regulation of negative emotion but also an additional and very specific form of emotion regulation, namely, the regulation of craving.

Craving, defined here as "intense desire for drugs," has long been considered a key contributor to drug use (e.g., O'Brien, Childress, Ehrman, & Robbins, 1998). Although this view has been challenged (Perkins, 2009) substantial evidence links drug craving to drug-taking behavior, and it has been suggested that loss of control over cue-induced craving is at the root of compulsive drug taking (e.g., Goldstein & Volkow, 2011). For example, levels of reported craving predict drug use as well as relapse to drug taking following abstinence (e.g., Crits-Christoph et al., 2007; Epstein, Marrone, Heishman, Schmittner, & Preston, 2010; Galloway, Singleton, & the Methamphetamine Treatment Project Corporate Authors, 2008). Conversely, the ability to use various strategies to regulate craving is associated with lower craving (Kober, Kross, Mischel, Hart, & Ochsner, 2010; Kober, Mende-Siedlecki, et al., 2010; Westbrook et al., 2013) and lower drug use (O'Connell, Hosein, Schwartz, & Leibowitz, 2007). Further, the acquisition of strategies during cognitive-behavioral therapy (as discussed below) is associated with better long-term outcomes (Carroll, Nich, Frankforter, & Bisighini, 1999). Furthermore, the use of cognitive strategies to regulate craving both during and after treatment is associated with reduced craving and reduced relapse over time (O'Connell et al., 2007; Shiffman et al., 1996). These findings suggest that craving is a key motivator of substance use, and that effective regulation of craving is associated with lower drug use

and better outcomes for those with SUDs. This in turn suggests that regulation of craving is a specific form of regulation that is particularly important in the maintenance of drug use behavior in SUDs.

The neural mechanism by which regulation of craving operates to reduce drug use is a topic of current research. It has been shown previously that exposure to drug cues (e.g., drug-related images, movies, or paraphernalia) increases craving, as well as drug use (e.g., Shiffman et al., 2013). Furthermore, several meta-analyses have shown that such cue-induced craving is consistently associated with neural activity in a network of regions including the ventral striatum, the subgenual anterior cingulate, and the amygdala (e.g., Chase, Eickhoff, Laird, & Hogarth, 2011). These regions, which are thought to relate to learning, salience, and value encoding, previously have been associated with the acute effects of drugs. We have recently shown that when cigarette smokers use cognitive strategies in instances of craving (e.g., when they think about the long-term negative consequences of smoking), they report lower craving (Kober, Kross, et al., 2010), and exhibit lowered activity in the neural systems that underlie craving, such as the ventral striatum (Kober, Mende-Siedlecki, et al., 2010). Importantly, the regulation of craving with cognitive strategies is associated with concurrently increased activity in PFC regions including the dorsolateral (dlPFC) and ventral PFC—regions previously associated with regulation of negative emotion (see Ochsner & Gross, this volume). These findings have since been replicated with positron emission tomography in cocaine users (Volkow et al., 2010) and electrophysiological measurements in cigarette smokers (Littel & Franken, 2011).

Interestingly, we've recently shown that use of mindfulness-based strategies to regulate cue-induced craving is also associated with reductions in reported craving, and with reduced neural activity in "craving regions," including the subgenual cingulate. However, the use of such mindfulness-based strategies was not associated with concurrent increase in PFC activity (Westbrook et al., 2013). Taken together, these findings are consistent with the hypothesis that, across strategies, regulation of craving operates by reducing neural activity in regions that are thought to instantiate the experience of craving. Preliminarily, it further appears that the use of cognitive strategies to regulate craving may depend on increased activity in PFC but that mindfulness-based strategies may not, although additional data are required to confirm this pattern of results.

To summarize, this section has reviewed evidence suggesting that emotion regulation is implicated in SUDs, both as an early risk factor and as an ongoing motivator of drug use. For example, individual differences in emotion regulation and impulsivity during development are predictive of drug use initiation and SUDs. Furthermore, individuals with ongoing SUDs exhibit deficits in emotion regulation compared to healthy controls, and negative affect in such individuals is associated with instances of drug use. Importantly, most of the available evidence centers around regulation of negative emotion. However, regulation of craving is emerging as another form of regulation that is important in the etiology and maintenance of these disorders, and may constitute one key route by which targeted treatments can ameliorate SUDs, as discussed further below.

PFC in SUDs: Mechanism for Emotion (dys)Regulation?

In the prior sections I have reviewed evidence suggesting that PFC development may underlie the role of emotion dysregulation as a distal causal factor for development of SUDs in adolescence. But is this the neural mechanism that underlies general deficits in emotion regulation present in SUDs? Indeed, many current models of SUDs propose that the loss of control over craving and drug taking (as evident in the diagnostic criteria for the disorder) is a result of reduced or compromised PFC function (e.g., Everitt & Robbins, 2005; Goldstein & Volkow, 2011; Potenza, Sofuoglu, Carroll, & Rounsaville, 2011; Volkow, Wang, Fowler, & Tomasi, 2011). And this "PFC hypothesis" is consistent with the already-established link between cognitive control generally—and emotion regulation specifically—and the function of PFC in healthy adults (see Ochsner & Gross, this volume). However, neu-

roimaging studies directly testing emotion regulation abilities in SUDs are scarce. Nevertheless, in the following sections, I review findings that individuals with SUDs exhibit structural abnormalities in various PFC regions, as well as functional differences in studies of nonaffective forms of cognitive control (for a brief review of neuroimaging methodologies used in such studies, see Kober & DeLeone, 2011). Importantly, although some of these PFC abnormalities may precede the development of SUDs, I review evidence suggesting that chronic drug use is associated with both structural and functional changes in PFC. Such drug-induced changes, in turn, suggest that SUDs may also lead to decrements in PFC that may underlie further emotion dysregulation (Figure 26.1B).

In reviewing this evidence, it is important to note that the PFC is a very large structural division in the brain, and that different subregions within the PFC perform very different computations and subserve different functions—even within the general "cognitive control" framework (Miller & Cohen, 2001). However, at this stage, there are not yet sufficient data to make finer distinctions about the functional role of PFC subdivisions in SUDs, or to begin to speculate about the role each subregion might have in the neuropathology of these disorders. I hope that data collected in the next decade will allow us to answer such questions with far greater specificity than we can today.

Structural Differences in the PFC

Differences in brain structure have been reported between those with SUDs and healthy controls, using several different methodologies, especially in various subregions of PFC. For example, using voxel-based morphometry, cigarette smokers exhibited reduced PFC gray matter density compared to healthy controls, and PFC thickness was negatively correlated to reported smoking (measured in packs-per-year; Brody et al., 2004). In cocaine-dependent individuals, relatively reduced gray matter density in orbitofrontal cortex (OFC) and anterior cingulate cortex (ACC) was reported (Franklin et al., 2002). Similarly, lower thickness and volume were reported for other stimulant users in various prefrontal regions (e.g., Daumann et al., 2011) and in right ventrolateral PFC (vlPFC) specifically, where thickness was inversely correlated with drug craving (Tabibnia et al., 2011). In alcohol-dependent subjects compared to controls, lower gray matter volume was reported across the PFC (Fein, Di Sclafani, & Meyerhoff, 2002) and more specifically in the lateral and superior PFC and OFC (Durazzo et al., 2011) and medial and lateral PFC (Rando et al., 2011). In these latter studies, lower medial PFC volume was associated with more drinking posttreatment or shorter time to relapse. In addition, in some studies (but not all) PFC volume was inversely associated with cognitive control measures. For example, PFC gray matter volume correlated inversely with executive function measures in cocaine-dependent individuals (Fein et al., 2002).

Consistent with these gray matter findings in PFC, diffusion tensor imaging (DTI) measures of PFC white matter integrity distinguish between individuals with alcohol use disorders and controls (e.g., Pfefferbaum, Rosenbloom, Rohlfing, & Sullivan, 2009) and further differ between individuals who relapsed and those who sustained abstinence following treatment (Sorg et al., 2012). In cocaine-dependent participants, lower measurements of white matter integrity are consistently found in various PFC regions (e.g., Romero, Asensio, Palau, Sanchez, & Romero, 2010). Similar findings were reported in methamphetamine (Alicata, Chang, Cloak, Abe, & Ernst, 2009) and in opiate users (Bora et al., 2012; Liu et al., 2008).

Taken together, this body of structural neuroimaging work suggests that there are consistent anatomical differences between those with SUDs and healthy controls. A cautionary note here is that it is not yet clear what these differences mean. While it is tempting to interpret these differences as indicating impairment in individuals with SUDs, this link has not yet been consistently demonstrated. For instance, although reportedly lower than that in controls, cortical thickness and cognitive function are often within normal range in SUDs (see Hart, Marvin, Silver, & Smith, 2011, for extended discussion). Nevertheless, these differences are consistently reported across PFC and across types of SUDs. Furthermore, although indi-

vidual studies differ with respect to the localization of these differences (possibly due to sample characteristics, drug pharmacology, drug use patterns, and methodological and statistical differences), and only some studies find association with clinical outcomes, the PFC is repeatedly implicated, especially lateral portions. Reported differences are especially salient given the known role for PFC in emotion regulation and cognitive control in healthy adults. Taken together, these structural findings are consistent with the hypothesized mechanism by which PFC abnormalities may contribute to or underlie deficient emotion regulation in SUDs. However, future work could more directly link structural findings in PFC with emotion regulation in SUDs.

Functional Differences in PFC

Differences in measures of PFC function between individuals with SUDs and healthy controls have been consistently reported since the early days of functional neuroimaging. For example, using various forms of positron emission tomography (PET), it has been established that those with SUDs often exhibit relative reductions in "D2" type receptors of the neurotransmitter dopamine in striatum and PFC, with some evidence that these reductions persist even after months of abstinence (see Volkow et al., 2011, for review). PET measures of glucose metabolism have repeatedly shown decreased activity in OFC, ACC, and dlPFC. In stimulant users decreased activity is further related to relatively decreased D2 receptor availability in striatum (Volkow et al., 2011). Notably, in alcohol-dependent individuals, striatal D2 availability is linked to not only to PFC activity but also to self-reported alcohol craving, suggesting that all three processes may be functionally related (i.e., OFC function, D2 receptor availability, and craving; Heinz et al., 2005).

More recently, several studies have specifically investigated brain function during performance of non-affective cognitive control tasks comparing individuals with SUDs and healthy controls, typically with functional magnetic resonance imaging (fMRI). One frequently used task is the go/no-go response inhibition task, in which participants are asked to respond to all letters except *X* with a button press, and to withhold responding to *X*. Using this task, a series of studies reported relatively worse performance in cocaine- and heroin-dependent individuals compared to healthy controls, along with reduced activity in several PFC regions, including the dorsal ACC (dACC), dlPFC, and vlPFC (e.g., Fu et al., 2008; Hester & Garavan, 2004). Such findings suggest at least some functional alterations in PFC circuits in SUDs, even in the absence of emotion regulation demand. Similarly, Li, Luo, Yan, Bergquist, and Sinha (2009) used the stop-signal response inhibition task, and reported lower activity in dlPFC in alcohol dependence, which further related to higher alcohol craving self-reports. In cocaine-dependent individuals, dACC activity was lower than that in controls and negatively correlated with self-reported difficulties in emotion regulation (Li et al., 2008).

The Stroop color–word task has also been used to probe inhibitory control in SUDs by comparing neural activity during incongruent (BLUE written in red ink) and congruent (BLUE written in blue ink) trials. Using PET, both marijuana- and cocaine-dependent participants showed reduced "Stroop effect" activity in dACC and dlPFC (Bolla et al., 2004; Eldreth, Matochik, Cadet, & Bolla, 2004). In the cocaine-dependent sample only, dlPFC activity negatively correlated with cocaine use (least "Stroop effect" activity for the heaviest users; Bolla et al., 2004). Similarly, DeVito, Kober, Carroll, and Potenza (in preparation) recently used the Stroop task and fMRI in cocaine-dependent participants, and found reduced Stroop-related PFC activity compared to controls. Similar findings have been reported with methamphetamine users (Nestor, Ghahremani, Monterosso, & London, 2011). In marijuana-dependent individuals who were about to begin treatment, Kober, DeVito, DeLeone, Carroll, and Potenza (under review) found reduced "Stroop effect" activity in dlPFC compared to healthy controls, and positive correlations between PFC activity and treatment success. Similarly, Berkman, Falk, and Lieberman (2011) related neural activity during go/no-go task performance to treatment outcome and reported that increased PFC activity during the task was related to a weaker association between

craving and cigarette smoking during subsequent abstinence.

Taken together, these studies suggest that those with SUDs exhibit poorer performance in cognitive control tasks, as well as lower activity in PFC regions typically associated with emotion regulation and executive function more generally, including dlPFC and dACC. Some studies reported direct association between lower PFC function and less cognitive control or emotion regulation, while others link greater PFC activity to better treatment outcomes (e.g., Berkman et al., 2011; Kober et al., under review). These findings are therefore consistent with the hypothesis that PFC abnormalities in structure or function underlie emotion dysregulation in SUDs.

Effects of Drug Use on PFC

Data reviewed thus far suggests that those with SUDs exhibit deficits in emotion regulation, and both structural and functional differences in PFC compared to healthy controls. Notably, most of the reviewed data were generated in the context of case–control studies—measured at a single point in time, in individuals with active SUDs. Therefore, it is not clear whether some of these reported abnormalities precede the development of SUDs (and may serve as a risk factor, as discussed previously), whether they are the result of chronic drug use (and reflect the effects of drug exposure) or an interaction of both. Evidence for PFC abnormalities as a preexisting risk factor includes a recent study of individuals with SUDs (cocaine or amphetamine dependence) and their unaffected siblings compared to healthy adults. Both individuals with SUDs and their unaffected siblings shared a neurological phenotype of reduced structural connectivity in the right vlPFC, which was further related to performance on the stop-signal response inhibition task (Ersche et al., 2012). These findings suggest that potential abnormalities in lateral PFC may underlie regulatory deficits that in fact predate the onset of SUDs.

On the other hand, there is ample evidence, mostly from animal studies, that chronic/regular drug use alters both function and structure of PFC and other brain circuits (for an excellent recent review, see Lüscher & Malenka, 2011). Although it is outside of the scope of this chapter to discuss the unique mechanism of action or pharmacological effects of individual drugs, one now-classic finding is that all drugs that are abused by humans share one common effect. That is, all drugs of abuse—either directly or indirectly—increase concentrations of the neurotransmitter dopamine in the "mesocorticolimbic" pathway, which includes the ventral tegmental area, the ventral striatum, and the PFC (e.g., Dichiara & Imperato, 1988; Volkow et al., 2011).[3] In turn, this drug-induced increase in dopamine is associated with long-term changes or adaptations to neurons in this pathway, including in PFC (Lüscher & Malenka, 2011). These changes are thought to facilitate associations between drugs and drug-related cues (e.g., alcohol and the bar where one drinks; cigarettes and the lighter one uses for smoking), lead to future cue-induced drug craving, and reduce cognitive control (Volkow et al., 2011).

Furthermore, it is thought that some of the effects of acute as well as chronic drug use are *neurotoxic*—damaging to neural tissue (Weiss & Koob, 2001). Such claims emerge primarily from an animal literature experimentally documenting various forms of neuronal damage following heavy drug administration (e.g., Gouzoulis-Mayfrank & Daumann, 2009). Although it is not clear that such findings translate to human drug users (Hart et al., 2011), some studies in humans have linked length of drug use with measures of structural or functional integrity, which is consistent with animal findings. For example, in opiate users, PFC white matter integrity correlated negatively with length of opiate use (Bora et al., 2012; Liu et al., 2008). Similarly, some human studies have shown that various functional and structural abnormalities normalize following drug abstinence, implicating drug use itself in the originally observed differences in PFC (e.g., Gouzoulis-Mayfrank & Daumann, 2009). Taken together, the evidence suggests that even if some PFC abnormalities precede the development of SUDs, drug use itself is associated with long-term changes to many brain circuits, including PFC. Furthermore, these changes may create or exacerbate deficits in emotion regulation in SUDs. In essence, this suggests that chronic drug use may lead to a vicious cycle,

in which impaired emotion regulation leads to drug use, and drug use may further lead to impaired emotion regulation.

Treatment for SUDs: The Role of Emotion Regulation

Treatments for SUDs are varied and complex, as is appropriate given the heterogeneous and complex nature of the disorders that they treat. At this time, despite repeated scientific efforts, there are few FDA-approved pharmacological treatments for SUDs. Therefore, nonpharmacological (e.g., psychological) treatments are most common. While the goal of treatment may be conceptualized as reductions in drug use and in drug-related harm, and increases in psychosocial functioning, treatment success is most often measured in *abstinence*, or complete cessation of drug use. As such, the available treatments are only moderately effective; indeed, across all treatments for SUDs, the most common outcome is relapse (Dutra et al., 2008). This suggests that while some individuals successfully remain abstinent, the majority of patients either do not achieve abstinence or return to drug use within a year, even with the best of treatments. These grim findings underscore the need to better understand the mechanisms of action behind the treatments that do work, in order to improve them further.

From a clinical perspective, treatment for SUDs can be divided into three phases: detoxification, recovery, and relapse prevention (e.g., Potenza et al., 2011). The goal of the detoxification stage is to achieve abstinence and undergo withdrawal symptoms safely, until they abate. The onset, character, and length of this stage depend on the pharmacological properties of the individual drug, as well as treatment type (some treatments begin with recovery elements, then set a "quit date" to begin detoxification). The main stage of treatment is recovery, which can last from 1 week to many weeks. The goal of the recovery stage is to develop motivation to avoid drug use and relapse, as well as learn the skills to do so successfully. In that sense, what does recovery entail? The data reviewed in this chapter suggest that difficulties regulating emotions are a core feature of SUDs. Specifically, I have argued that difficulties regulating both negative (stress, anxiety, or depression) and appetitive (drug craving) states are associated with drug use and with relapse to drug use following abstinence. Therefore, it is no surprise that at this recovery phase, many of the leading treatments include training of emotion regulation skills in general, and regulation of craving in particular (e.g., Potenza et al., 2011; see Figure 26.1B). Indeed, learning to tolerate or regulate cravings and not to act on them is the cornerstone of many of the available treatments, as discussed below. Finally, the last phase of treatment, relapse prevention, focuses on implementing long-term strategies for maintaining abstinence, which includes replacing old behaviors with a new and healthy, drug-free lifestyle. Overall, there are many types of treatments for SUDs, and each type has many unique features. The following sections focus on two types of treatment that are related to the role of emotion regulation in SUDs: cognitive-behavioral and mindfulness-based treatments.

Cognitive-Behavioral Therapies

Cognitive-behavioral therapies (CBTs) are considered the most effective treatment for many psychiatric disorders (e.g., depression and anxiety). A version developed specifically for SUDs (Carroll, 1998) has been empirically validated in multiple randomized controlled trials and is considered by many to be the "gold standard" (e.g., Dutra et al., 2008; Potenza et al., 2011). CBT for SUDs has two critical components: functional analysis and skills training. *Functional analysis* is used to identify and assess the individualized circumstances that are likely to lead to drug use, and provides insights into some of the reasons the individual may be using drugs. These "high-risk situations" are those in which new skills may be applied to avoid drug use during and after treatment. Therefore, in a complementary fashion, skills training (including emotion regulation) is individualized to help those with SUDs "unlearn old habits associated with [drug] . . . abuse and learn or relearn healthier skills and habits" (Carroll, 1998, p. 2). More specifically, these skills initially include regulating thoughts about drugs, learning strategies to regulate cravings for

drugs, and managing situations related to drug-use opportunities (e.g., refusing offers of drugs).

Subsequent treatment sessions (*modules*) focus on problem solving, tolerating and regulating negative affect, and improving social skills more generally. Ultimately, individuals who undergo CBT (compared to other treatments) are more likely to decrease drug use and/or achieve abstinence during and even after treatment has ended (i.e., "the sleeper effect"; Potenza et al., 2011). Although the treatment includes many modules and stages, one important mechanism of action is thought to be via enhancing cognitive control over negative affect that may lead to drug craving, and over drug craving and drug taking behavior (e.g., Kiluk, Nich, Babuscio, & Carroll, 2010; Potenza et al., 2011). This hypothesis is supported by the finding that the number and quality of strategies for regulation of craving increase from pre- to post-CBT treatment, and predict relapse (e.g., Carroll et al., 1999), and formally mediate the relationship between treatment and duration of abstinence (Kiluk et al., 2010). In turn, this increase in regulation skills is hypothesized to be mediated by improved PFC function from pre- to post-treatment (Potenza et al., 2011; see Figure 26.1B for a schematic illustration). Consistent with this hypothesis, Kober, Kross, et al. (2010) have shown that use of CBT-like cognitive strategies during cue-induced craving is associated with decreases in self-reported craving, as well as increased activity in dlPFC and vlPFC (Kober, Mende-Siedlecki, et al., 2010). In addition, DeVito et al. (2011) recently reported that those who underwent CBT exhibited increased efficiency in vlPFC and dlPFC during the Stroop task from pre- to post-CBT treatment, which is consistent with improvements in cognitive control and emotion regulation. However, future studies should test that hypothesis more directly.

Mindfulness-Based Treatments

Mindfulness-based treatments (MBTs) for a variety of psychiatric conditions have emerged in recent years, beginning with mindfulness-based stress reduction (MBSR). *Mindfulness* has been defined as a two-component construct: (1) self-regulation of attention to the present moment, coupled with (2) an attitude of acceptance toward the present moment (Bishop et al., 2004). As such, mindfulness is often practiced through mindfulness meditation, which consists of focusing attention on one's immediate experience (e.g., sensations, breathing, thoughts, emotion), and regarding it nonjudgmentally. This is thought to cultivate the ability to observe—rather than be caught up in—one's own experience, and to further facilitate more skillful or mindful responding (as opposed to automatic reaction; Zgierska et al., 2009). Importantly, meditation and MBTs have been associated with beneficial effects on stress, anxiety, pain, cardiac health, immune functions, psychological well-being, cognitive functioning, and several psychiatric disorders (including mood and anxiety disorders; see Hölzel et al., 2011, for review). Therefore, it is not difficult to extrapolate how MBTs could be beneficial for fostering better emotion regulation in SUDs.

Indeed, several mindfulness-based treatments have recently been adapted for SUDs. Unlike the well-established CBTs, these treatments have just shown preliminary efficacy and are now the focus of rigorous randomized controlled trials. MBTs for SUDs typically include training in mindfulness meditation, and a focus on attention to and acceptance of present-moment experience (including negative emotion and drug craving). The modal instruction is to regard internal experiences (e.g., drug craving) as transient, and to observe and accept them as-is, without reacting (e.g., without engaging in drug use). For example, both mindfulness-based relapse prevention (MBRP; Bowen, Chawla, & Marlatt, 2010) and mindfulness training for smoking (MTS; Brewer et al., 2011) make use of the concept of "urge surfing," the practice of regarding craving like a wave that rises, reaches a peak, and subsides naturally. Patients are instructed to attend to and accept the sensations as they rise, fall, and finally disappear—and this technique is likened to tolerating cravings rather than actively regulating them, as in CBT (Brewer et al., 2011). Consistently, Westbrook et al. (2013) have shown that mindful attention to smoking cues is associated with lower self-reported craving and lower neural activity in regions previously associated with craving, without concomitant increases in PFC activ-

ity. As such, mindful attention and acceptance may be regulatory, by preventing the amplification of craving rather than dampening them down.

Clinically, MBRP, typically administered as a follow-up to inpatient treatment, is reportedly efficacious in reducing drug use and relapse across several different populations with SUDs, including alcohol and polysubstance users (e.g., Witkiewitz & Bowen, 2010). In addition, Brewer et al. (2011) have recently shown in a pilot randomized controlled trial that MTS administered as a stand-alone treatment was effective in achieving smoking cessation. Finally, similar elements of mindful attention and acceptance are parts of dialectical behavior therapy and acceptance and commitment therapy, both of which have shown preliminary efficacy for SUDs (Hernández-López, Luciano, Bricker, Roales-Nieto, & Montesinos, 2009; Linehan et al., 2002). Taken together, these data show substantial promise for MBTs in the treatment of SUDs, although the research is still in its infancy. Nevertheless, one prominent hypothesis suggests that these treatments work by enhancing emotion regulation, as patients learn to practice mindfulness in the face of craving as well as negative emotion. This is supported by several findings, including consistent reductions in craving post-MBRP (e.g., Witkiewitz & Bowen, 2010), and a negative correlation after MTS between amount of meditation practice and smoking (Brewer et al., 2011).

Concluding Remarks

Casual drug use is quite prevalent, and a percentage of drug users develop SUDs, which are severe psychiatric conditions with staggering social, economic, and personal costs. This underscores the need to identify risk factors that render specific individuals more vulnerable to the development of SUDs. Furthermore, once established, SUDs are chronic, relapsing, and very difficult to treat psychiatric conditions; this underscores the need to better characterize the proximal causal factors that lead to continued drug use, and to better understand the mechanisms that underlie effective treatments for these disorders. In this chapter, I reviewed data suggesting that emotion regulation is one such crucial factor. Indeed, difficulties in emotion regulation in childhood and adolescence serve as predictive factors for future drug use and the development of SUDs. Subsequently, those with SUDs report greater difficulty regulating negative emotions than do healthy controls, which contributes to ongoing drug use. Furthermore, I reviewed evidence suggesting that craving for drugs is one of the key predictors of drug use, and that the ability to regulate craving effectively is directly related to reduced drug use in SUDs.

Consistent with these observations, psychosocial treatments for SUDs often focus on emotion regulation and on the regulation of craving as means for reducing drug use. Indeed, improvement in those skills following treatments such as CBT and MBTs is associated with improved abstinence. Finally, I reviewed data suggesting that differences in PFC structure, as well as function, may underlie impaired emotion regulation in those with SUDs, and that ongoing drug use leads to adaptations in PFC that may further impair emotion regulation. However, it will be critical to focus future research on more precisely characterizing the neural mechanisms behind observed PFC deficits in SUDs and behind treatment-related improvements. Indeed, it is my sincere hope that in the coming years, additional data will allow us to establish these links more firmly. This could lead to the development of better treatments that improve emotion regulation in individuals suffering from these devastating disorders.

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Notes

1. Psychoactive drugs are those that primarily act on the brain and change thinking, mood, and behavior. These include legal drugs (e.g., alcohol, nicotine, caffeine, and opioid pain medications), as well as illicit drugs (e.g., heroin, cocaine, amphetamines, and marijuana).
2. *Self-control* is often defined as the process of inhibition of an otherwise imminent thought, emotion, or action—and as such, it includes emotion regulation. Related to this is the construct of cognitive control, which more broadly includes goal maintenance, selective attention, conflict monitoring and resolution, response inhibition, and emotion regulation. See Gross (this volume) for discussion.
3. It is now known that many other neurotransmitter systems are involved in drug taking and in the development of SUDs, and the next decade will likely bring additional investigations into other neurotransmitter systems and their relation to SUDs.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Alicata, D., Chang, L., Cloak, C., Abe, K., & Ernst, T. (2009). Higher diffusion in striatum and lower fractional anisotropy in white matter of methamphetamine users. *Psychiatry Research: Neuroimaging, 174*(1), 1–8.

August, G. J., Winters, K. C., Realmuto, G. M., Fahnhorst, T., Botzet, A., & Lee, S. (2006). Prospective study of adolescent drug use among community samples of ADHD and non-ADHD participants. *Journal of American Academy of Child and Adolescent Psychiatry, 45*(7), 824–832.

Badiani, A., Belin, D., Epstein, D., Calu, D., & Shaham, Y. (2011). Opiate versus psychostimulant addiction: The differences do matter. *Nature Reviews Neuroscience, 12*(11), 685–700.

Baker, T. B., Piper, M. E., McCarthy, D. E., Majeskie, M. R., & Fiore, M. C. (2004). Addiction motivation reformulated: An affective processing model of negative reinforcement. *Psychological Review, 111*(1), 33–51.

Berking, M., Margraf, M., Ebert, D., Wupperman, P., Hofmann, S. G., & Junghanns, K. (2011). Deficits in emotion-regulation skills predict alcohol use during and after cognitive-behavioral therapy for alcohol dependence. *Journal of Consulting and Clinical Psychology, 79*(3), 307–318.

Berkman, E. T., Falk, E. B., & Lieberman, M. D. (2011). In the trenches of real-world self-control: Neural correlates of breaking the link between craving and smoking. *Psychological Science, 22*(4), 498–506.

Bishop, S. R., Lau, M., Shapiro, S., Carlson, L., Anderson, N. D., Carmody, J., et al. (2004). Mindfulness: A proposed operational definition. *Clinical Psychology: Science and Practice, 11*(3), 230–241.

Bolla, K., Ernst, M., Kiehl, K., Mouratidis, M., Eldreth, D., Contoreggi, C., et al. (2004). Prefrontal cortical dysfunction in abstinent cocaine abusers. *Journal of Neuropsychiatry and Clinical Neurosciences, 16*(4), 456–464.

Bonn-Miller, M. O., Vujanovic, A. A., & Zvolensky, M. J. (2008). Emotional dysregulation: Association with coping-oriented marijuana use motives among current marijuana users. *Substance Use and Misuse, 43*(11), 1656–1668.

Bora, E., Yücel, M., Fornito, A., Pantelis, C., Harrison, B. J., Cocchi, L., et al. (2012). White matter microstructure in opiate addiction. *Addiction Biology, 17*(1), 141–148.

Bowen, S., Chawla, N., & Marlatt, G. A. (2010). *Mindfulness-based relapse prevention for addictive behaviors: A clinician's guide*. New York: Guilford Press.

Bradizza, C. M., Stasiewicz, P. R., & Paas, N. D. (2006). Relapse to alcohol and drug use among individuals diagnosed with co-occurring mental health and substance use disorders: A review. *Clinical Psychology Review, 26*(2), 162–178.

Brewer, J. A., Mallik, S., Babuscio, T. A., Nich, C., Johnson, H. E., Deleone, C. M., et al. (2011). Mindfulness training for smoking cessation: Results from a randomized controlled trial. *Drug and Alcohol Dependence, 119*(1–2), 72–80.

Brody, A. L., Olmstead, R. E., London, E. D., Farahi, J., Meyer, J. H., Grossman, P., et al. (2004). Smoking-induced ventral striatum dopamine release. *American Journal of Psychiatry, 161*(7), 1211–1218.

Carroll, K. M. (1998). *Therapy manuals for drug addiction: A cognitive-behavioral approach: Treating cocaine addiction*. Bethesda, MD: National Institute of Drug Abuse.

Carroll, K. M., Nich, C., Frankforter, T. L., & Bisighini, R. M. (1999). Do patients change in the way we intend?: Treatment-specific skill acquisition in cocaine-dependent patients using the Cocaine Risk Response Test. *Psychological Assessment, 11*, 77–85.

Chase, H. W., Eickhoff, S. B., Laird, A. R., & Hogarth, L. (2011). The neural basis of drug stimulus processing and craving: An activation likelihood estimation meta-analysis. *Biological Psychiatry, 70*(8), 785–793.

Childress, A. R., Hole, A. V., Ehrman, R. N., Robbins, S. J., McLellan, A. T., & O'Brien, C. P. (1993). Cue reactivity and cue reactivity interventions in drug dependence. *NIDA Research Monograph, 137*, 73–73.

Cordovil de Sousa Uva, M., Timary, P., Cortesi, M., Mikolajczak, M., Blicquy, P. R., & Luminet, O. (2010). Moderating effect of emotional intelligence on the role of negative affect in the motivation to drink in alcohol-dependent subjects undergoing protracted withdrawal. *Personality and Individual Differences, 48*(1), 16–21.

Crits-Christoph, P., Gibbons, M. B. C., Barber, J. P., Hu, B., Hearon, B., Worley, M., & Gallop, R. (2007). Predictors of sustained abstinence during psychosocial treatments for cocaine dependence. *Psychotherapy Research, 17*(2), 240–252.

Daughters, S. B., Lejuez, C., Bornovalova, M. A., Kahler, C. W., Strong, D. R., & Brown, R. A. (2005). Distress tolerance as a predictor of early treatment dropout in a residential substance abuse treatment facility. *Journal of Abnormal Psychology, 114*(4), 729–734.

Daumann, J., Koester, P., Becker, B., Wagner, D., Imperati, D., Gouzoulis-Mayfrank, E., & Tittgemeyer, M. (2011). Medial prefrontal gray matter volume reductions in users of amphetamine-type stimulants revealed by combined tract-based spatial statistics and voxel-based morphometry. *NeuroImage, 54*(2), 794–801.

DeVito, E. E., Kober, H., Carroll, K. M., & Potenza, M. N. (in preparation). *Sex differences in the neural correlates of cognitive control in cocaine dependence.*

DeVito, E. E., Worhunsky, P. D., Carroll, K. M., Rounsaville, B. J., Kober, H., & Potenza, M. N. (2011). A preliminary study of the neural effects of behavioral therapy for substance use disorders. *Drug and Alcohol Dependence, 122*(3), 228–235.

Dichiara, G., & Imperato, A. (1988). Drugs abused by humans preferentially increase synaptic dopamine concentrations in the mesolimbic system of freely moving rats. *Proceedings of the National Academy of Sciences USA, 85*(14), 5274–5278.

Durazzo, T. C., Tosun, D., Buckley, S., Gazdzinski, S., Mon, A., Fryer, S. L., & Meyerhoff, D. J. (2011). Cortical thickness, surface area, and volume of the brain reward system in alcohol dependence: Relationships to relapse and extended abstinence. *Alcoholism: Clinical and Experimental Research, 35*, 1187–1200.

Dutra, L., Stathopoulou, G., Basden, S. L., Leyro, T. M., Powers, M. B., & Otto, M. W. (2008). A meta-analytic review of psychosocial interventions for substance use disorders. *American Journal of Psychiatry, 165*(2), 179–187.

Eldreth, D. A., Matochik, J. A., Cadet, J. L., & Bolla, K. I. (2004). Abnormal brain activity in prefrontal brain regions in abstinent marijuana users. *NeuroImage, 23*(3), 914–920.

Epstein, D. H., Marrone, G. F., Heishman, S. J., Schmittner, J., & Preston, K. L. (2010). Tobacco, cocaine, and heroin: Craving and use during daily life. *Addictive Behaviors, 35*(4), 318–324.

Ersche, K. D., Jones, P. S., Williams, G. B., Turton, A. J., Robbins, T. W., & Bullmore, E. T. (2012). Abnormal brain structure implicated in stimulant drug addiction. *Science, 335*(6068), 601–604.

Everitt, B. J., & Robbins, T. W. (2005). Neural systems of reinforcement for drug addiction: From actions to habits to compulsion. *Nature Neuroscience, 8*(11), 1481–1489.

Fein, G., Di Sclafani, V., Cardenas, V., Goldmann, H., Tolou-Shams, M., & Meyerhoff, D. J. (2002). Cortical gray matter loss in treatment-naive alcohol dependent individuals. *Alcoholism: Clinical and Experimental Research, 26*(4), 558–564.

Fein, G., Di Sclafani, V., & Meyerhoff, D. J. (2002). Prefrontal cortical volume reduction associated with frontal cortex function deficit in 6-week abstinent crack-cocaine dependent men. *Drug and Alcohol Dependence, 68*(1), 87–93.

Fox, H. C., Hong, K. A., & Sinha, R. (2008). Difficulties in emotion regulation and impulse control in recently abstinent alcoholics compared with social drinkers. *Addictive Behaviors, 33*(2), 388–394.

Franklin, T. R., Acton, P. D., Maldjian, J. A., Gray, J. D., Croft, J. R., Dackis, C. A., et al.

(2002). Decreased gray matter concentration in the insular, orbitofrontal, cingulate, and temporal cortices of cocaine patients. *Biological Psychiatry, 51*(2), 134–142.

Fu, L., Bi, G., Zou, Z., Wang, Y., Ye, E., Ma, L., et al. (2008). Impaired response inhibition function in abstinent heroin dependents: An fMRI study. *Neuroscience Letters, 438*(3), 322–326.

Fucito, L. M., Juliano, L. M., & Toll, B. A. (2010). Cognitive reappraisal and expressive suppression emotion regulation strategies in cigarette smokers. *Nicotine and Tobacco Research, 12*(11), 1156–1161.

Galloway, G. P., Singleton, E. G., & the Methamphetamine Treatment Project Corporate Authors. (2008). How long does craving predict use of methamphetamine?: Assessment of use one to seven weeks after the assessment of craving. *Substance Abuse: Research and Treatment, 1*, 63–79.

Gamble, S. A., Conner, K. R., Talbot, N. L., Yu, Q., Tu, X. M., & Connors, G. J. (2010). Effects of pretreatment and posttreatment depressive symptoms on alcohol consumption following treatment in project MATCH. *Journal of Studies on Alcohol and Drugs, 71*(1), 71–77.

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., et al. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience, 2*, 861–862.

Gilbert, D. G., Crauthers, D. M., Mooney, D. K., McClernon, F. J., & Jensen, R. A. (1999). Effects of monetary contingencies on smoking relapse: Influences of trait depression, personality, and habitual nicotine intake. *Experimental and Clinical Psychopharmacology, 7*(2), 174–181.

Goldstein, R. Z., & Volkow, N. D. (2011). Dysfunction of the prefrontal cortex in addiction: Neuroimaging findings and clinical implications. *Nature Reviews Neuroscience, 12*(11), 652–669.

Gouzoulis-Mayfrank, E., & Daumann, J. (2009). Neurotoxicity of drugs of abuse—the case of methylenedioxy amphetamines (MDMA, ecstasy), and amphetamines. *Dialogues in Clinical Neuroscience, 11*(3), 305–317.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Haney, M., Maccari, S., Le Moal, M., Simon, H., & Vincenzo Piazza, P. (1995). Social stress increases the acquisition of cocaine self-administration in male and female rats. *Brain Research, 698*(1–2), 46–52.

Hart, C. L., Marvin, C. B., Silver, R., & Smith, E. E. (2011). Is cognitive functioning impaired in methamphetamine users?: A critical review. *Neuropsychopharmacology, 37*(3), 586–608.

Hart, C. L., Ward, A. S., Haney, M., Foltin, R. W., & Fischman, M. W. (2001). Methamphetamine self-administration by humans. *Psychopharmacology, 157*(1), 75–81.

Heinz, A., Siessmeier, T., Wrase, J., Buchholz, H. G., Gründer, G., Kumakura, Y., et al. (2005). Correlation of alcohol craving with striatal dopamine synthesis capacity and D2/3 receptor availability: A combined [18F]DOPA and [18F]DMFP PET study in detoxified alcoholic patients. *American Journal of Psychiatry, 162*(8), 1515–1520.

Hernández-López, M., Luciano, M. C., Bricker, J. B., Roales-Nieto, J. G., & Montesinos, F. (2009). Acceptance and commitment therapy for smoking cessation: A preliminary study of its effectiveness in comparison with cognitive behavioral therapy. *Psychology of Addictive Behaviors, 23*(4), 723–730.

Hester, R., & Garavan, H. (2004). Executive dysfunction in cocaine addiction: Evidence for discordant frontal, cingulate, and cerebellar activity. *Journal of Neuroscience, 24*(49), 11017–11022.

Hölzel, B. K., Lazar, S. W., Gard, T., Schuman-Olivier, Z., Vago, D. R., & Ott, U. (2011). How does mindfulness meditation work?: Proposing mechanisms of action from a conceptual and neural perspective. *Perspectives on Psychological Science, 6*(6), 537–559.

Ivanov, I., Newcorn, J., Morton, K., & Tricamo, M. (2011). Inhibitory control deficits in childhood: Definition, measurement, and clinical risk for substance use disorders. In M. T. Bardo (Ed.), *Inhibitory control and drug abuse prevention: From research to translation* (pp. 125–144). New York: Springer Science+Business Media.

Jaffe, J. H., & Jaffe, F. K. (1989). Historical perspectives on the use of subjective effects measures in assessing the abuse potential of drugs. *NIDA Research Monographs, 92*, 43–72.

Johnstone, T., & Walter, H. (this volume, 2014). The neural basis of emotion dysregulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 58–75). New York: Guilford Press.

Jones, B. T., Corbin, W., & Fromme, K. (2001). A review of expectancy theory and alcohol consumption. *Addiction, 96*, 57–72.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*(6), 593–602.

Khantzian, E. J. (1985). The self-medication hypothesis of addictive disorders: focus on heroin and cocaine dependence. *American Journal of Psychiatry, 142*(11), 1259–1264.

Kiluk, B. D., Nich, C., Babuscio, T., & Carroll, K. M. (2010). Quality versus quantity: Acquisition of coping skills following computerized cognitive behavioral therapy for substance use disorders. *Addiction, 105*(12), 2120–2127.

Kober, H., & DeLeone, C. M. (2011). Smoking and neuroimaging: A review. *Current Cardiovascular Risk Reports, 5*(6), 484–491.

Kober, H., DeVito, E. E., DeLeone, C. M., Carroll, K. M., & Potenza, M. N. (under review). *Pre-treatment neural activity in cannabis users differs from controls and relates to long term treatment outcome.*

Kober, H., Kross, E. F., Mischel, W., Hart, C. L., & Ochsner, K. N. (2010). Regulation of craving by cognitive strategies in cigarette smokers. *Drug and Alcohol Dependence, 106*(1), 52–55.

Kober, H., Mende-Siedlecki, P., Kross, E. F., Weber, J., Mischel, W., Hart, C. L., et al. (2010). Prefrontal–striatal pathway underlies cognitive regulation of craving. *Proceedings of the National Academy of Sciences, 107*(33), 14811–14816.

Kober, H., Turza, A. C., & Hart, C. L. (2009). Risk factors for substance use, abuse, and dependence: Learning. In H. Kranzler & P. Korsmeyer (Eds.), *Encyclopedia of drugs, alcohol, and addictive behavior* (3rd ed.). Macmillan Reference USA.

Koob, G. F., & Le Moal, M. (2008). Neurobiological mechanisms for opponent motivational processes in addiction. *Philosophical Transactions of the Royal Society B: Biological Sciences, 363*(1507), 3113–3123.

Kun, B., & Demetrovics, Z. (2010). Emotional intelligence and addictions: A systematic review. *Substance Use and Misuse, 45*(7–8), 1131–1160.

Le Moal, M. (2009). Drug abuse: Vulnerability and transition to addiction. *Pharmacopsychiatry, 42*(S1), 542–555.

Li, C. S. R., Huang, C., Yan, P., Bhagwagar, Z., Milivojevic, V., & Sinha, R. (2008). Neural correlates of impulse control during stop signal inhibition in cocaine-dependent men. *Neuropsychopharmacology, 33*(8), 1798–1806.

Li, C. S. R., Luo, X., Yan, P., Bergquist, K., & Sinha, R. (2009). Altered impulse control in alcohol dependence: Neural measures of stop signal performance. *Alcoholism: Clinical and Experimental Research, 33*(4), 740–750.

Linehan, M. M., Dimeff, L. A., Reynolds, S. K., Comtois, K. A., Welch, S. S., Heagerty, P., et al. (2002). Dialectical behavior therapy versus comprehensive validation therapy plus 12-step for the treatment of opioid dependent women meeting criteria for borderline personality disorder. *Drug and Alcohol Dependence, 67*(1), 13–26.

Litt, M. D., Cooney, N. L., & Morse, P. (2000). Reactivity to alcohol-related stimuli in the laboratory and in the field: Predictors of craving in treated alcoholics. *Addiction, 95*(6), 889–900.

Littel, M., & Franken, I. H. A. (2011). Intentional modulation of the late positive potential in response to smoking cues by cognitive strategies in smokers. *PloS ONE, 6*(11), e27519.

Liu, H., Li, L., Hao, Y., Cao, D., Xu, L., Rohrbaugh, R., et al. (2008). Disrupted white matter integrity in heroin dependence: A controlled study utilizing diffusion tensor imaging. *American Journal of Drug and Alcohol Abuse, 34*(5), 562–575.

Luerssen, A., & Ayduk, O. (this volume, 2014). The role of emotion and emotion regulation in the ability to delay gratification. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., 111–125). New York: Guilford Press.

Lüscher, C., & Malenka, R. C. (2011). Drug-evoked synaptic plasticity in addiction: From molecular changes to circuit remodeling. *Neuron, 69*(4), 650–663.

Marlatt, G. A., & Witkiewitz, K. (2005). Relapse prevention for alcohol and drug problems. In G. Marlatt & D. Donovan (Eds.), *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors* (2nd ed., pp. 1–44). New York: Guilford Press.

Marshall-Berenz, E. C., Vujanovic, A. A., & MacPherson, L. (2011). Impulsivity and alcohol use coping motives in a trauma-exposed sample: The mediating role of distress tolerance. *Personality and Individual Differences, 50*(5), 588–592.

McRae, K., Gross, J. J., Weber, J., Robertson, E.

R., Sokol-Hessner, P., Ray, R. D., et al. (2012). The development of emotion regulation: An fMRI study of cognitive reappraisal in children, adolescents and young adults. *Social Cognitive and Affective Neuroscience, 7*(1), 11–22.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*(1), 167–202.

Mischel, W., Ayduk, O., Berman, M. G., Casey, B., Gotlib, I. H., Jonides, J., et al. (2011). 'Willpower' over the life span: Decomposing self-regulation. *Social Cognitive and Affective Neuroscience, 6*(2), 252–256.

Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H. L., et al. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *Proceedings of the National Academy of Sciences, 108*(7), 2693–2698.

Morasco, B. J., Gritzner, S., Lewis, L., Oldham, R., Turk, D. C., & Dobscha, S. K. (2011). Systematic review of prevalence, correlates, and treatment outcomes for chronic non-cancer pain in patients with comorbid substance use disorder. *Pain, 152*(3), 488–497.

National Institute of Mental Health (NIMH). (2007). National Comorbidity Survey: Lifetime prevalence estimates. Available from *www.hcp.med.harvard.edu/ncs*.

Nestor, L. J., Ghahremani, D. G., Monterosso, J., & London, E. D. (2011). Prefrontal hypoactivation during cognitive control in early abstinent methamphetamine-dependent subjects. *Psychiatry Research: Neuroimaging, 194*(3), 287–295.

Nunes, E. V., & Levin, F. R. (2004). Treatment of depression in patients with alcohol or other drug dependence. *Journal of the American Medical Association, 291*(15), 1887–1896.

O'Brien, C. P., Childress, A. R., Ehrman, R., & Robbins, S. J. (1998). Conditioning factors in drug abuse: Can they explain compulsion? *Journal of Psychopharmacology, 12*(1), 15–22.

Ochsner, K. N., & Gross, J. J. (this volume, 2014). The neural bases of emotion and emotion regulation: A valuation perspective. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 23–42). New York: Guilford Press.

O'Connell, K. A., Hosein, V. L., Schwartz, J. E., & Leibowitz, R. Q. (2007). How does coping help people resist lapses during smoking cessation? *Health Psychology, 26*(1), 77–84.

Perkins, K. A. (2009). Does smoking cue-induced craving tell us anything important about nicotine dependence? *Addiction, 104*(10), 1610–1616.

Pfefferbaum, A., Rosenbloom, M., Rohlfing, T., & Sullivan, E. V. (2009). Degradation of association and projection white matter systems in alcoholism detected with quantitative fiber tracking. *Biological Psychiatry, 65*(8), 680–690.

Pfeifer, J. H., Lieberman, M. D., & Dapretto, M. (2007). "I know you are but what am I?": Neural bases of self-and social knowledge retrieval in children and adults. *Journal of Cognitive Neuroscience, 19*(8), 1323–1337.

Potenza, M. N., Sofuoglu, M., Carroll, K. M., & Rounsaville, B. J. (2011). Neuroscience of behavioral and pharmacological treatments for addictions. *Neuron, 69*(4), 695–712.

Rando, K., Hong, K. I., Bhagwagar, Z., Li, C. S. R., Bergquist, K., Guarnaccia, J., et al. (2011). Association of frontal and posterior cortical gray matter volume with time to alcohol relapse: A prospective study. *American Journal of Psychiatry, 168*(2), 183–192.

Riediger, M., & Klipker, K. (this volume, 2014). Emotion regulation in adolescence. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., 187–202). New York: Guilford Press.

Riley, H., & Schutte, N. S. (2003). Low emotional intelligence as a predictor of substance-use problems. *Journal of Drug Education, 33*(4), 391–398.

Romero, M. J., Asensio, S., Palau, C., Sanchez, A., & Romero, F. J. (2010). Cocaine addiction: Diffusion tensor imaging study of the inferior frontal and anterior cingulate white matter. *Psychiatry Research: Neuroimaging, 181*(1), 57–63.

Shiffman, S., Dunbar, M., Kirchner, T., Li, X., Tindle, H., Anderson, S., et al. (2013). Smoker reactivity to cues: Effects on craving and on smoking behavior. *Journal of Abnormal Psychology, 122*(1), 264–280.

Shiffman, S., Paty, J. A., Gnys, M., Kassel, J. A., & Hickcox, M. (1996). First lapses to smoking: Within-subjects analysis of real-time reports. *Journal of Consulting and Clinical Psychology, 64*(2), 366–379.

Silvers, J. A., McRae, K., Gabrieli, J. D. E., Gross, J. J., Remy, K. A., & Ochsner, K. N. (2012). Age-related differences in emotional reactivity, regulation, and rejection sensitivity in adolescence. *Emotion, 12*(6), 1235–1247.

Sinha, R., & Li, C. S. R. (2007). Imaging stress- and cue-induced drug and alcohol craving: Association with relapse and clinical implications. *Drug and Alcohol Review, 26*(1), 25–31.

Sorg, S. F., Taylor, M. J., Alhassoon, O. M., Gongvatana, A., Theilmann, R. J., et al. (2012). Frontal white matter integrity predictors of adult alcohol treatment outcome. *Biological Psychiatry, 71*, 262–268.

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Sciences, 9*(2), 69–74.

Substance Abuse and Mental Health Services Administration (SAMHSA). (2011). *Results from the 2010 National Survey on Drug Use and Health (NSDUH): Summary of national findings* (NSDUH Series H-41). Rockville, MD: Author.

Tabibnia, G., Monterosso, J. R., Baicy, K., Aron, A. R., Poldrack, R. A., Chakrapani, S., et al. (2011). Different forms of self-control share a neurocognitive substrate. *Journal of Neuroscience, 31*(13), 4805–4810.

Taioli, E., & Wynder, E. L. (1991). Effect of the age at which smoking begins on frequency of smoking in adulthood. *New England Journal of Medicine, 325*(13), 968–969.

Tarter, R. E., Kirisci, L., Mezzich, A., Cornelius, J. R., Pajer, K., Vanyukov, M., et al. (2003). Neurobehavioral disinhibition in childhood predicts early age at onset of substance use disorder. *American Journal of Psychiatry, 160*(6), 1078–1085.

Verdejo-García, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a vulnerability marker for substance-use disorders: Review of findings from high-risk research, problem gamblers and genetic association studies. *Neuroscience and Biobehavioral Reviews, 32*(4), 777–810.

Volkow, N. D., Fowler, J., Wang, G., Telang, F., Logan, J., Jayne, M., et al. (2010). Cognitive control of drug craving inhibits brain reward regions in cocaine abusers. *NeuroImage, 49*, 2536–2543.

Volkow, N. D., Wang, G. J., Fowler, J. S., & Tomasi, D. (2011). Addiction Circuitry in the Human Brain. *Annual review of pharmacology and toxicology, 52*(1), 321–336.

Weiss, F., & Koob, G. F. (2001). Drug addiction: Functional neurotoxicity of the brain reward systems. *Neurotoxicity Research, 3*(1), 145–156.

Westbrook, C., Creswell, J. D., Tabibnia, G., Julson, E., Kober, H., & Tindle, H. A. (2013). Mindful attention reduces neural and self-reported cue-induced craving in smokers. *Social Cognitive and Affective Neuroscience, 8*(1), 73–84.

Witkiewitz, K., & Bowen, S. (2010). Depression, craving, and substance use following a randomized trial of mindfulness-based relapse prevention. *Journal of Consulting and Clinical Psychology, 78*(3), 362–374.

Zgierska, A., Rabago, D., Chawla, N., Kushner, K., Koehler, R., & Marlatt, A. (2009). Mindfulness meditation for substance use disorders: A systematic review. *Substance Abuse, 30*(4), 266–294.

CHAPTER 27

A Psychological Construction Account of Emotion Regulation and Dysregulation: The Role of Situated Conceptualizations

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For the last several centuries, philosophers, and later, psychologists have assumed that the mind works like a machine—a printing press, a switchboard, a computer. According to the machine metaphor, the mind consists of a number of functionally distinct processes (mental "modules" or "faculties") that interact with one another; if separated, these processes would still retain their identity and function. The machine metaphor dictates a particular view of mental causation: "Psychological process A" localized in one swath of brain tissue (a region or a network) causes a change in a separate and distinct "psychological process B" localized in another swath of tissue (see Figure 27.1). A good example of the machine metaphor at work can be found in the science of emotion regulation. It is largely assumed, for example, that an emotion, like fear, is created by one process computed in one part of the brain (usually in subcortical limbic or paralimbic cortex) that is regulated by executive or other cognitive processes located elsewhere in the brain (typically somewhere in prefrontal cortex). In the process model of emotion regulation (Gross, this volume), an emotion can be triggered first, then subsequently is regulated (e.g., you are walking in the woods, and a fuzzy bee buzzing around your head triggers a state of fear, which you then regulate by suppressing the urge to run and by distracting yourself with a close examination of the local scenery, such as an interestingly shaped rock or tree). Regulation might also occur before the response occurs, preempting the

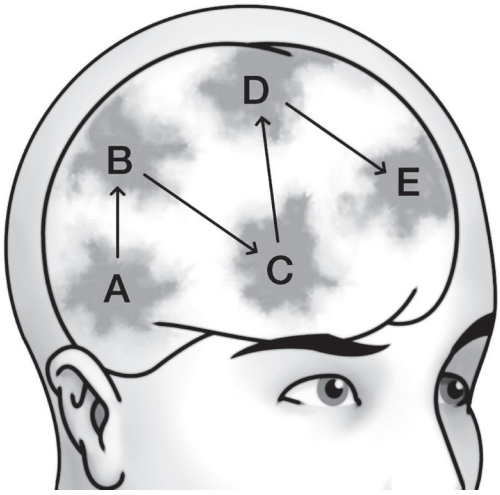

FIGURE 27.1. A depiction of the machine metaphor of brain function.

emotion from ever taking place (e.g., before you start your walk, you might remind yourself that bees are a part of nature; they pollinate beautiful flowers and make delicious honey). Regardless of which comes first, the emotion is separate from its regulation.

In the last several years, scientists have come to question whether the mind and brain work like a machine with separate, interacting bits and pieces (e.g., Barrett, 2009), and assumptions about modularity, even in sensory cortices, are strongly in question. As a consequence, other working metaphors for the mind and the brain are more apt—say, molecules that are constructed of atoms, chamber music emerging from the interplay of instruments, or recipes from a well-stocked pantry full of ingredients. These metaphors begin with a deceptively simple observation: During every moment of waking life, the brain takes in sensory input captured from the world outside the skin (light, vibrations, odors, etc.) and sensations captured from within the body that holds the brain (the internal "milieu"), and uses knowledge from prior experience (also variously called concepts, memories, associations, beliefs, predictions, etc.)—stored in association cortex, and in sensory neurons and subcortical regions—and makes those sensory inputs meaningful. This occurs by creating *situated conceptualizations* (Barrett, 2006, 2012; Barsalou, 2003, 2009; Barsalou, Niedenthal, Barbey, & Ruppert, 2003; Wilson-Mendenhall, Barrett, Simmons, & Barsalou, 2011). A situated conceptualization initially is like a prediction of what sensory input stands for in the world (i.e., object or event identification), which properties are salient (i.e., deserving of attention), what to do about that sensory input (i.e., a predicted action), and what the homeostatic and metabolic consequences will be (i.e., affective changes). From our perspective, the brain's architecture can be thought of as a situated conceptualization generator producing the individual brain states that correspond to each individual mental state, such as an individual instance of fear or an instance of regulation.

Building on the kitchen metaphor (in Barrett, 2009), we have proposed that each brain state, each situated conceptualization, can be understood in terms of more core systems (i.e., the ingredients), which can themselves be characterized at both the psychological level (e.g., Barrett, 2006, 2012) and the level of brain networks (e.g., Barrett & Satpute, 2013; Lindquist & Barrett, 2012). These core systems are like the "mental state variables" (see Salzman & Fusi, 2010), facets or core systems that describe the brain state. As basic "ingredients" of the mind, they are necessary for but not specific to emotion generation, or to emotion regulation per se, just as flour and salt are necessary for but not specific to bread. As the brain transitions from one state to another, mental states ebb and flow, and people give special names to these different states. We refer to this as a *psychological constructionist*, or merely a *constructionist*, approach to the mind and brain. From this constructionist point of view, emotions are not unique mental states that are caused by dedicated mechanisms, to be modified by another set of dedicated regulatory mechanisms. Instead, emotions emerge, and regulation occurs, as the consequence of an ongoing, continually modified constructive process that makes sensory inputs meaningful. Every mental state, including an emotion both before and after regulation is said to have occurred, is a situated conceptualization, constructed from assemblies of neurons that perform sensory, conceptual, attentional, and action functions.

In this chapter, we examine in more detail the concept of *emotion regulation* as resulting from the never ending sequence of situated conceptualizations that occur as the brain transitions from one state to another. First, we introduce the general idea that knowledge (as reactivation and recombination of prior experience) gives meaning to incoming sensory input and is itself enactive (i.e., adds novel features via perceptual inference). Next, we link these notions to the idea of situated conceptualizations from the literature on concepts and categories, and discuss how we have broadened it into a general proposal of constructed mental states that involve making meaning of sensory input and even modifying sensations during the process. We then discuss how emotions might be understood as situated conceptualizations, and how emotion regulation might be reconceptualized as shifting from one situated conceptualization to another. Finally, we use this framework to consider how emo-

tional dysregulation might be understood in terms of the situated conceptualizations that are constructed.

Making Meaning of Sensory Input

Take a look at the image in Figure 27.2. As you look at this image, your brain is trying to make sense of the visual input it is receiving. If you are having difficulty making sense of the visual input from the image (e.g., you cannot recognize an object in it), then you are in a state of experiential blindness (e.g., Fine, Wade, Brewer, May, Goodman, et al., 2003). This is because, usually, in the blink of an eye, quite automatically and with no effort whatsoever, your brain is able to integrate impinging sensory stimulation seamlessly with its vast amount of stored knowledge (from prior experience), allowing you to construct a visual representation of the object. Such knowledge is not merely helpful—it is necessary to normal perception. Without prior experience, the sensations are meaningless, and if this were an object before you, in three-dimensional space, you would not know how to act on it. The definition of adaptive behavior is the use of past experience to guide future action.

Now look at the image at the end of the chapter (on page 465). Then return to look again at Figure 27.2.

We hope you can now see the object, because you have had an experience to help you make sense of the visual input. The first lesson here is that it very difficult to "unsee" the object in the original "blobby" black-and-white image. A second lesson is that no matter how hard you try, you cannot gain introspective access to the processes in your brain that underlie using stored knowledge to make incoming sensations meaningful. Experimental methods are necessary to unmask its workings. The third lesson from this example is that your brain infers elements of the experience that are not immediately present (e.g., the lines that link the black and gray blobs together into the shape of a bee). Although you cannot gain introspective access to these inferred features, you can get a sense that this inference process works by conjuring some additional perceptual detail—the soft drone of buzzing, or the delicate flutter of wings. In your mind's eye, you might see the object nose around as it searches for pollen. You might even be able to smell the sweet fragrance of the flower. Inference is considered one of the primary purposes of memory and is how experiences of the past help to inform situated action in the present. You could not survive in the world without this capacity. Some scientists refer to this inference process as *simulation* (e.g., Barsalou, 1999, 2009), where you can connect immediate sensory input with a vast body of sensory, motor, affective, and other, related information stored in memory; we also refer to it as *categorization* (Barrett, 2006).

The fourth lesson is that these inferences prepare you for situated action. For some people, perhaps, who have experienced bees as part of a beautiful garden and/or as producing a sweet, tasty delight (honey), the image of a bee is calming and bucolic.

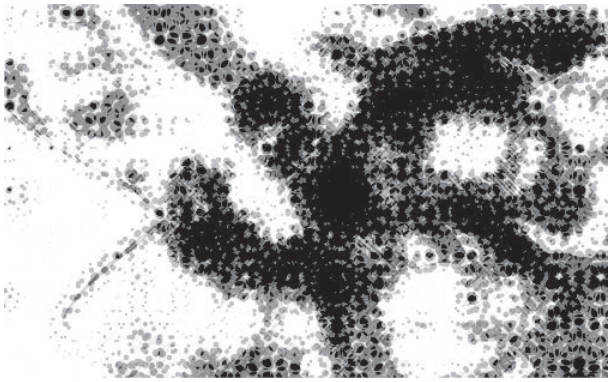

FIGURE 27.2. An illustration of experiential blindness.

For them, seeing a bee might mean moving in to get a closer look, with an associated reduction in heart rate, blood pressure, and skin conductance. For other people, perhaps who have been stung, resulting in pain and swelling, the image of a bee is terrifying. For these people, seeing a bee might mean freezing, with an associated increase in heart rate, blood pressure, and skin conductance. Or, it might mean waving their arms or running away, with an increase in heart rate and skin conductance but a decrease in blood pressure. These are the sorts of physiological changes that we scientists record when we show study participants images from the International Affective Picture System (IAPS; Lang, Bradley, & Curthbert, 2008) stimulus set (e.g., Bradley, Codispoti, Cuthbert, & Lang, 2001). They arise during a prediction of how the body should respond in a specific situation (what we have previously referred to as an "affective prediction"; Barrett & Bar, 2009).

The fifth lesson from this example is that the process of making meaning of external sensations will always produce some kind of automatic change in your physical state. It is these internal sensations that likely form the basis of your pleasant or unpleasant core affective tone that accompanies any mental states of which it is a part (Barrett & Bliss-Moreau, 2009; Russell, 2003; Wundt, 1897); the actual visceral changes are not necessary for feeling, of course, although some representation of them in the brain is required. In the same way that your brain used prior experience to make meaning of the visual sensations in Figure 27.2, it will also use such knowledge to make meaning of these bodily sensations. These two meaning-making achievements are not happening in quick succession—they are occurring simultaneously, as a function of how the brain understands the current sensory array to create a unified conscious moment (cf. Barrett, 2009). They are not occurring in a single instant, but they are evolving over time. This meaning making rarely happens deliberately, but more often occurs as instantaneously, continuously, and effortlessly for internal sensations as it does for external sensations. These insights form the basis of our conceptual act theory of emotion (Barrett, 2006). You experience a perception of the situation versus an emotion as a function of your attentional focus. When sensations from the visual world are foregrounded (and sensations from the body are in the background), you experience the bee as friendly or wicked because you are focused on the bee, and not on how your body is responding to the bee (e.g., Anderson, Siegel, White, & Barrett, 2012). When sensations from the body are foregrounded, either because they are particularly intense, because such focus has been useful and reinforced in a prior situation like this one, or because you focus explicitly on them, you will experience either tranquility or distress.

The sixth lesson from this example is that prior experiences *seed* the construction of present and future experiences by shaping the meaning of momentary, incoming sensory input. Why might you automatically experience the calm of a bee buzzing in a bucolic garden, whereas another person might automatically experience the terror of a bee attacking and stinging the body? The answer lies in the nature of prior experience. Actual experiences with bees, movie scenes that involve bees, stories, or simply instruction about bees constitute the knowledge that is used to make sensations meaningful. Your learning history predisposes you to experience sensations from the world and from your own body in particular modal ways. All things being equal, you have developed experiential "habits"—what you have experienced in the past is very likely what you will experience in the present, because stored representations of the past help to constitute the present (hence, the phrase "the remembered present"; Edelman, 1998). It is now well known that the same brain network (termed the *default mode* or *mentalizing* network) involved in long-term memory is also important for imagining the future (Andrews-Hanna, Reidler, Sepulcre, Poulin, & Buckner, 2010), and recent evidence suggests that these networks are also important for constructing emotions in the present (Kober et al., 2008). It is very likely that when faced with the visual input in Figure 27.2 your brain reconstituted a number of different associations that were in competition with one another, and that via a variety of selection processes (Barrett, Tugade, & Engle, 2004; Sporns, Tononi, & Edelman, 2000a, 2000b), only one was fully realized (perhaps according to the logic of

constraint satisfaction) (e.g., Barrett, Ochsner, & Gross, 2007; Barsalou et al., 2003). The same logic might hold for making meaning of internal sensations. When is a high arousal state fear, or anger, or excitement? It might depend on your prior experience with these sensations in different contexts, and in particular, how those various instances were paired with emotion words such as *"fear," "anger,"* or *"excitement"* (cf. Barrett, 2006; Barrett, Lindquist, & Gendron, 2007). With additional learning or training, it should be possible to change your experiential habits. By deliberately cultivating certain types of experiences, it should be possible to modify the population of representations that are available for use in the present.

Situated Conceptualizations

In our prior writing, we have focused on the process of meaning making as a *psychological construction of emotion* that involves creating *situated conceptualizations* of internal bodily sensations that are highly context-dependent and coordinated with the immediate situation (cf. Barrett, 2006; Wilson-Mendenhall et al., 2011). In fact, however, the notion of a situated conceptualization implies that people are making meaning of both internal and external sensations at the same time, to create a unified conscious field (cf. Barrett, 2009). Conceptual knowledge is distributed throughout the brain's modal systems for perception and action in the form of simulators that reenact sensory, action, affect, and other elements of situations captured through experience (e.g., Barsalou, 1999; Simmons & Barsalou, 2003). From this perspective, knowledge is central to not only the cognition involved in thinking and imagining offline (reenacting or simulating a situation that is not present) but also to perceiving external sensations from the world and internal sensations from the body (in which case knowledge fuses with impinging sensory input—this fusion occurs seamlessly because knowledge is stored and represented in the same format or "language" as the sensations), both of which are involved in predicting and guiding one's actions. In this sense, conceptual knowledge is enactive. When your brain was foregrounding your bodily sensations while viewing the bee a few paragraphs earlier, for example, perhaps you experienced the moment as an emotion; when the focus was on the visual sensations, perhaps you experienced it merely as a perception of the bee. In each case, the visual input was the same—what differed was the situated conceptualization.

In the past, we have referred to the creation of situated conceptualizations as an act of categorization, because situated conceptualizations are enactments that develop for categories of experience (a variety of situated conceptualizations develop for the categories *fear, anger*, etc.; Barrett, 2006). To understand how situated conceptualizations work, then, it is important to understand how concepts and categories work. Categorization is not a narrow, limited process—it does not happen only when you explicitly attempt to assign an object to one grouping or another. Categorization plays a central role in all cognitive activity, including the sorts of high-level perceptions that are involved in emotion and emotion regulation. Categorizing is a fundamental cognitive activity. To categorize sensory input is to determine what it is, why it is, and what to do with it.

A central property of human knowledge is that it is organized categorically. Unlike a recording device that simply stores each individual, holistic, bitmapped image of the world, the human brain is constantly interpreting aspects of experience, using concepts in memory to make sensations meaningful. A concept can be viewed as aggregated memories that accumulate for a category across experiences with its instances. By focusing attention on some aspect of experience repeatedly, a concept develops over time from instances of the respective category experienced across situations (Barsalou, 1999; Barsalou & Hale, 1993; Murphy, 2002). The concept of *bee*, for example, aggregates diverse information about the category of bees across a variety of situations into a loosely organized representation that includes properties (e.g., yellow and black, with wings), relations (e.g., flowers), rules (for something to be a bee, it must have black and yellow stripes, it must fly, etc.), and exemplars (e.g., instances of honey bees, carpenter bees, a queen bee, etc.).[1] Concepts develop for all aspects of

human experience related to *bee*, including objects, settings, and actions (e.g., *flowers, honey, gardens, freezing, running, swatting, flying, buzzing, stinging*). From simpler concepts, more complex concepts emerge for events (e.g., *strolling in a garden, fear of the bee*). Concepts also develop for a wide variety of internal states (e.g., *aroused, quiet*), as well as for the properties and relations that describe instances of concepts (e.g., *yellow, fast, sweet, above, after, cause*). Although concepts reflect experience to a considerable extent, they undoubtedly have biological bases that scaffold learning (Barsalou, 1999, 2008; Carey, 2009; Rips, 2010; Simmons & Barsalou, 2003).

Extensive evidence now exists that an instance of conceptual knowledge (an instance of a concept, or a conceptualization) emerges from different multimodal systems in the brain (e.g., McClelland, 2010). Depending on the modalities relevant for processing a concept's instances, particular modal areas of the brain store information about the category and can later represent the category in the absence of actual instances. Martin (2001, 2007) reviews evidence, for example, that different multimodal profiles represent living versus nonliving things. Other research has similarly established the multimodal profiles that represent the self and others (cf. Legrand & Ruby, 2009; Northoff et al., 2006; Van Overwalle, 2009), people, buildings, and tools (e.g., Simmons, Reddish, Bellgowan, & Martin, 2010), the external world versus internal states (Golland, Golland, Bentin, & Malach, 2008), and so forth.

Category instances (e.g., a bee) are never encoded alone into conceptual knowledge, even though their context may not explicitly be the focus of attention. Initially, when encoding a category instance of a bee, for example, from actual prior experience with bees, observational learning about bees, hearing stories about bees, or being told rules about bees, the brain captures the elements of the setting in which the bee occurs (i.e., other agents and objects), internal sensory (i.e., somatovisceral) cues from the body, as well as actions, instructions from others (in the form of rules) and words (e.g., the phonological form for "bee"). Over time, these situated conceptualizations create a heterogeneous population of information that is available to represent new instances of the category "bee."[2] Later, when your brain requires conceptual knowledge to process some incoming sensory input, it samples from the populations of situated conceptualizations, associated with relevant concepts, to create a novel situated conceptualization, integrating current sensory input and retrieved (modal) conceptual knowledge (Barsalou, 2009). From this perspective, conceptual processing is actually more like scene perception, because the brain produces a conceptual state using multimodal information about entire situations. In this way, a situated conceptualization allows an experiencer to interpret incoming information and draw inferences that go beyond the information given.

It is impossible to have conscious access to the processes that create situated conceptualizations, because they are initiated in the first milliseconds of perception (or perhaps even before sensory input is actually encountered), and evolve over time, but it is possible to demonstrate the brain's computational power in creating them by engaging in a little imagination. For example, close your eyes and create an image of a yellow and black bee in your mind (i.e., simulate a bee). In doing this, your brain is creating a representation that includes the sights, sounds, smells, and so forth, of the bee, along with a situation in which the bee occurred, all of which would prepare you for a certain type of action (to run, to peer closer, to swat, to freeze). This representation involves the activation of neurons throughout your brain, including sensory and motor neurons, as well as neurons that regulate and represent an internal body (somatovisceral) state. All these elements (activation of sensory and motor neurons, changes in the physical state of the body, preparations for action) are examples of what it means to say that a representation is "embodied" (Barrett & Lindquist, 2008). This is also what it means to say that the brain is making a prediction about an object and how to act on it (Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012) or, as we noted before, to say that the brain is simulating a bee. This sort of simulation would occur if you were presented with an image of a bee and asked to recognize it (as in the prior section of this chapter), to categorize it explicitly (assign it

to one stimulus grouping over another), to judge it in some way, or to perform any kind of cognitive task with it. It would occur if you were being asked to remember a bee, talk about a bee, think about a bee, or when perceiving bees during an outdoor walk.

Once concepts become established in memory, they play central roles throughout cognition and perception (e.g., Barsalou, 2003; Murphy, 2002), and, as we suggest, emotion. As people experience incoming sensory input from the world and the body, they use prior experience to categorize the agents, objects, setting, behaviors, events, properties, relations, and bodily states that are present. As described in Wilson-Mendenhall et al. (2011), a situated conceptualization is the conceptualization of the current situation across parallel streams of conceptual processing for all of these elements. As information from the current situation registers simultaneously in these processing streams, conceptual systems on each of them categorize the respective information and draw inferences. At a more global level, abstract relational concepts integrate conceptualizations on the individual processing streams into coherent interpretations of larger events taking place across the situation as a whole. Categorical inferences (i.e., predictions) follow, including inferences about how an object, or entity, is likely to behave, how one can best interact with it, the likely value to be obtained from interacting with it, and so forth, and on a larger scale, about how situations may unfold during an event. From the perspective of grounded cognition, situated conceptualizations are responsible for producing the action, internal states, and perceptual construals that underlie goal-related activity in the current situation. Because modalities for action, internals states, and perceptual construals are typically active when a concept is learned, situated conceptualizations generate activity in these systems as they become active on later occasions. On activating the concept for *bee*, a situated conceptualization might activate representations of situation-specific approach–avoid actions (e.g., swatting the bee), representations of internal states such as pleasure or displeasure, and perceptual construals that direct the body toward a particular instance of pleasure or displeasure. Not only does *bee* represent perceptual instances of the concept, it also controls interactions and predicts the resultant events.

Emotions and Emotion Regulation as Situated Conceptualizations

Initial work on situated conceptualizations focused on how this theory of concepts can be applied to perceiving or interacting with concrete objects in relevant situations (for a review, see Barsalou, 2009), with conceptual knowledge represented using the brain's modal systems for perception, action, and internal bodily states. We further developed these ideas into a theory of emotion (Barrett, 2006) and a broader theory of mental states more generally (Barrett, 2009), although in the present discussion we focus on emotion. We hypothesize that situated conceptualizations have relevance for not only understanding the nature of emotion but also presenting a computational framework for understanding emotion regulation.

Emotions as Situated Conceptualizations

In our view, an emotion concept typically forms when a given emotion word (e.g., "fear") is explicitly uttered (e.g., by a caregiver or teacher) during many different instances involving a variety of changes in feelings, physiology, and actions, becoming the statistical regularity that holds the concept together across instances involving different sensory input and actions (cf. Barrett, Lindquist, et al., 2007). Selectively attending with some consistency to components of experience results in category knowledge that is captured in memory (Schyns, Goldstone, & Thibaut, 1998). Because emotions are abstract, language appears to guide selective attention to the changes in internal states that characterize an emotion in a given situation. For example, each time a parent (or some other person) labels a child's internal state or behavior with an emotion term, or a child observes the emotion term being used to label someone else's behavior, the child extracts information about that instance (including the phonological form of the word) and integrates it with past information associated with the same term that

is stored in memory. In this way, the phonological form for "fear" could become a perceptual regularity that occurs repeatedly across situations, and a concept *fear* forms (it is certainly the case that young infants can use abstract words to make conceptual inferences about objects that differ in their sensory properties; Dewar & Xu, 2009). The consequence is that accumulating conceptual knowledge for *fear*, for example, will vary within a person over instances as context and situated action demand. No single situated conceptualization for *fear* need give a complete account of the category *fear*. There is not one script for *fear* or one abstract representation for *fear*.[3] For example, fear might occur when excitedly declaring a risky bet, when lethargically sensing the first signs of flu, when frantically fleeing a blazing fire, or when casually flirting with an attractive stranger. On any given occasion, the content of a situated conceptualization for *fear* will be constructed to contain mainly those properties of *fear* that are contextually relevant, and it therefore contains only a small subset of the knowledge available in long-term memory about the category *fear*. [4] In a given instance, then, the situated conceptualization for *fear* has the potential to change the internal state of the perceiver, because when retrieving information about *fear*, sensory, motor, and interoceptive states are partially reinstated in the relevant aspects of cortex, simulating an instance.

We have hypothesized that concepts and categories for emotion work in essentially the same way as other kinds of abstract concepts in the conceptual system, where each individual situated conceptualization for a specific emotion (e.g., fear) refers to an entire situation, including both the internal and external sensations (Wilson-Mendenhall et al., 2011). In this way, emotions can be thought of as affective changes that are linked to the situation in some way (cf. Barrett, 2006; Clore & Ortony, 2008) and emotions can be said to reflect the structure of situations. The key hypothesis can be stated as follows: A momentary array of sensations from the world (light, sound, smell, touch, and taste) combined with sensations from the body (X) counts as perception of emotion (Y) during a situated conceptualization (C) (Barrett, 2012). Here, a "perception" is meant to indicate perceiving an instance of sensations in the self as the experience of emotion, or sensory input (from facial actions, voice, etc.) coming from others as emotional expressions. The meaning acquired by the sensations is not based solely on the physical properties of sensations alone (as body states or actions, as represented in the physiology of the body and/or in neural activations within the brain). Conceptual knowledge is required to give it additional functionality and meaning. For example, an increase in heart rate (X_1) counts as feeling afraid (Y_1) when category knowledge about *fear* is activated as a specific, embodied representation of *fear*, such as when a bee is attempting to sting you (C_1). In this example, the increase in heart rate takes on a meaning and allows a predicted behavior that it would not otherwise have alone. Emotion regulation might be characterized in the same way. A decrease in heart rate (X_2) counts as evidence of reappraisal (Y_2) when another embodied, situation-specific representation of a bee is activated, such as when it is floating above a brightly colored flower petal (C_2). In these examples, the concept *fear* might be applied to internal sensations from the body, because they are in the focus of attention, or because *fear* is part of a situated conceptualization that is part of the concept *bee*. It is a misnomer to refer to conceptual knowledge as merely psychological or social. For physical actions and body states (X) to count as an emotion (Y), some kind of physical change has to take place somewhere in the brain of a perceiver to complete a situated conceptualization in that perceiver at that moment in time (C). So a psychological construction approach makes predictions about the brain basis of emotion (and emotion regulation), but one that is different from the typical machine metaphor illustrated in Figure 27.1 and found in most natural kind models of emotion (also see Jones, Kirkland, & Cunningham, this volume). An instance of emotion is hypothesized to correspond to an entire brain state—or a series of states changing over time—including representations of the body and/or actions *and* the additional information that is necessary to create the new meanings that make emotions real, that is, the parts that are crucial for creating the situated conceptualizations.

In our view, then, changes in heart rate or blood pressure, facial actions such as

smiles or frowns, and behaviors such as crying or freezing, are not evidence of emotions in and of themselves. Instead, they become part of an emotional episode when they take on a certain meaning in a certain situation (Barrett, 2012). Via situated conceptualizations, physical changes acquire the ability to perform functions that they do not have on their own (creating social meaning, prescribing actions, allowing communication, aiding social influence). In this view, category knowledge about emotions does not *cause* emotions per se—it *constitutes* emotions by adding epistemologically novel functions to sensory input and action. Said another way, an emotion is constructed when embodied conceptual knowledge is enacted to shape the perception of sensory information from the body and the world, binding a physical state to an event in the world (as opposed to being merely a physical sensation or action). A body state or an action has a certain physical function (e.g., changes in respiration might regulate autonomic reactivity, or widened eyes might increase the size of the visual field), but these events do not intrinsically have certain functions *as an emotion*; events are assigned those functions in the act of categorizing them as emotion during the construction of a situated conceptualization.

If a situated conceptualization is represented as a distributed brain state (with both cortical and subcortical contributions), or even a series of brain state transitions across time, then mental causation is not mechanistic per se, but probabilistic, such that Brain State *A* at Time *T* (bee in the forest) increases the probability of Brain State *B* at Time *T* + 1 (fear of the bee expressed as a racing heart and sweaty hands and the perception of a stick as a weapon), making swatting more likely (but perhaps also a bee sting more likely) (Figure 27.3). Alternatively, the encounter with the bee might be followed by situated conceptualization (an image of bees making honey) as Brain State *C* at Time *T* + 1, decreasing the probability of a racing heart and sweating, and so forth. From this perspective, an emotion, such as fear, is itself not a *process* but instead represents a category of phenomena—a collection of instances of probabilistic situated conceptualizations. This example also illustrates that situated conceptualizations are not independent from one another in time—each occurs in a context of what came before, and what is predicted in the future.

Furthermore, we hypothesize that each situated conceptualization (as a brain state or series of states) can be understood as a construction of more basic, domain-general operations and their interactions. These operations can themselves be characterized at both the psychological level (e.g., Barrett,

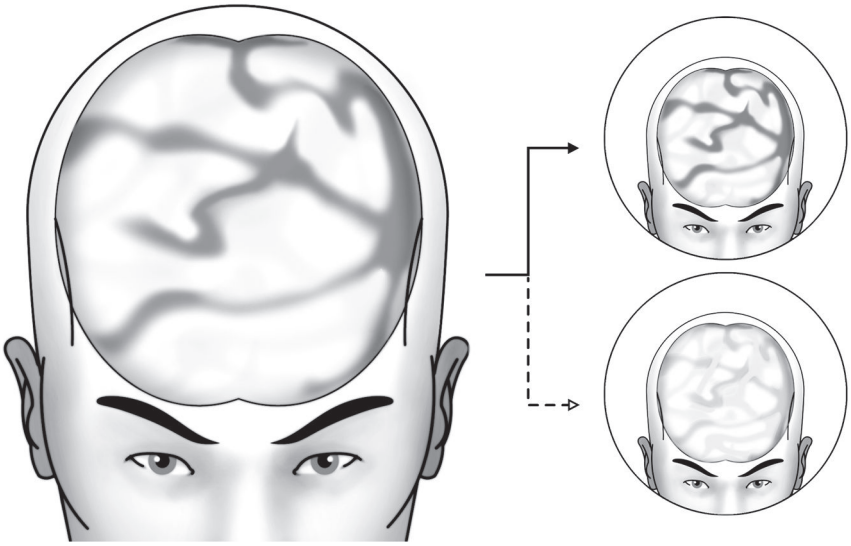

FIGURE 27.3. A depiction of the probabilistic state-space metaphor of brain function.

2006, 2012) and at the level of brain networks that emerge from neural integration across time and space within the brain (e.g., Barrett & Satpute, 2013; Lindquist & Barrett, 2012). Such basic operations are like the "mental state variables" (see Salzman & Fusi, 2010), facets or core systems that describe the brain state, or, to return to our kitchen metaphor, these can be thought of as the mind's "basic ingredients." Rather than presuming that these ingredients function in a modular, mechanistic way, each operation can be thought of as a set of "functional motifs" arising from the structural motif that undergirds each network (e.g., Sporns & Kotter, 2004). Moreover, if these operations serve as the functional architecture for how mental events and behaviors are constructed, then this implies that the science of emotion should focus on modeling emotions as high dimensional brain states (reflecting the engagement of domain-general networks, their internal operations, and their interactions). Such a componential, constructionist functional architecture of the human brain would not only reveal the distinctions between social, affect, and cognitive neuroscience to be artificial (Barrett & Satpute, 2013), but it would also present a set of hypotheses for how the phenomena that we refer to as *emotion* and *emotion regulation* are derived within a common mechanistic framework.

Emotion Regulation as Changing Situated Conceptualizations

To the extent that emotions are situated conceptualizations grounded in the modal systems of the brain, then shifting from one situated conceptualization to another intensifies, diminishes, or alters the autonomic and endocrine responses that underlie actions and feelings. We propose that a situated conceptualization framework offers an account of emotion regulation that undergirds the process model (Gross, this volume) at a different level of analysis, which has the potential to inspire new scientific research and practical applications. Our hypothesis is that stages of emotion regulation are often describing the difference between two consecutive situated conceptualizations rather than individual processes that can be chained together by a series of linear causal linkages in which cognitive systems in the brain modulate separate and anatomically distinct affective or emotional systems. Emotion regulation strategies *describe the changes* from one mental state to another, but these changes *can themselves be decomposed into more basic facets* (i.e., the mental operations and their associated networks that create the situated conceptualizations). As a consequence, the process model (Gross, this volume) can be thought of as offering a more abstract description of what occurs during emotion regulation, whereas the core systems that implement situated conceptualizations are a more mechanistic approach that produces reappraisals, distraction, suppression, and other instances of emotion regulation.

This distinction between levels of analysis maps onto the three levels of analysis described by Marr (1982), which include: a computational level that describes the phenomenon at hand (What problem does the system try to solve? What is the goal when transforming input to output?), an algorithmic level that describes how the transformation from input to output is achieved (How does the system do what it does? What are the representations used by the system? What processes act on these representations?) and an implementation or physical level (How is the system physically realized?). Our hypothesis is that the process model, with its emphasis on situation selection, situation modification, and so on, describes emotion regulation at Marr's computational level, whereas our situated conceptualization account describes emotion regulation at Marr's algorithmic and implementational levels. Each class of regulatory strategies discussed within the process model of emotion regulation can be understood as situated conceptualizations that are constructed from more basic domain-general core systems. The componential, constructionist functional architecture of the human brain for emotion is also the architecture that creates instances of emotion regulation.

Situation selection can be understood as a case in which situated conceptualizations are constructed to anticipate what will happen in the future. Elsewhere, Barrett and colleagues (2009; Barrett & Satpute, 2013; Lindquist & Barrett, 2012; Lindquist et al., 2012) have hypothesized that the nodes within the "men-

talizing" network (e.g., Andrews-Hanna et al., 2010) interact to help guide the construction of situated conceptualizations by integrating elements of prior experience (which are represented modally across the brain). These regions help to produce the multimodal simulations that are strongly situated in a particular background context, making sensory input meaningful and supporting specific courses of situated action. Using prior knowledge to simulate possible future situations may guide the decision making that underlies situation selection. More specifically, situated conceptualizations support simulating what it would be like to experience specific situations (by reenacting and reassembling prior knowledge) that produce information about their value and potential outcomes (e.g., deciding whether to walk in a meadow or a forest depending on the probability of encountering a bee). This hypothesis is consistent with the idea that the "mentalizing" network constructs mental models or simulations that facilitate future behavior (Buckner, 2011).

Because situated conceptualizations are dynamically constructed when thinking about future events, their dynamic assembly is likely influenced by a number of factors, including the current state of the individual (and the situated conceptualization that is being used to interpret this state) and the executive control resources available (e.g., via the "salience" and "frontoparietal control" networks) to help guide the situated conceptualization. These factors can influence elements of the situation that become the focus of the simulation and how detailed or vague the simulation becomes, both of which would impact the inferred outcomes and thus the decision making that underlies situation selection. The complexity inherent to situation selection is often acknowledged in the literature on emotion regulation (e.g., Gross, 2008), but the dynamics involved in creating such complexity remain unclear, perhaps because these discussions tend draw on traditional approaches to emotion that overlook such dynamics. In contrast, our approach highlights investigating the dynamic integration of multimodal facets involved in situated conceptualization as an important goal for future research.

The predictive and inferential capacities provided by situated conceptualizations also allow for situation modification (i.e., they provide a perceiver with the ability to predict what actions in the present will facilitate a change in mental state in the future; for example, using a branch as a weapon to swat a bee). A situated conceptualization approach draws attention to the underlying processes that facilitate or detract from taking actions to modify the external environment to alter its emotional impact. Because a situation is already in place during situation modification, an individual is drawing on prior knowledge about specific facets present in the situated conceptualization to modify the situation itself. For example, when the individual focuses on a nearby tree branch with the goal of killing the bee to avoid being stung, he or she now infers that the branch could be used a weapon because it can be manipulated much like a baseball bat or a fly swatter (i.e., prior knowledge about using a bat to strike an object is being dynamically applied within the situated conceptualization). In this way, the situated conceptualization used to interpret the environment is shifting dynamically as its multimodal facets change (e.g., heightened arousal and hyperfocus on the environment), which guides action.

Cognitive change (e.g., a stinging bee transformed into a flower-loving honey producer) and response modulation (e.g., to keep walking forward rather than to run away) naturally unfold as the brain shifts from one situated conceptualization to another, making predictions about how to act (i.e., a predicted action) and what the homeostatic and metabolic consequences will be (i.e., affective changes). Cognitive framing and response modulation are perhaps two of the most obvious goals of the brain's functional architecture—knowing what the current sensory array means and how to act on it. They are not unique to emotion regulation; they describe what happens during the construction of every mental state. During emotion regulation, though, an individual is often more aware of these changes because he or she has an explicit goal to regulate through cognitive change or response modulation. An important prediction of a situated conceptualization approach is that these types of changes can also occur without awareness if they become habitual. Bringing situated conceptualizations into awareness and

manipulating them with effort, intention, and a feeling of agency appears to be one key characteristic that distinguishes the mental events people refer to as "emotion regulation" (vs. those they refer to as "emotion").

If conceptual knowledge is enactive, and if situated conceptualizations actually have the capacity to shape the physiology and actions that are observed in any mental state, then changing a conceptualization via any core system can modify said physiology and action (as well as the feelings to which they give rise). Such regulation might occur when the same physical sensations and actions are conceptualized as a different emotion (e.g., tears are not sadness but anger; a racing heart is not fear but excitement), or when the intensity of physical activation is enhanced or reduced by changing the conceptualization (e.g., a *bee* means the pain of a sting or the tranquility of a meadow). In such cases, our hypothesis is that emotion regulation is the *result* of conceptual knowledge being activated as part of a situated conceptualization. At the level of subjective experience, it may feel as if a special mechanism is being used to down-regulate fear, such as reappraisal or suppression, or to up-regulate anger. Our hypothesis is that these emotion regulation constructs reflect changes in emotions (as mental states) that result from successive situated conceptualizations, using the same operations that constitute an emotion in the first place. Thus, an instruction to reappraise, to distract by shifting attention, and so forth, actually manipulates the underlying core systems of situated conceptualizations, which in turn alters the biological signals that produce sensations, not just how bodily sensations are understood (i.e., it alters the experience of them).

A situated conceptualization framework also suggests a novel hypothesis that is not discussed within the process model: Deconstructing an emotion, by attempting to undo its situated conceptualization, is another form of emotion regulation. When the perception and experience of physical sensations from the body are decoupled from the concept knowledge (e.g., a deactivated unpleasant feeling conceptualized more basically as fatigue or glucose depletion as opposed to sadness in a specific situation), they become less potent and result in less suffering. This suspension of conceptualization can be difficult to achieve, and it usually requires training. If you were to attempt to learn to paint an image of a bee on a flower, you would have to train yourself over a series of months not to see objects (a bee and flower) but to "undo" this perception and paint pieces of light. Only by doing this can you render a reasonable three-dimensional image on a two-dimensional page. Similarly, when learning to deconstruct emotion, you would have to train yourself not to experience emotions, but to experience physical sensations instead. Meditation practices offer a variety of tools for deconstructing emotion experience that may work in this manner (e.g., Holzel et al., 2011; Papies, Barsalou, & Custers, 2012). In other meditation approaches, an existing emotion is deconstructed and then is replaced with an alternative, more positive experience (Lutz, Brefczynski-Lewis, Johnstone, & Davidson, 2008; Lutz, Greischar, Perlman, & Davidson, 2009).

Recent empirical evidence from our laboratory also suggests that this may be a productive strategy. During a neuroimaging study (Wilson-Mendenhall et al., 2011), participants immersed themselves in affectively charged situations that involved either physical danger or social evaluation. After immersing in a situation, a word cued participants to emote in the situation (experience fear or anger) or to observe in the situation. Significantly greater activity in visual cortex and significantly less activity in medial prefrontal cortex occurred when participants experienced situations by observing the scenes from a third-person perspective rather than experiencing them as first-person, self-relevant emotions. These results suggest that sensations became the focus of the affectively charged situation when participants were observing in the situation. In this study, observing was an explicit goal, but a situated conceptualization approach suggests that this process of observing could eventually occur automatically (without effort) if repeatedly used in specific situations (e.g., potential anger-inducing situations).

Finally, psychotherapy—particularly cognitive-behavioral therapy (CBT) approaches—might be understood as helping clients to construct new situated conceptualizations (thereby modifying their conceptual system) that either reduce the intensity of their

physical responses, or better calibrate the constructed meaning of those responses to the situation at hand. CBT interventions also appear to provide training for when to use these alternative situated conceptualizations. The psychotherapeutic process might be thought of as creating a new population of learned neural assemblies (for the same emotion categories, or for new categories) that would be available to create new or different emotional meaning for the same sensations, or that would modify those sensations (particularly those related to the body). The emotional changes that occur with psychotherapy, then, might result from changes in the conceptual system, or how conceptual knowledge is used to construct the situated conceptualizations that are emotion.

Psychopathology and Dysregulation from a Situated Conceptualization Framework

From a situated conceptualization framework, psychopathology would result from two classes of problems. First, we hypothesize that emotional deficits within certain types of psychopathology (e.g., schizophrenia, autism spectrum disorders), and neurodegenerative diseases (e.g., frontotemporal dementia) arise due to damage to the brain's structural architecture for situated conceptualizations, making it difficult to construct meaning for sensory inputs that constitute normal mental states. Perhaps deficits in white matter connectivity, such as those seen in autism (Zikopoulous & Barbas, 2010), compromise the ability to construct and use the distributed conceptual structure that underlies situated conceptualizations. Similarly, autism is related to deficits in connectivity that develop during the final stages of cortical development in paralimbic areas, particularly within the supragranular layers of cortext (Zikopoulous & Barbas, 2010). These layers mainly contain the corticocortical connections that are important for synchronizing the distributed neuronal assemblies responsible for constructing normal situated conceptualizations.

In addition to structural considerations, psychopathology could result from entrenched conceptualizations that are overly ritualized and not sufficiently situation-specific. Deficits in the vocabulary or content of emotion concepts (perhaps due to poor socialization), or problems in accessing and using this knowledge (perhaps associated with problems with long-term memory or executive function) might result in a failure to regulate autonomic (and therefore affective) reactivity using situated conceptualizations, as in alexithymia (for a discussion, see Lindquist & Barrett, 2008), or might produce inappropriate or ritualized situated conceptualizations. To the extent that repeated processing of a situated conceptualization omits situational details and focuses instead on general abstract themes, chronic emotional responses develop that operate inappropriately across too many situations. For example, imagine that a situated conceptualization develops for shame associated with performing poorly on math tests during elementary school. Subsequently, if attention focuses too much on shame associated with poor performance and omits the situational details associated with elementary school math classes, a decontextualized shame for poor intellectual performance could develop that pervades experience inappropriately. Psychopathology also arise from problems in forming situated conceptualizations that are not well calibrated to the immediate situation, resulting in dysregulated autonomic responses and less effective actions. To the extent that situated conceptualizations are learned assemblies of perceptual, conceptual, interoceptive, and action processes, psychological disorders of one type or another might appear to be disorders of specific emotions (e.g., posttraumatic stress disorder [PTSD] as a disorder of fear) because a person's learning history has created a "conceptual habit" or regularity of certain situated conceptualizations, resulting in a sort of entrenchment of certain changes in bodily state, central representations of that state, and meanings that emerge regardless of the immediate situation (e.g., even when no actual threat is present).

More generally, psychopathology might also occur when sensations from the body are overly personalized and inaccurately construed to be self-evaluative as a function of how situated conceptualizations are constructed. Even an overreliance on such conceptual knowledge (e.g., conceptualizing interoceptive cues as psychological instead of

physical) would also result in psychopathology, and might help explain sex differences in certain disorders such as depression and anxiety. Problems might also arise when situated conceptualizations are too internally driven (from the body and prior experience) and insufficiently incorporate external sensory input, as in depression. For example, the subgenual portion of the anterior cingulate cortex (Brodmann Area 25), which is an important cortical site for regulating homeostasis and other autonomic functions (Ongur, Ferry, & Price, 2003), appears to keep the mentalizing network in a state of continual engagement via its connections to BA 10 in ventromedial prefrontal cortex; this region is part of the mentalizing network core (Andrews-Hanna et al., 2010) and is a hub in a range of phenomena, including homeostatic- and allostatic-related phenomena (e.g., subjective emotional experiences, autonomic and neuroendocrine function, reward, pain), social cognition and self-relevance, and memory-related phenomena (e.g., memory, mind-wandering, etc.) (Roy, Shohamy, & Wager, 2012). It is a region that allows brainstem autonomic function to be guided by conceptual information about past, present and future outcomes, and is important to the creation of affective meaning (Roy et al., 2012; Lindquist et al., 2012). This connection between BA 25 and BA 10 keeps depressed people locked "inside their own heads" and suffering a constant state of anguish that is marked by all consuming self-focus that paralyzes them. Deep brain stimulation to the white matter tract that connect these two regions in effect releases the mentalizing network, allowing people to experience relief and to focus more on events in the world (Mayberg, personal communication; also see Holtzheimer, Kelly, Gorss, Filkowski, Garlow, et al., 2012; Lujan, Chaturvedi, Choi, Holdzheimer, Gross, et al., 2013). From this research, it is easy to hypothesize that overly internally focused situated conceptualizations might occur because a person possesses a very reactive autonomic nervous system, producing frequent and intense internal sensations that demand conceptualization, or because of limited executive control resources. Moreover, conceptualizations that are not well-tailored to the situation in terms of sociocultural norms (which could happen in a variety of ways) could produce actions that are not effective in a particular cultural context.

To understand how the content of disordered situated conceptualizations emerge, it is important to focus on the mechanisms of the core systems or "ingredients" that create instances of emotion or implement the moment-to-moment changes that are experienced as emotion regulation, such as an overly active autonomic nervous system that is experienced as affective reactivity, and other related problems with attention, working memory, and context insensitivity (e.g., Kring & Moran, 2008; Poch & Campo, 2012; Williamson & Allman, 2012). These, in turn, can be understood in terms of the dynamics and structure of core networks, such as the "salience" network, the "frontoparietal control" network, and the "mentalizing" network (Barrett & Satpute, 2013). Indeed, these networks, which are intrinsic to the human brain and are structured by anatomical connectivity (and which we understand as the core systems that implement situated conceptualizations; Barrett & Satpute, 2013), are implicated in a range of psychopathologies, including not only schizophrenia, autism, and frontotemporal dementia, but also depression and anxiety disorders (Menon, 2011). For example, instead of understanding PTSD as an exaggerated activation of fear circuitry, it is possible that PTSD symptoms are the result of hyperaffective reactivity (associated with the "salience" network) combined with problems in conceptualization (associated with the "mentalizing" network) related to working memory deficits (associated with the "frontoparietal" control network) (Suvak & Barrett, 2011).

These ideas present an alternative to the traditional approach to mental illness, in which all forms of psychopathology (and many forms of physical illness, e.g., cardiovascular disease and cancer) are conceived of as involving either excessive or deficient amounts of one emotion or another. For example, various anxiety disorders, such as PTSD and panic disorder, are presumed to be disorders of fear, thought to arise from a hyperreactivity of fear processing. Depression is presumed to be a disorder of sadness and guilt. Hypertension is thought to involve an excess amount of anger. And so on. From

the perspective of the traditional machine metaphor, each type of illness would arise from problems with emotions being triggered too frequently or not enough. Also, the development and maintenance of psychiatric disorders involve problems in emotion regulation (Kring & Sloan, 2010), so psychopathology might also arise from an inability to regulate said emotions once they erupt. Our situated conceptualization approach connects these insights with transdiagnostic approaches that attempt to identify psychological and biological processes that are common to many types of psychological disorders (e.g., Fairholme, Boisseau, Ellard, Ehrenreich, & Barlow, 2010; Harvey, Watkins, Mansell, & Shafran, 2004; Haslam, 2002; Kendler, 2008; Kring, 2008; Krueger, Watson, & Barlow, 2005; Millan, 2003; Sanislow et al., 2010). Just as science is coming to the conclusion that emotion categories are not natural kinds (Barrett, 2006), clinical science is also coming to the conclusion that categories for disorders of emotion do not cut nature at its joints (Haslam, 2002; Kendler, 2008).

Summary

Situated conceptualizations can be thought of as cognitive tools used by the human brain to modify and regulate the body (i.e., homeostasis and allostasis, metabolism, and/or inflammatory processes), to create feelings, and to create dispositions toward action. In this sense, they provide an alternative framework for describing how mental states arise, and how actions and feelings, and the physiological changes that support them, are formulated and regulated. A focus on how situated conceptualizations are constructed from patterns (or functional motifs) across the brain's core systems (or structural motifs) adds utility to existing explanatory frameworks for emotion and emotion regulation by focusing on the mechanistic changes that produce emotional and regulatory phenomena to which we give abstract names.

Perhaps the final lesson of the bee example is that *states* and *processes* are easy to confuse when it comes to meaning making. Regardless of whether you automatically experience the calm of a bee buzzing in a bucolic garden or the terror of a bee attacking and stinging the body, it is possible to retrieve different associations of bees in the next instance, which in turn has the capacity to change the sensations that your brain receives from your body. Our hypothesis is that the same processes that were engaged during the initial instance of meaning making (creating tranquility or fear) are engaged again, and again, and again. When your bodily response changes, along with the corresponding feelings and actions, you experience this as emotion regulation. If this is correct, then what we call "emotion regulation" is grounded in the more basic meaning-making processes that are operating all the time to create the flow of mental states that constitutes your mind. Reappraisal, distraction, and other terms might refer not to processes per se, but to changes that occur as one mental state flows into another (and one physical state transitions to another) as meaning changes. A series of sequential mental states that are experientially distinct is easy to understand as distinct psychological processes, even though scientists have known for a long time that experiences don't reveal the processes that make them.

Notes

1. Hereafter in this chapter, we use italics to indicate a concept (e.g., *fear*) and quotes to indicate the word or phrase associated with it (e.g., "fear").
2. Theory and research strongly suggest that concepts do not have conceptual cores (i.e., information that is necessary and sufficient for membership in the associated category). Instead, concepts are represented with loose collections of situated exemplars that are related by family resemblance. Exemplar theories of categorization further illustrate that loose collections of memories for category members can produce sophisticated classification behavior, demonstrating that abstractions for prototypes and rules are not necessary. Neural net systems similarly demonstrate that only loose statistical coherence is necessary for sophisticated categorization. To the extent that abstraction does occur for a category, it may only occur partially across small sets of category instances, reflect the abstraction of nondefining properties and

relations that can be used to describe category members in a dynamical manner, or reflect an online abstraction at retrieval rather than stored abstractions in memory. Nevertheless, people often believe mistakenly that categories do have cores, perhaps because a word can lead people to essentialize.

3. As goal-directed categories that develop to guide action, the most typical member of a category such as *fear* is not the one that is most frequently encountered, but rather the one that maximally achieves the theme or goal of the category (Barsalou, 2003). As a result, the most typical instances of a category contain properties that represent the ideal form of the category, that is, whatever is ideal for meeting the goal around which the category is organized, not those that most commonly appear as instances of the category. From a situated conceptualization viewpoint, prototypes do not exist as stored representations in memory, but they can be constructed (or simulated) when needed (Barsalou et al., 2003).
4. Highly different instances for the same category can become integrated over time, and become available to construct novel simulations that have never been experienced before. This, in part, may help to explain why people believe that emotions such as anger, sadness, fear, and so on, have specific response signatures, even though the available data do not support this view. A simulation of *fear* could allow a person to go beyond the information given to fill in aspects of a internal sensation that are not present at a given perceptual instance. In such a case, the simulation essentially produces an illusory correlation between response outputs, helping to explain why researchers continue to search for coordinated autonomic, behavioral, and experiential aspects of a fear response.

References

Anderson, E., Siegel, E. H., White, D., & Barrett, L. F. (2012). Out of sight but not out of mind: Unseen affective faces influence evaluations and social impression. *Emotion, 12*, 1210–1221.

Andrews-Hanna, J. R., Reidler, J. S., Sepulcre, J., Poulin, R., & Buckner, R. L. (2010). Functional–anatomic fractionation of the brain's default network. *Neuron, 65*(4), 550–562.

Barrett, L. F. (2006). Solving the emotion paradox: Categorization and the experience of emotion. *Personality and Social Psychology Review, 10*(1), 20–46.

Barrett, L. F. (2009). The future of psychology: Connecting mind to brain. *Perspectives on Psycholical Science, 4*(4), 326–339.

Barrett, L. F. (2012). Emotions are real. *Emotion, 12*(3), 413–429.

Barrett, L. F., & Bar, M. (2009). See it with feeling: Affective predictions in the human brain. *Philosphical Transactions of the Royal Society B: Biological Sciences, 364*, 1325–1334.

Barrett, L. F., & Bliss-Moreau, E. (2009). Affect as a psychological primitive. *Advances in Experimental Social Psychology, 41*, 167–218.

Barrett, L. F., & Gross, J. J. (2001). Emotion representation and regulation: A process model of emotional intelligence. In T. Mayne & G. Bonnano (Eds.), *Emotion: Current issues and future directions* (pp. 286–310). New York: Guilford Press.

Barrett, L. F., & Lindquist, K. (2008). The embodiment of emotion. In G. Semin & E. Smith (Eds.), *Embodied grounding: Social, cognitive, affective, and neuroscience approaches* (pp. 237–262). New York: Cambridge University Press.

Barrett, L. F., Lindquist, K. A., & Gendron, M. (2007). Language as context for the perception of emotion. *Trends in Cognitive Sciences, 11*(8), 327–332.

Barrett, L. F., Ochsner, K. N., & Gross, J. J. (2007). On the automaticity of emotion. In J. Bargh (Ed.), *Social psychology and the unconscious: The automaticity of higher mental processes* (pp. 173–217). New York: Psychology Press.

Barrett, L. F., & Satpute, A. (2013). Large-scale brain networks in affective and social neuroscience: Towards an integrative architecture of the human brain. *Current Opinion in Neurobiology*, 23, 1–12.

Barrett, L. F., Tugade, M. M., & Engle, R. W. (2004). Individual differences in working memory capacity and dual-process theories of the mind. *Psychological Bulletin, 130*(4), 553–573.

Barsalou, L. W. (1999). Perceptual symbol systems. *Behavioral and Brain Sciences, 22*(4), 577–609.

Barsalou, L. W. (2003). Situated simulation in the human conceptual system. *Language and Cognitive Processes, 18*, 513–562.

Barsalou, L. W. (2008). Grounded cognition. *Annual Review of Psychology, 59*, 617–645.

Barsalou, L. W. (2009). Simulation, situated conceptualization, and prediction. *Philosophical Transactions of the Royal Society B: Biological Sciences, 364*(1521), 1281–1289.

Barsalou, L. W., & Hale, C. R. (1993). Components of coceptual representation: From feature lists to recursive frames. In J. Hampton, R. Michalski, & P. Theuns (Eds.), *Categories and concepts: Theoretical views and inductive data analysis* (pp. 97–144). San Diego, CA: Academic Press.

Barsalou, L. W., Niedenthal, P. M., Barbey, A., & Ruppert, J. (2003). Social embodiment. In B. Ross (Ed.), *The psychology of learning and motivation* (Vol. 43, pp. 43–92). San Diego, CA: Academic Press.

Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and motivation I: Defensive and appetitive reactions in picture processing. *Emotion, 1*(3), 276–298.

Buckner, R. L. (2011). The serendipitous discovery of the brain's default network. *NeuroImage, 62*, 1137–1145.

Carey, S. (2009). *The origin of concepts.* New York: Oxford University Press.

Clore, G. L., & Ortony, A. (2008). Appraisal theories: How cognition shapes affect into emotion. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *Handbook of emotions* (3rd ed., pp. 628–642). New York: Guilford Press.

Dewar, K. M., & Xu, F. (2009). Do early nouns refer to kinds or distinct shapes?: Evidence from 10-month old infants. *Psychological Science, 20*, 252–257.

Edelman, G. M. (1998). *The remembered present: A biological theory of consciousness.* New York: Basic Books.

Fairholme, C. P., Boisseau, C. L., Ellard, K. K., Ehrenreich, J. T., & Barlow, D. H. (2010). Emotions, emotion regulation, and psychological treatment: A unified perspective. In A. M. Kring & D. M. Sloan (Eds.), *Emotion regulation and psychopathology* (pp. 283–309). New York: Guilford Press.

Fine, I., Wade, A. R., Brewer, A. A., May, M. G., Goodman, D. F., Boynton, G. M., et al. (2003). Long-term deprivation affects visual perception and cortex. *Nature Neuroscience, 6*, 915–916.

Golland, Y., Golland, P., Bentin, S., & Malach, R. (2008). Data-driven clustering reveals a fundamental subdivision of the human cortex into two global systems. *Neuropsychologia, 46*(2), 540–553.

Gross, J. J. (2008). Emotion regulation. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *Handbook of emotions* (3rd ed., pp. 497–512). New York: Guilford Press.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Harvey, A., Watkins, E., Mansell, W., & Shafran, R. (2004). *Cognitive behavioural processes across psychological disorders: A transdiagnostic approach to research and treatment.* New York: Oxford Univeristy Press.

Haslam, N. (2002). Kinds of kinds: A conceptual taxonomy of psychiatric categories. *Philosophy, Psychiatry, and Psychology, 9*, 203–217.

Holzel, B. K., Lazar, S. W., Gard, T., Schuman-Oliver, Z., Vago, D. R., & Ott, U. (2011). How does mindful meditation work?: Proposing mechanisms of action from a conceptual and neural perspective. *Perspectives on Psychological Science, 6*(6), 537–559.

Holtzheimer, P. E., Kelley, M. E., Gross, R. E., Filkowski, M. M., Garlow, S. J., Barrocas, A., et al. (2012). Subcallosal cingulate deep brain stimulation for treatment-resistant unipolar and bipolar depression. *Archives of General Psychiatry, 69*(2),150–158.

Jones, C. R., Kirkland, T., & Cunningham, W. A. (this volume, 2014). Attitudes, evaluation, and emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 251–266). New York: Guilford Press.

Kendler, K. S. (2008). Explanatory models for psychiatric illness. *American Journal of Psychiatry, 165*(6), 695–702.

Kober, H., Barrett, L. F., Joseph, J., Bliss-Moreau, E., Lindquist, K., & Wager, T. D. (2008). Functional grouping and cortical–subcortical interactions in emotion: A meta-analysis of neuroimaging studies. *NeuroImage, 42*(2), 998–1031.

Kring, A. M. (2008). Emotion disturbances as transdiagnostic processes in psychopathology. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *Handbook of emotion* (3rd ed., pp. 691–705). New York: Guilford Press.

Kring, A. M., & Moran, E. K. (2008). Emotional response deficits in schizophrenia: Insights from affective science. *Schizophrenia Bulletin, 34*(5), 819–834.

Kring, A. M., & Sloan, D. M. (Eds.). (2010). *Emotion regulation and psychopathology.* New York: Guilford Press.

Krueger, R. F., Watson, D., & Barlow, D. H. (2005). Introduction to the special section: Toward a dimensionally based taxonomy of psychopathology. *Journal of Abnormal Psychology, 114*(4), 491–493.

Lang, P. J., Bradley, M. M., & Curthbert, B. N. (2008). *International affective picture system (IAPS): Affective ratings of pictures and instruction manual.* Gainsville: University of Florida.

Legrand, D., & Ruby, P. (2009). What is self-specific?: Theoretical investigation and critical review of neuroimaging results. *Psychological Review, 116*(1), 252–282.

Lindquist, K., & Barrett, L. F. (2008). Emotional complexity. In M. Lewis, J. M. Haviland-Jones & L. F. Barrett (Eds.), *Handbook of emotion* (pp. 513–530). New York: Guilford Press.

Lindquist, K. A., & Barrett, L. F. (2012). A functional architecture of the human brain: Insights from the science of emotion. *Trends in Cognitive Sciences, 16*, 533–540.

Lindquist, K. A., Wager, T. D., Kober, H., Bliss-Moreau, E., & Barrett, L. F. (2012). The brain basis of emotion: A meta-analytic review. *Behavioral and Brain Sciences, 35*(3), 121–143.

Lujan, J. L., Chaturvedi, A., Choi, K. S., Holdzheimer, P. E., Gross, R. E., Mayberg, H. S., et al. (2013). Tractography–activation models applied to subcallosal cingulate deep brain stimulation. *Brain Stimulation.*

Lutz, A., Brefczynski-Lewis, J., Johnstone, T., & Davidson, R. J. (2008). Regulation of the neural circuitry of emotion by compassion meditation: Effects of meditative expertise. *PLoS ONE, 3*(3), e1897.

Lutz, A., Greischar, L. L., Perlman, D. M., & Davidson, R. J. (2009). BOLD signal in insula is differentially related to cardiac function during compassion meditation in experts vs. novices. *NeuroImage, 47*(3), 1038–1046.

Martin, A. (2001). Functional neuroimaging of semantic memory. In R. Cabeza & A. Kingstone (Eds.), *Handbook of functional neuroimaging of cognition* (pp. 153–186). Cambridge, MA: MIT Press.

Martin, A. (2007). The representation of object concepts in the brain. *Annual Review of Psychology, 58*, 25–45.

Marr, D. (1982). *Vision: A computational investigation into the human representation and processing of visual information.* New York: Freeman.

McClelland, J. L. (2010). Emergence in cognitive science. *Topics in Cognitive Science, 2*, 751–770.

Menon, V. (2011). Large-scale brain networks and psychopathology: A unifying triple network model. *Trends in Cognitive Sciences, 15*(10), 483–506.

Mesquita, B., Barrett, L. F., & Smith, E. (Eds.). (2010). *The mind in context.* New York: Guilford Press.

Millan, M. J. (2003). The neurobiology and control of anxious states. *Progress in Neurobiology, 70*(2), 83–244.

Murphy, G. L. (2002). *The big book of concepts.* Cambridge, MA: MIT Press.

Neumeister, A., Henry, S., & Krystal, J. H. (2007). Neural circuitry and neuroplasticity in PTSD. In M. J. Friedman, T. M. Keane, & P. A. Resick (Eds.), *Handbook of PTSD: Science and practice* (pp. 151–165). New York: Guilford Press.

Northoff, G., Heinzel, A., de Greck, M., Bermpohl, F., Dobrowolny, H., & Panksepp, J. (2006). Self-referential processing in our brain—A meta-analysis of imaging studies on the self. *NeuroImage, 31*(1), 440–457.

Ongur, D., Ferry, A. T., & Price, J. L. (2003). Architectonic subdivision of the human orbital and medial prefrontal cortex. *Journal of Comparative Neurology, 460*, 425–449.

Papies, E. K., Barsalou, L. W., & Custers, R. (2012). Mindful attention prevents mindless impulses. *Social Psychological and Personality Science 3*, 291–299.

Poch, C., & Campo, P. (2012). Neocortical–hippocampal dynamics of working memory in healthy and diseased brain states based on functional connectivity. *Frontiers in Human Neuroscience, 6*, 36.

Rips, L. J. (2010). *Lines of thought.* New York: Oxford University Press.

Roy, M., Shohamy, D., Wager, T. D. (2012). Ventromedial prefrontal–subcortical systems and the generation of affective meaning. *Trends in Cognitive Science, 16*, 147–156.

Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review, 110*, 145–172.

Salzman, C. D., & Fusi, S. (2010). Emotion, cognition, and mental state representation in amygdala and prefrontal cortex. *Annual Review of Neuroscience, 33*, 173–202.

Sanislow, C. A., Pine, D. S., Quinn, K. J., Kozak, M. J., Garvey, M. A., Heinssen, R. K., et al.

(2010). Developing constructs for psychopathology research: research domain criteria. *Journal of Abnormal Psychology, 119*(4), 631–639.

Schell, T. L., Marshall, G. N., & Jaycox, L. H. (2004). All symptoms are not created equal: The prominent role of hyperarousal in the course of posttraumatic psychological distress. *Journal of Abnormal Psychology, 113*(2), 189–197.

Schyns, P. G., Goldstone, R. L., & Thibaut, J. P. (1998). The development of features in object concepts. *Behavioral and Brain Sciences, 21*(1), 1–17.

Simmons, W. K., & Barsalou, L. W. (2003). The similarity-in-topography principle: Reconciling theories of conceptual deficits. *Cognitive Neuropsychology, 20*(3), 451–486.

Simmons, W. K., Reddish, M., Bellgowan, P. S., & Martin, A. (2010). The selectivity and functional connectivity of the anterior temporal lobes. *Cerebral Cortex, 20*(4), 813–825.

Sporns, O., & Kotter, R. (2004). Motifs in brain networks. *PLoS Biology, 2*, e369.

Sporns, O., Tononi, G., & Edelman, G. M. (2000a). Connectivity and complexity: The relationship between neuroanatomy and brain dynamics. *Neural Networks, 13*(8–9), 909–922.

Sporns, O., Tononi, G., & Edelman, G. M. (2000b). Theoretical neuroanatomy: Relating anatomical and functional connectivity in graphs and cortical connection matrices. *Cerebral Cortex, 10*(2), 127–141.

Suvak, M. K., & Barrett, L. F. (2011). Considering PTSD from the perspective of brain processes: A psychological construction approach. *Journal of Traumatic Stress, 24*(1), 3–24.

Van Overwalle, F. (2009). Social cognition and the brain: A meta-analysis. *Human Brain Mapping, 30*(3), 829–858.

Williamson, P. C., & Allman, J. M. (2012). A framework for interpreting functional networks in schizophrenia. *Frontiers in Human Neuroscience, 6*, 184.

Wilson-Mendenhall, C. D., Barrett, L. F., & Barsalou, L. W. (in preparation). *Emoting vs. observing: Conceptualizing affective situations as self-relevant emotions or sensory-focused mental states.*

Wilson-Mendenhall, C. D., Barrett, L. F., Simmons, W. K., & Barsalou, L. W. (2011). Grounding emotion in situated conceptualization. *Neuropsychologia, 49*(5), 1105–1127.

Wundt, W. (1897). *Outlines of psychology* (C. H. Judd, Trans.). Leipzig: Wilhelm Engelmann.

Zikopoulous, B., & Barbas, H. (2010). Changes in prefrontal axons may disrupt the network in autism. *Journal of Neuroscience, 30*, 14595–14609.

PART VIII

INTERVENTIONS

CHAPTER 28

Emotion Regulation Therapy

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Over the past 25 years, considerable progress has been achieved in treating adult psychiatric disorders (e.g., Butler, Chapman, Forman, & Beck, 2006). Despite these significant advances, a sizable subgroup of individuals with commonly co-occurring disorders such as generalized anxiety disorder (GAD) and major depressive disorder (MDD) fail to make sufficient treatment gains, thereby prolonging their deficits in life functioning and satisfaction. For example, in a meta-analysis of cognitive-behavioral therapy (CBT) for GAD, Borkovec and Ruscio (2001) found that only 50–60% of treated individuals demonstrated clinically meaningful change. Furthermore, although more efficacious, the difference between current psychological treatments for MDD and nondirective supportive therapy reflects only a small effect size (Cuijpers, van Straten, Andersson, & van Oppen, 2008). Also, among individuals who have GAD with comorbid MDD, gains in treating depression are not especially durable (Newman, Przeworski, Fisher, & Borkovec, 2010). Finally, in the recently concluded Sequenced Treatment Alternatives to Relieve Depression Study,funded by the National Institute of Mental Health (NIMH), the subgroup of individuals with mixed anxiety–depressive disorder (e.g., MDD + apprehensive anxious symptoms) was most treatment refractory (Farabaugh et al., 2010).

One characteristic common to these disorders, which are often comorbid (Kessler, Berglund, Demler, Jin, & Walters, 2005), is that they both strongly (more so than other conditions) reflect heightened emotional experience (often referred to as *emotionality, neuroticism*, or *emotional intensity*; see Barlow, 2002; Mennin, Heimberg, Turk, & Fresco, 2005; Mennin, Holaway, Fresco, Moore, & Heimberg, 2007). Factor-analytic evidence demonstrates that these disorders jointly reflected a higher order factor of prolonged negative affect or *distress* that has been distinguished from a "fear" factor that relates to disorders more characterized by acute arousal (e.g., Krueger & Markon, 2006; Watson, 2005). Furthermore, both GAD and MDD possess heritability factors reflective of increased emotionality (e.g., Kendler, Gardner, Gatz, & Pedersen, 2006). One possibility is that the "distress" in these disorders reflects an inherent dispositional tendency for heightened emotional salience related to motivational impetuses such as the need to avoid threat. These underlying emotional and motivational factors may be more difficult to ameliorate and change (Brown, 2007). Indeed, these higher order emotional factors, in particular, have been found to

account for the underperformance of otherwise efficacious treatments (Olatunji, Cisler, & Tolin, 2010).

Repetitive or perseverative thought (Watkins, 2008) represents another characteristic common to the distress disorders that may arise as a desperate means of coping or compensating with strongly felt emotional experiences. For instance, pathological worry (Borkovec, Alcaine, & Behar, 2004) functions as a regulatory strategy aimed at reducing distress that arises from conflicting emotional and motivational states (e.g., Mennin & Fresco, 2009; Newman & Llera, 2011). Similarly, *depressive rumination* represents a perseverative cognitive process commonly defined as repetitive thinking about past mistakes and failures, and is strongly associated with worry (e.g., Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Watkins, 2008). In essence, individuals with distress disorders may be more prone to utilize perseverative cognitive strategies (e.g., worry, rumination) to escape or dampen emotionality at the cost of accurately gleaning the motivational message that is being conveyed, undermining immediate behavioral action in response to their emotions, and ultimately, losing sight of the enriching and fulfilling aspects of life.

Thus, given the emerging profile of this difficult to treat, distress-disordered patient (e.g., strong motivational impetuses accompanied by excessive reliance on compensatory strategies to escape or avoid these impetuses reactively, and impoverished contextual learning repertoires), there is increasing interest in understanding the role of emotions and how this knowledge might generate new targets for intervention, particularly for more treatment-resistant cases. The affective science field provides an opportunity to expand paradigms regarding the role of emotion-related processes in conceptualizing and treating psychopathology. Increasingly, treatments have adopted a functional approach to emotions to improve existing interventions a variety of disorders (e.g., Barlow et al., 2011; Linehan, 1993).

Articulating a model of distress disorders predicated on underlying emotional and motivational mechanisms and processes aligns with two recent initiatives promoted by the NIMH with the goal of accelerating the payoff from basic and translational research into treatment application and identifying factors that promote or diminish the effectiveness of evidence-based treatments. The first of these, called the Research Domain Criteria Initiative (RDoC; Craske, 2012), aims to understand what is expected in these domains at different levels of inquiry, so that these normative findings can be contrasted with disordered subgroups to identify mechanistic regions of interest that may in turn become the targets of treatment development. The second initiative comes from a recent NIMH's National Advisory Mental Health Council report emphasizing "treatment personalization" as a means to identify factors that predict who will benefit from a given intervention process, then to determine systematically ways of matching patients to a particular treatment, as well as to optimize and augment care. Taken together, these initiatives highlight the need to specify transdiagnostic mechanisms within individuals to further the development of treatment processes that specifically target these mechanisms.

Congruent with current directions in affect science, these NIMH initiatives, and advances in the science of behavior therapy, we have developed an emotion regulation therapy (ERT; Mennin & Fresco, 2009) to improve treatment of distress disorders including GAD and MDD. ERT integrates traditional and contemporary CBT principles and practices, and emotion-focused interventions within a framework that reflects basic and translational findings in affect science. The result is a theoretically derived, evidence-based treatment that builds on the solid foundation of CBT by identifying and targeting putative mechanisms common to the distress disorders (e.g., motivational–emotional activation, perseverative thinking, and resultant narrowed learning repertoires) while striving to normalize emotion generation and regulation functioning. In the remainder of this chapter, we (1) describe and review relevant work related to normative and disordered motivation, regulation, and contextual learning consequences; (2) demonstrate how ERT reflects an approach to treatment that draws from common principles of CBT and affective science to target proposed mechanisms directly and provide preliminary efficacy and mechanism findings; and (3) provide an overview of future directions for ERT.

Normative and Disordered Emotional Processing and Learning Consequences

An affect science perspective on adult psychopathology and its treatment includes (1) *motivational mechanisms*, reflecting the functional and directional properties of an emotional response tendency; (2) *regulatory mechanisms*, reflecting the alteration of response trajectories to be more congruent with contextual demands and constraints, as well as one's personal values or goals; and (3) *contextual learning consequences*, reflecting, optimally, the promotion of broad and flexible behavioral repertoires. In distress disorders, dysfunction may occur via strong motivational salience and conflict, as well as deficits in systems of regulation, which may result in diminished, narrow, and rigid behavioral repertoires. In the remainder of this section, we discuss both a normative account of these characteristics and how these normative processes may go awry in the emotional dysfunction of distress disorders.

Normative Emotional Functioning

Motivation

One of our most basic, primary directives is to bring balance with respect to engaging reward and minimizing loss, while seeking safety and avoiding threat (Dollard & Miller, 1950). As we increasingly become creatures of habit over the course of our lives, we are continually pushed and pulled by motivations to maintain security and gain reward. Our actions and preparation for actions are guided by the motivational salience of the stimuli we encounter (Gray & McNaughton, 2000). In particular, a *reward system* mobilizes a behavioral approach toward rewarding or appetitive stimuli or to minimize loss. Reward can be further specified in relation to consummatory pleasure (i.e., *liking*, the hedonic impact that a reward produces) and anticipatory pleasure (i.e., *wanting*, the incentive salience associated with a particular reward; Berridge, Robinson, & Aldridge, 2009). These two facets of the reward system have shared (e.g., nucleus accumbens, comprising a large part of a region called the ventral striatum; Stein & Paulus, 2009) and dissociable (e.g., the orbitofrontal cortex [OFC], involved in assigning reward values to stimuli; Berridge et al., 2009) neurobehavioral correlates. In terms of neurochemistry, liking is primarily associated with endogenous opioids and endorphins (Stein & Paulus, 2009), whereas wanting chiefly involves dopamine, which is involved in marshaling organisms to exert the effort required to obtain a reward (Berridge et al., 2009; Stein & Paulus, 2009).

By contrast, a *security system* instigates avoidance of novel, potentially threatening, or painful stimuli or end states, as well as engagement of safety stimuli that protect an individual from such perceived threats and can reinstate a state of quiescence and calm. This system encompasses both reflexive motivational behavior and reflective, goal-setting, behavior (Carver, Avivi, & Laurenceau, 2008; Gray & McNaughton, 2000; Higgins, 1997) aimed toward avoidance of threats and engagement of safety signals. At the neural level, whereas the amygdala is activated in response to novel and immediate threats, the bed nucleus of the stria terminalis is activated in response to more distal or prolonged threats (Lang, Davis, & Öhman, 2000). Furthermore, the ventromedial prefrontal cortex (vmPFC) is involved in higher order processing of goals reflective of safety seeking and threat avoidance. Neurochemically, norepinephrine and gamma-aminobutyric acid (GABA) are centrally involved in threat detection and safety signaling (Stein & Paulus, 2009).

The security and reward systems are relatively independent and can be activated alone or in unison, in response to a prompt (Stein & Paulus, 2009). In essence, normative functioning represents a constant state of engaging and resolving situations that provoke conflicts of motivational systems in the service of taking effective behavioral action. Stein and Paulus suggest that the insula, in relation to the vmPFC, may be important for detecting motivational conflict and helping to shape, and be shaped by, goal intentions and implementation. Similarly, Aupperle and Paulus (2010) propose that optimal balancing of approach and avoidance systems is coordinated by a network consisting of the OFC (valuation of stimuli in service of making choices; inhibiting limbic regions and behavioral responses

during fear processing), the ventral striatum (orientation toward reward), amygdala (providing salience/intensity of fear and avoidance stimuli), and the insula (monitoring current and predicting future interoceptive states in relation to reward and fear). Correspondingly, engaging behavioral actions to achieve or restore motivational balance likely consists of responding in a manner reflecting a contextually and situationally appropriate balance of reward and safety–threat systems, while also informing one's actions with higher order, values-based decision making (Wilson & Murrell, 2004). Attaining goals that reflect motivational salience and one's personal values provides feedback relating one's behavioral effort to an outcome.

Regulation

Emotions are part of a larger self-regulation system that allows us to respond flexibly to events in our lives in accordance with both personal goals/values and changing contexts. In some instances, the optimal tuning in a given situation results in the accentuation (i.e., up-regulation) of the emotional salience of the situation; in other instances, toning down (i.e., dampening) the emotional aspects of the situation is warranted. Neurobiological evidence supports the notion that there are multiple pathways to emotion generation, including automated, "hard-wired" or lower order systems (largely involving physiological responses and their subcortical control) and more controlled, higher order systems (largely involving subjective, cortical responses) that are separate but interactive and mutually essential for differing aspects of emotional experience (LeDoux, 1996). These higher order and lower order neural systems also actively regulate each other (LeDoux, 1996). A functional systems approach to emotion regulation argues that these systems work together to maintain dynamic homeostasis between bodily systems and internal and external stimuli in a context-appropriate manner.

Gross (2002) highlights the importance of the temporal unfolding of emotional experience in understanding how individuals self-regulate. Specifically, one can impact an emotional event via early selection or modification of a situation, early stages of emotional processing chiefly involving attention, later stages of emotional processing chiefly involving verbal representation and cognitive change, or, once an emotional response has occurred, by attempting to impact the quality of that response. Influencing the information-processing aspects of an emotion (i.e., antecedent-focused strategies) is more effective than acting on the products of that processing, such as physiological responses (i.e., response-focused strategies). Building on these structural and temporal characteristics, regulation efforts can be differentiated by degree of elaboration, which essentially refers to the degree of cognitive effort required to engage a particular capacity, from less elaborative regulatory components, such as attention, to more elaborative regulatory components, such as working memory and verbal representations (Badre & D'Esposito, 2007), which, at the neural level, are associated with greater recruitment of dorsal and lateral areas of the PFC (Ochsner, Bunge, Gross, & Gabrieli, 2002). Activation of these elaborative areas is associated with greater mental effort and can result in greater resource depletion given competition for these resources (Muraven & Baumeister, 2000). Optimal emotion regulation may begin by engaging less elaborative capacities, followed by more elaborative capacities as needed (Sheppes & Gross, 2011). Nonetheless, both less elaborative and more elaborative regulation efforts modify security- and reward-based motivational patterns according to contextual demands (e.g., Delgado, Gillis, & Phelps, 2008; Sokol-Hessner, Camerer, & Phelps, 2013).

Broad and Flexible Contextual Learning

Adaptation refers to the process by which an organism becomes better suited to prospering in their habitats (Dobzhansky, 1970). Knowing and providing the contextually appropriate behavioral response may mean the difference between life and death, and between love and loss. Adaptive and flexible behavioral responses are dependent on the ability to increase awareness of cues and contingencies in the environment and respond in ways to promote survival and success. Adap-

tive motivational responses and regulatory capacities provide a foundation for behavioral flexibility in that they help us attain maximal emotional clarity (e.g., Gohm & Clore, 2002) and subsequently implement effective and goal-relevant responses for optimal behavioral outcomes.

Optimal reward learning requires us to take behavioral actions that are informed by the assignment of value to possibly rewarding stimuli and subsequent predictions about when and where we might encounter these stimuli (O'Doherty, 2004). Bogdan and Pizzagalli (2006) have examined factors such as reward sensitivity (increased likelihood of responding to "rich" rewarding stimuli based on past learning history) as evidence of the influence of emotion and accurate cue detection on reward learning and behavior. Similarly, reliably detecting and responding to cues that signal a clear and present danger are crucial for survival (LeDoux, 1996). However, equally important is learning to detect safety cues accurately and differentiate these signals from threat, so that we do not expend valuable resources (e.g., time and energy) in attempts to escape from "nonthreats." Contemporary models of threat and safety learning are predicated on principles of Pavlovian conditioning and the knowledge that successful fear extinction represents new, inhibitory learning (Bouton, Mineka, & Barlow 2001). Emotion regulation plays an important role in inhibitory learning via selection of optimal responses that promote abolishment of a conditioned emotional response. Commensurately, activation in the vmPFC, an area central to implementation of emotion regulation intentions, has been found to decrease during acquisition of conditioned responses and increase during fear extinction (Schiller & Delgado, 2010).

Dysfunctional Emotional Functioning

Motivational Dysfunction

Individuals with distress disorders may be subject to frequent conflicting pulls from reward and safety–threat systems. However, unlike healthy individuals, they may be relatively less effective in resolving these motivation conflicts. Drawing upon the Aupperle and Paulus (2010) model of motivation balancing, individuals with anxiety and mood disorders likely struggle to overcome motivational conflicts because they possess one or more of the following deficits: (1) an over-representation of safety–threat valuation as reflected in limbic (amygdala, insula) overactivation; (2) over- or underrepresentation of approach valuation as a function of either attenuated or exaggerated striatal activation, respectively; and (3) insufficient mediation or arbitration of approach and/or avoidance valuations resulting from attenuated OFC and vmPFC activation. Klenk, Strauman, and Higgins (2011) offer a similar view of motivational conflict, especially with respect to GAD and MDD, by drawing from regulatory focus theory (RFT; Higgins, 1997), a normative model of promotion (i.e., reward) and prevention (i.e., security) motivations, in which these two systems are conceptualized as separate and mutually inhibitory of one another. In accounting for GAD, especially when it co-occurs with MDD, Klenk et al. (2011) postulate primary failure in the prevention system (i.e., hyperactivation) that in turn can lead to failure (e.g., hypoactivation) in the promotion system. One possibility is that salience in one or both of these motivational systems may increase levels of subjective intensity and corresponding distress. Although more rigorous experimental and biobehavioral research is needed, preliminary findings support a role for both motivational dysfunction (i.e., Campbell-Sills, Liverant, & Brown, 2004) and subjective intensity (i.e., Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M., 2005; Mennin, Holaway, Fresco, Moore, & Heimberg, 2007) in the distress disorders.

Regulatory Dysfunction

Rather than processing emotion information and utilizing its motivational value, individuals with distress disorders often fail to enhance or diminish emotional experiences in a manner appropriate to a particular environmental context. Emotion regulation deficits commonly occur in GAD (Etkin, Prater, Hoeft, Menon, & Schatzberg, 2010; Etkin & Schatzberg, 2011; Mennin et al., 2005; Mennin et al., 2007) and

MDD (Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007). Rottenberg and Gross (2003) recommend that investigators need to parse emotion-generative processes from regulation efforts and to recognize that, similar to conceptualizations of healthy regulation, dysregulation occurs dynamically throughout different points in the emotion-generative process. Individuals with distress disorders demonstrate deficits earlier in less elaborative, attentional regulation, as well as later in more verbally elaborative, regulation in response to motivationally salient stimuli.

At a less elaborative level, these individuals are characterized by attentional rigidity in processing both interoceptive and exteroceptive emotional stimuli (Clasen, Wells, Ellis, & Beevers, 2013; Mogg & Bradley, 2005). For example, GAD with and without MDD is characterized by a failure to regulate emotional conflict spontaneously by shifting attention in response to a motivationally salient emotional stimulus in conflict adaptation tasks (Etkin et al., 2010; Etkin & Schatzberg, 2011). Each day, we are confronted with the simultaneous occurrence of emotionally conflicting information that may perturb our goal-directed behavior, requiring us to attend to, consider, and possibly inform our actions before completing the task at hand. The ability to handle these instances efficiently is called *conflict monitoring* (Botvinick, Braver, Barch, Carter, & Cohen, 2001). This capacity represents a facet of normative emotion regulation and consistently corresponds to activation of the anterior cingulate cortex (ACC), a region associated with attentional and motivational processes. By contrast, individuals with GAD + MDD struggle to regulate emotional conflict, fail to engage the ventral ACC in ways that dampen amygdala activity, and do not show regulation-related connectivity between the ventral ACC and the amygdala (Etkin et al., 2010).

Individuals with distress disorders also struggle to implement more verbally elaborative strategies. Aldao and Mennin (2012) found that control individuals, while watching emotionally evocative films, demonstrated increased heart rate variability (HRV; an index of parasympathetic flexibility; Porges, 2001) when instructed to reappraise (i.e., employ a different cognitive vantage point regarding an emotionally provocative event; see Gross, 2002) or accept (i.e., defined as openly turning toward, allowing, and remaining in personal contact with an emotional experience; see Hayes, Strosahl, & Wilson, 2012). Participants with GAD showed a paradoxical pattern of decreased HRV when trying to implement these more elaborative strategies, suggesting a failure of efficiency of these strategies for these individuals. Furthermore, studies comparing individuals with MDD (Johnstone et al., 2007) to healthy control participants found dissociable patterns of neural activity when participants were instructed to reappraise. Whereas control participants demonstrated a negative relationship between activation in the vlPFC and the amygdala that was mediated by the vmPFC, MDD participants showed a positive association between the vmPFC and the amygdala as well as no vlPFC activation. Rather than effectively engaging adaptive strategies, there is considerable evidence that individuals with distress disorders alternatively employ perseverative strategies to compensate for a negative emotional state, chiefly by enveloping it in elaborative self-conscious processing (Borkovec et al., 2004; Nolen-Hoeksema et al., 2008). However, cognitively elaborative processing, such as repetitive thought, is depleting and requires an expenditure of resources to employ (Muraven & Baumeister, 2000). This depletion may come at the cost of not only effective emotion management and maintenance of distress but it also appears to preclude effective emotional processing and learning.

Narrow and Rigid Contextual Learning

Individuals with distress disorders often exhibit impoverished and inflexible repertoires of behavior in response to the situations that typically function to promote escape, avoidance, or inactivity as a means of attempting to manage emotional–motivational signals (e.g., Ferster, 1973). These behavioral patterns negatively impact reward learning. For example, in contrast to healthy controls, depressed individuals are less responsive to future opportunities for reward, despite a firsthand learning history for the availability of reward (Bogdan & Piz-

zagalli, 2006). Similarly, depressed individuals, when given the choice between rewards of different sizes, fail to distinguish between options yielding large versus small rewards (Forbes, Shaw, & Dahl, 2006). Bar (2009) proposes a model of optimal functioning characterized by broad and contextual associative processing of historical and environmental factors to imagine future events and outcomes accurately. Depressive rumination is one strategy common to distress disorders that narrows associative processing, and in turn decreases the likelihood of new reward-based learning and obfuscates focusing on purposeful action (Bar, 2009). In support of this assertion, Whitmer and Gotlib (2012) found that instructing depressed individuals to ruminate during a laboratory-based task reduced sensitivity to stimuli associated with reward and punishment.

With respect to threat and safety learning, adaptively attending to motivational and emotional signals can facilitate inhibitory learning. By contrast, one factor important to achieving durable inhibitory learning is the degree of stimulus generalization that an individual displays in relation to the acquisition of a conditioned stimulus (CS; Lissek, 2012). In particular, individuals prone to anxiety disorders are less successful in discriminating the properties of stimuli that share characteristics with a training CS, thereby resulting in stimulus overgeneralization and eliciting fear in response to a broader array of stimuli. Similarly, for most organisms, signals or cues in the environment of unambiguous safety from fear leads to new inhibitory learning that helps to abolish the conditioned emotional response. Individuals prone to anxiety disorders are less likely to achieve a durable and broad-based abolishment of a conditioned fear response because of deficits in detecting cues of unambiguous safety. Instead, their search for safety is often characterized by hypervigilance and overactivity, thereby resulting in an inferior and less durable acquisition of inhibitory learning (Lohr, Olatunji, & Sawchuck, 2007; Woody & Rachman, 1994). Further, resorting to worry to regulate perceived threat experiences has been shown to encourage avoidance of emotional processing (Borkovec et al., 2004; Newman & Llera, 2011).

Clinical Application of the Emotion Regulation Perspective

As we have argued elsewhere (Mennin, Ellard, Fresco, & Gross, 2013), one important step in advancing behavioral treatments for refractory conditions such as distress disorders is the delineation of core principles that underlie various efficacious therapeutic processes and corresponding mechanistic targets refined from our growing knowledge of normative behavioral, biological, and social functioning. *Change principles* can be defined as broad frameworks for guiding both what is being targeted in a given intervention recipient and characteristics of the intervention itself. *Target mechanisms* refer to the characteristics within the recipient of the intervention that are the focus of change for a given principle (Mennin et al., 2013). ERT reflects these common principles and target mechanisms by drawing therapeutic processes from (1) CBT (e.g., psychoeducation, self-monitoring, cognitive perspective taking, problem solving, relaxation and diaphragmatic breathing exercises; Borkovec et al., 2004; Dugas & Robichaud, 2007); (2) acceptance-, dialectic-, and mindfulness-based behavioral treatments (e.g., mindfulness exercises to broaden awareness of sensations, bodily responses, and emotions in the present moment; exercises to increase willingness to accept emotions, commitment to action related to personal values; Hayes et al., 2012; Linehan, 1993; Roemer & Orsillo, 2009; Segal, Williams, & Teasdale, 2002); and (3) experiential therapy (e.g., a focus on empathic attunement, the importance of agency, delineation of emotion function, engagement of experiential tasks, see Elliot, Watson, Goldman, & Greenberg, 2004). ERT also deliberately seeks to integrate findings from basic and translational affect science, while simultaneously being responsive to emerging NIMH priorities. The net effect is an integrated, mechanism-targeted CBT that strives to improve the acute and enduring treatment efficacy for individuals suffering from distress disorders such as GAD and MDD, especially when the conditions are comorbid.

ERT is organized around core principles and target mechanisms related to an affect science framework, which, specifically seeks

to (1) increase motivational awareness, (2) develop regulatory capacities, and (3) engage new contextual learning repertoires. ERT comprises 16 weekly sessions and utilizes a phasic structure that helps clients build skills in the first half of treatment that are deployed in the second half of therapy during exposure exercises. This phasic structure draws from Gross's (2002) emotion regulation model, which distinguishes between efforts to regulate emotions earlier (i.e., antecedent-focused strategies) and later (i.e., response-focused strategies) in the emotion-generative process. Specifically, clients progress through ERT first by learning skills to increase mindful awareness of emotional and motivational states; learning skills to increase adaptive emotion regulatory responses congruent with contextual demands ("counteractive" as an alternative to reactive response-focused regulation); and increasing values-informed behavioral actions that strike a balance between security and reward motivational pulls ("proactive" regulation akin to antecedent-focused regulation).

Motivational Awareness Skills Training

The first component of ERT is the cultivation of skills to increase motivational awareness (i.e., the accurate and rapid detection of cues that signal the arrival of motivational pulls). The benefit of emotional and motivational clarity is a consistent finding across numerous studies. For instance, Lanaj, Chang, and Johnson (2012) offer meta-analytic support for RFT (Higgins, 1997) in relation to workplace performance, such that a promotion (reward) focus was positively associated with task performance and innovativeness in the workplace, and negatively associated with counterproductive workplace behaviors. In contrast, a prevention (security) focus that was unrelated to task performance but positively associated with engaging in safe workplace practices was also associated with counterproductive workplace behaviors. Furthermore, Gohm and Clore (2002), in a series of studies, have consistently shown that emotional clarity, assessed via self-report, is associated with a variety of beneficial wellness characteristics and the tendency to approach life circumstances with planful, active coping.

From a treatment perspective, motivational interviewing (MI) represents a standalone or adjunctive intervention that strives to bring awareness to one's motivations, and the discrepancies between one's stated goals and actions as a means of enhancing one's willingness to change. Westra, Arkowitz, and Dozois (2009) reported that the addition of MI to traditional CBT was associated with greater clinical efficacy in GAD, especially among clients with higher pretreatment levels of pathological worry. Finally, training in mindfulness meditation may be one means of enhancing emotional awareness and clarity. For example, Farb et al. (2010) found that a sadness provocation is associated with activation of self-referencing neural circuits (e.g., medial PFC), but training in mindfulness meditation is associated with activation of additional neural regions associated with visual attention (e.g., pulvinar nucleus), as well as visceral and somatosensory areas (e.g., right insula). Greater activation of these neural regions was also negatively correlated with concurrent levels of depression.

Drawing from this empirical basis, ERT helps clients develop motivational awareness initially through psychoeducation aimed at increasing understanding of emotions and underlying motivations in the context of personally relevant historical and proximal events. Clients receive a rationale for the benefits of observing emotions with greater granularity and clarity (i.e., better differentiation of each emotion even when multiple emotions are present). For instance, clients imagine a musical orchestra in which each musical instrument represents a different emotion. The orchestral composition represents the motivational pulls in their lives. Ideally, the composition is harmonious and the music moves clients to take some action in their lives. Clients are encouraged to listen in such a way that each and every part of the orchestra can be heard and discerned for its contribution to the overall composition.

As noted by Aupperle and Paulus (2010), individuals with distress disorders struggle to resolve motivational conflicts that occur in their lives, via overrepresentation of threat valuation, inconsistent representation of approach valuation, and impaired arbitration of these motivation systems. The net result of this imbalance of motivational

pulls is that the discrepancy becomes the salient signal, and individuals with distress disorders often respond reactively in a desperate attempt to resolve the discrepancy. Thus, building on the orchestra metaphor, clients also engage in a motivational anchoring exercise that begins their training in cultivating greater motivational and emotional awareness. Specifically, clients imagine an important but difficult situation in their lives that likely is marked by pulls for approach and avoidance. As clients imagine this situation, they envision the kinds of behavioral responses they are likely to take from that particular balance of approach and avoidance motivations. ERT therapists then systematically help clients to envision this situation from various motivational stances, with the goal of helping them see that most life circumstances comprise both approach and avoidance motivations, and different responses are possible when one attends and follows to differing degrees one's emotions and motivations.

As clients gain an appreciation and understanding for how emotions and motivations can effectively inform their lives, they are introduced to a metaphor designed to help them improve the accuracy and rapidity of detecting cues in their lives. This work is predicated on the well-validated behavioral interventions of self-monitoring (e.g., Linehan, 1993) and functional analysis (e.g., Ferster, 1973), in which clients are taught to increase their self-awareness of life situations as a means of better identifying antecedents (triggers), thoughts, feelings, and behaviors that are prompted by these antecedents, and the consequences of these behavioral actions. Self-monitoring helps individuals identify patterns of responding reactively, often with little awareness of the emotional and motivational pulls as a first step in bringing more conscious, deliberate, and ideally effective action when certain cues arise. In ERT, the therapist begins self-monitoring and cue detection by asking clients to imagine a pristine snowball (i.e., a pure emotional experience) rolling down a hill, and in the course of its travel, picking up dirt and twigs, and becoming hard and icy (i.e., the unfolding of emotional experience, particularly in the aftermath of failures in nonelaborative and elaborative emotion capacities). Essentially, the snowball metaphor informs clients about the temporal model of emotion regulation and how less effort is required when emotions are handled closer to their arrival in the temporal unfolding.

Drawing from this snowball metaphor, ERT clients are then encouraged to practice a form of self-monitoring called Catch Yourself Reacting (CYR). This practice begins as a simple exercise to help identify triggers of clients' emotions, the actual emotions themselves (fear, anxiety, disgust, etc.), and the intensity of each emotion they listed. Later in ERT, the CYR is also utilized to practice the emotion regulation skills clients will learn in the course of treatment (see below). CYRs are completed at home, then reviewed in the subsequent session with the therapist. When clients experience difficulties with CYR, therapists may choose to conduct a "Do-Over." This activity resembles both the cognitive rehearsal task, derived from traditional cognitive therapy of depression (Beck, Rush, Shaw, & Emery, 1979), and evocative unfolding in experiential therapy (Elliott et al., 2004). The CYR "Do-Over" essentially represents a therapist-supervised opportunity to shape and solidify self-monitoring and cue detection.

Regulatory Skills Training

A principle of regulatory skills training assumes that individuals with psychopathology do not have immutable regulation deficits; rather, with appropriate training, these capacities can be grown and utilized in their lives. Based on Gross's model and the extant clinical and experimental literature, ERT targets different regulatory capacities by utilizing a number of CBT intervention processes that vary in their entry points along the trajectory of unfolding emotional experience. Specifically, we promote four increasingly elaborative (i.e., that use working memory/verbal representations and are resource intensive; Badre & D'Esposito, 2007) regulatory capacities that rely on antecedent-focused processing: *attending* (i.e., the ability to focus, sustain, and flexibly move attention); *allowance* (i.e., the ability to openly turn toward, allow, and remain in personal contact with an emotional experience); *distancing* (i.e., the ability to identify, observe, and generate psychological perspective from inner experiences); and *reframing*

(i.e., the ability to change one's evaluation of an event such that the event is altered in its emotional significance).

Attending

A capacity for directed attention implies flexible responsivity to different contexts. Whereas constricted, narrowly focused attention is adaptive during times of potential threat, broadened attention facilitates exploratory behavior, thereby allowing for the possibility of increased detection of new information and novel incentives (Friedman & Forster, 2010). One facet of directed attention is *focused attention*, which involves actively choosing the stimulus to which one will attend (Kabat-Zinn, 1990). Similarly, *sustained attention* refers to maintaining this focus on the target stimulus (see Posner & Rothbart, 1992), as well as actively redirecting attention back to the target stimulus when attention has wandered (e.g., Smallwood & Schooler, 2006). Finally, flexible attention entails deliberately attending to various aspects of an experience (Kabat-Zinn, 1990). Converging lines of research suggest that reduced attentional flexibility and rigid attentional biases distinguish individuals with distress disorders from healthy controls (Clasen et al., 2013; Mogg & Bradley, 2005).

ERT targets directed attention by promoting skills to encourage greater awareness of emotions, including sensations, bodily responses, and subjective experience. Mindful attention training is utilized to increase a healthy awareness of motivations and emotional responding, with a particular emphasis on less elaborative, less linguistic processing of one's experience (Kabat-Zinn, 1990). Both in-session and daily practices are derived from mindfulness-based stress reduction (MBSR; Kabat-Zinn, 1990) and mindfulness-based cognitive therapy (MBCT; Segal et al., 2002). ERT promotes directed attention toward external stimuli by increasing mindfulness of senses. A broadened awareness of appetitive and aversive stimuli alike is encouraged through practices that increase mindful ingestion of senses (e.g., eating a raisin mindfully; Kabat-Zinn, 1990). ERT also promotes directed attention to internal, nonverbal stimuli or stimuli that can be felt in the body (e.g., Craig, 2009), thereby enhancing the ability to disengage from elaborative processes about sensory experiences. Clients are taught diaphragmatic breathing as a brief, nonelaborative practice that focuses attention on sensations in the body (e.g., Roemer & Orsillo, 2009). Clients also learn a practice adapted from the body scan work common in MBSR (Kabat-Zinn, 1990) and MBCT (Segal et al., 2002), coupled with an awareness-based progressive muscle relaxation approach that has been effectively utilized in the treatment of GAD (Roemer & Orsillo, 2009). After experiencing some success with directed attention practices, clients learn an "on the spot" skill of detecting tension and implementing relaxation techniques to promote greater flexibility in musculature response.

Finally, although not every emotion experienced is necessarily adaptive, broadening awareness to the full spectrum of emotional experience can help clients move through "cloudy" (i.e., secondary emotional reactions) toward "clear" (i.e., primary emotions that convey initial action tendencies and their associated meanings for behavior) emotions that better reflect the array of motivational cues that may be present (Elliot et al., 2004; Linehan, 1993). Specifically, clients learn a mindfulness of emotions exercise in which the goal is to bring to mind a situation that possesses conflicting emotions and motivations (i.e., simultaneous security and reward impetuses), and they are invited to sit with the experience until they can hold and more clearly delineate the emotions and motivational pulls in the situation.

Allowing

Emotional allowance involves maintaining contact with feeling states, without being dissuaded by elaborative thought processes, such as judgments about the experience (see Hayes et al., 2012). Individuals with highly developed capacities to accept their emotions are less likely to avoid certain endeavors wherein difficult emotions could arise and are more likely to stay in contact with emotions associated with aversiveness, increasing the possibility of updating contingencies related to their valuation (Hayes et al., 2012). Individuals with psychopathol-

ogy demonstrate difficulties acknowledging their emotional experiences, which includes being dissuaded by negative beliefs about difficult emotions, aversion toward difficult emotional experiences, engaging in maladaptive elaborative responses when difficult emotions arise (e.g., worrying, brooding, self-criticizing), attempting to reduce their awareness and limit their experience of difficult emotions, and avoiding situations and activities that could provoke difficult emotions, even when such activities are important to them (Barlow et al., 2011; Hayes et al., 2012; Linehan, 1993; Roemer & Orsillo, 2009; Segal et al., 2002).

By accepting and exploring emotions, clients gain an ability to be present with emotions and learn how to determine their functional utility in guiding actions. Specifically, clients conduct an "offline" practice that involves imaginally engaging and remaining in contact with sensations, emotions, and cognitions that arise in pursuit of a difficult to achieve but motivationally enhancing action—in effect, a personally salient approach–avoidance conflict. The on-the-spot version of this practice involves encouraging clients to "pause" by sustaining their connection to a cloudy, diffuse situation until they are able to connect to the primary emotions that reflect both motivations for reward and safety. Another borrowed, on-the-spot practice, designed to sustain or regain allowance is the 3-minute breathing space (Segal et al., 2002), which helps clients anchor themselves in an event with strong pulls to respond reactively.

Distancing

Various theorists (e.g., Hayes et al., 2012; Kross & Ayduk, 2009; Segal et al., 2002) have emphasized the importance of *distancing* or *decentering*, the metacognitive ability to observe items that arise in the mind (thoughts, feelings, memories, etc.) with healthy psychological distance, greater self-awareness, and perspective taking. Cognitive distancing helps individuals disengage from an intense emotion, its corresponding motivational impetus, and associated maladaptive self-referential processing, in favor of adopting a more experiential perspective. This ability also involves recognizing that one's thoughts, feelings, and urges are transient internal events rather than inherent, permanent aspects of the self or accurate representations of reality (Fresco et al., 2007; Segal et al., 2002). Studies reveal psychological benefits from promoting distance from the self in time (e.g., viewing inner experiences as temporary; Watkins, Teasdale, & Williams, 2000) and in space (e.g., viewing inner experiences as physical objects that are separate from oneself; Kalisch et al., 2005). Distress disorders have been associated with deficits in cognitive distancing (e.g., Fresco al., 2007) and can be reduced by experimental techniques that promote an observational distance from the self (e.g., Kross & Ayduk, 2009).

Distancing is targeted through two offline mindfulness practices and their corresponding on-the-spot versions. In the *mountain meditation*, derived from MBSR (Kabat-Zinn, 1994), clients are invited to internalize a living, breathing mountain to provide solidarity and permanence in their lives to help them "weather" the transient emotional upheavals in their lives. The mountain meditation provides a decentered perspective through the lens of time—helping clients tell themselves "This too will pass." Clients then practice a brief version of the mountain meditation designed to be utilized on the spot in moments that call for a security-first response to help sustain or regain a decentered stance. Similarly, drawing primarily from acceptance and commitment therapy (ACT; Hayes et al., 2012), ERT promotes a focus on mental spatial distance and is designed to teach clients to bring situations to mind, then to granulize the constituent parts of the situation by placing them externally on objects in the room. This offline practice is an invitation to create healthy distance in one's mind's eye so that these products of mind are more readily observable, and in turn, can inform our deliberate actions from a decentered perspective. This practice also has a corresponding on-the-spot skill, where clients imaginally place products of their mind on objects that they ordinarily carry in their daily lives. One of our clients actually carried around a shoehorn he crafted, which he used to "pry" himself away from products of his mind to promote better observation and nonjudgment.

Reframing

Reframing refers to the ability to change one's evaluation of an event so as to alter its emotional significance (Gross, 2002). Three of the most common methods include *realistic reappraisal* (e.g., reevaluating an event in a more accurate, objective, factual manner, while remaining sensitive to contextual factors; Ray, Wilhelm, & Gross, 2008), *positive reappraisal* (e.g., reevaluating an event in a manner that emphasizes possible desired, rewarding, or beneficial aspects of the event or consequences of the event that may have been overlooked in the original appraisal; Ray et al., 2008), and *compassionate reappraisal* (e.g., reevaluating an event in a manner that appreciates and validates the presence of emotional pain and desires to alleviate it, and identifies the pain as a natural aspect of the human experience; Leary, Tate, Adams, Batts, & Hancock, 2007). Researchers who have manipulated changing cognitions in the laboratory have found psychological benefits from instructing participants before they encounter a stimulus or task to reappraise it in a manner designed to promote personal detachment or objectivity (e.g., Gross, 2002). A capacity to change cognitions and relinquish one's original interpretation may undermine passive, repetitive elaborative processes central to transdiagnostic psychopathology (e.g., rumination; Nolen Hoeksema et al., 2008) and promote flexibility that allows more effective, rather than habitual, behavioral reactions. Indeed, reappraisal has been found to be positively associated with a number of indices of well-being, including effective interpersonal functioning (Gross, 2002).

ERT employs several strategies to develop cognitive change capacities to provide temporary relief from emotional distress, facilitate a restoration of emotional clarity, and promote effective action. Once clients have obtained a decentered perspective from their emotional and motivational pulls, they are encouraged to adopt a courageous perspective wherein they can address any security-driven responses and provide alternative statements that reflect their strength in the face of uncertainty. Clients are also encouraged to adopt a self-compassionate reappraisal stance wherein they can imagine telling a very caring, interested, compassionate individual about their difficult thoughts and feelings, and reminding themselves of their strengths and coping ability (Gilbert, 2009; Segal et al., 2002). Noticing one's self-critical thoughts is encouraged and "softening" them when they arise is accomplished by invoking alternative, self-validating statements. These courageous and compassionate statements are typically written down and carried with the patient in a pocket or put on a smartphone as a recurrent reminder. After committing these statements "to heart," clients are then encouraged simply to tap their pocket or bag containing the written statement or smartphone to regain quickly this sense of perspective.

Experiential Exposure

A hallmark goal of CBT is to assist clients in "new learning" to promote the imagining or enacting of novel experiences that counteract old and well-worn patterns of maladaptive associations and reinforcement. With respect to anxiety disorders, exposure therapy, derived from principles of classical conditioning, has demonstrated considerable acute and enduring treatment efficacy (Craske et al., 2008). Exposure therapy is often coupled with treatment components that reduce the reliance on escape and avoidance behaviors while cultivating repertoires of behaviors designed to pursue and obtain rewards (Craske et al., 2008; Ferster, 1973). Additionally, informed by important basic findings about the nature of classical extinction and inhibitory learning (e.g., Bouton, Mineka, & Barlow, 2001) implementations of exposure therapy have moved beyond sustained fear reduction and habituation accounts of extinction to promote superior inhibitory learning and extinction retrieval (e.g., Craske, & Vervliet, 2013). Similarly, behavioral accounts of depression depict depressed individuals as no longer able to engage the behavioral repertoires capable of delivering positive reinforcement (Lewinsohn, 1974) while they inordinately orient their lives in service of escape and avoidance of aversive situations (Ferster, 1973; Kanter et al., 2010). To overcome these deficits, treatment typically consists of interventions that include activity scheduling, contingency management, and skills training

as ways to help individuals gain awareness of the availability of positive reinforcement, engage in behaviors capable of obtaining the positive reinforcement contingently, and ideally, effecting change in the environment so that access to positive reinforcers remain available when certain behavioral responses are provided (Ferster, 1973; Kanter et al., 2010; Lewinsohn, 1974). In addition, recent innovative treatments for depression have benefited from basic and translational findings (e.g., Bouton et al., 2001) about developing exposure-based treatments for depression that deliberately provoke and activate historical negative content, so that this material can be explored alongside information that is dissonant and serve to facilitate broad-based change in maladaptive cognitive–affective–behavioral–somatic patterns (e.g., Hayes et al., 2007).

Exposure therapy is most effective when the fear stimulus is focal and difficult to avoid (e.g., specific phobia). Traditional exposure therapy may be less efficacious in distress disorders. Whereas individuals with uncomplicated presentations of anxiety have relatively more discrete and circumscribed fears, individuals with distress disorders have difficulties confronting emotional arousal irrespective of what external cue provokes it. That is, conducting exposure therapy in response to external fear cues does not effectively target the source of the difficulty for individuals with distress disorders. Consistent with an emotion regulation framework, some approaches promote exposure to emotional experiences themselves to increase acceptance of these experiences and diminish the need to utilize strategies such as worry or rumination in an attempt to escape aversively perceived emotional states (e.g., Barlow et al., 2011). However, this approach may not fully address dysfunction given that distress-disordered individuals commonly resort to cognitively elaborative responses such as worry and rumination to help them escape emotional processing and to preclude new inhibitory learning. Furthermore, a focus on negative emotional exposures may do less to expose individuals to core idiographic and schematic themes that certain emotions may convey. Also, this approach does not necessarily engage the reward system by directly encouraging proactive behavior toward desired outcomes. In other words, exposure in distress disorders may be most fruitful when it involves contexts that have recurrent themes such as threat (i.e., security motivation system) and encourage activation (i.e., reward motivation system).

In support of this contention, recent work by Newman and Llera (2011) demonstrates that worry is reinforced by creating a fixed, invariable, and predictive defensive emotional state that inevitably precludes emotional processing. For individuals with GAD, they argue, what they fear is a stark emotional contrast, such as moving from a positive state to a negative state, which may disincline these individuals to engage a positive state if it means increased possibility for a negative state. This conceptualization is consistent with the emotion regulation model in which individuals with distress disorders resort to worry and rumination in response to security motivations, which overshadow reward motivations. Furthermore, it is consistent with behavioral accounts of MDD that view depressed individuals as withdrawing to protect themselves from the aversion of loss, thus gaining no new rewards to help them counteract their depression. Taken as a whole, these perspectives suggest that exposure to rewarding contexts with the possibility of high risk may be most ameliorative for these individuals, since they simultaneously engage both the security and reward systems.

In ERT, exposure to threat–reward contrasts are accomplished by focusing clients' attention on their personal values (i.e., their highest priorities and most cherished principles; Hayes et al., 2012; Wilson & Murrell, 2004). Values-based exposure involves turning a problem on its head: Rather than being exposed to feared outcomes, one is exposed to the way he or she would like to be living, and the expected arrival of perceived fears, disappointments, and judgments are treated as obstacles to being able to live a valued life (Hayes et al., 2012). ERT expands values-based exposure commonly utilized in ACT to address not only "top-down" decisions about life goals but also "bottom–up" influences of security and reward motivational pulls, as well as their interaction. During an experiential exposure phase of ERT, clients explore acting in accordance with their values and confronting any accompanying per-

ceived obstacles that arise both within and between sessions. Specifically, new learning is targeted with three main exposure interventions to promote valued living: (1) imaginal exercises related to values-informed goals; (2) experiential dialogue exercises to explore perceived internal motivational conflicts that impede engaging in valued actions (Elliot et al., 2004); and (3) planned between-session exercises wherein clients engage valued actions outside of session. Clients also utilize regulatory capacity skills to help in engagement of in-session experiential tasks and to facilitate valued action outside of session.

Proactive Valued Action

In ERT, therapists and clients collaborate to identify cherished values in life domains (e.g., family, friends, relationships, work, personal care), with clients reporting discrepancies between the importance they place on this value and how consistently they have been living accordingly (Wilson & Murrell, 2004). Therapists then encourage clients to think about a salient value with a large discrepancy and how they want their actions to reflect this value *today*, even if it involves only a small action step. Specifically, imaginal exposure tasks that focus on engaging in *specific* valued actions are conducted to (1) provide clients with an experientially rich rehearsal of the steps that might be necessary to live by their values and (2) confront the emotional challenges that are likely to come up as clients imagine engagement of valued action. In this imagery exposure task, therapists help clients imagine each step involved in engaging this action, while noting changes in motivational levels and encouraging utilization of skills to address difficulties in awareness and balancing of emotional responses. Utilizing imagery to consolidate skills and promote functional action is also congruent with traditional interventions such as *cognitive rehearsal* (Beck et al., 1979).

Exploring Conflict Themes in Obstacles to Valued Living

The second component of exposure work in ERT involves addressing perceived obstacles to taking valued action. Obstacles are addressed through the lens of "conflict themes" and include primarily (1) a *motivational conflict* (e.g., security motivations are blocking or interrupting efforts that engage the reward system) and (2) *self-critical reactive responses to emotions* (i.e., judgmental negative beliefs about one's emotional responses and associated motivations are interrupting self-acceptance and engagement of reward). These conflict themes are addressed within the session using an experiential dialogue exercise derived from emotion-focused therapy (Elliot et al., 2004). The motivational conflict is most central to interrupting valued action and is addressed by encouraging clients to engage in a dialogue between the part of themselves strongly motivated to obtain security, and the part motivated toward self-actualization, to arrive at a more unified motivational stance that is conducive to valued action. Resolution comes when expressions from both sides of the dialogue demonstrate acknowledgment of the needs of the other and an agreement to commit to the valued action while allowing a place for a softened obstacle voice to be present without total control. The purpose of these tasks is to reduce negative emotional responses that are activated when obstacles reflecting these conflicts are perceived (i.e., exposure), to generate a new perspective (i.e., new meaning) on these obstacles, and to engage more adaptive emotions that are facilitative of valued action engagement.

Engaging Proactive Valued Action Outside of Session

Valued action is also promoted between sessions by building upon the in-session valued action exploration and obstacles confrontation exposure tasks. Therapists encourage clients to engage both planned (i.e., specific valued actions related to salient values explored in session and to which clients commit in the presence of therapists) and spontaneous (i.e., any other valued actions in which clients notice themselves engaging) valued actions outside of session (Hayes et al., 2012). Furthermore, clients are encouraged to utilize skills both proactively (in an antecedent-focused manner) when they are planning to engage valued actions and counteractively (in a response-focused manner) when they notice themselves getting

unexpectedly distressed and feel pulled to respond reactively with worry, reassurance seeking, self-criticism, or behavioral avoidance. Finally, external barriers (i.e., obstacles in the environment that are outside the client's control) that might have been deferred during exposure tasks can also be addressed more actively in between-session exercises. Therapists can help clients problem-solve these obstacles or utilize skills such as acceptance to further facilitate valued action.

Research Findings

To date, we have tested the efficacy and purported mechanisms of ERT in a number of trials at various sites. These findings have been presented in greater detail at a number of recent conference sessions (e.g., Mennin & Fresco, 2011; Mennin, Fresco, & Aldao, 2012) and are the focus of submitted manuscripts currently under review.

Preliminary Outcome Results

To date, ERT's efficacy has been demonstrated in a recently concluded NIMH-funded project that comprises an open trial (N = 19) and a randomized control trial (RCT; N = 60). In both trials, clients tolerated ERT, as evidenced by high ratings of personal satisfaction and low rates of attrition in the course of treatment. For instance, 18 of 19 open-trial clients and 26 of 30 RCT clients completed treatment. In terms of clinical outcomes, open-trial clients evidenced reductions in both clinician-assessed and self-report measures of GAD severity, worry, and trait-anxious and depression symptoms, and corresponding improvements in quality of life, with within-subjects effect sizes well exceeding conventions for large effects (Cohen's d's = 1.5 to 4.5). These gains were maintained for 9 months following treatment.

The RCT study compared ERT to a modified attention control (MAC) condition, which involved clients periodically speaking via telephone to a clinician who provided supportive listening. Clients received assessments at baseline, midtreatment, and postacute treatment. MAC clients were offered ERT in an open-label fashion following the immediate RCT period. RCT findings revealed that ERT clients, compared to MAC clients, evidenced significantly greater reductions in GAD severity, worry, trait anxiousness, and depression symptoms, and corresponding improvements in functionality and quality of life, with between-subjects effect sizes in the medium to large range (d = 0.50 to 2.0). These gains were maintained for 9 months following the end of treatment. In addition, these effect size estimates also take into account clients who dropped out of treatment.

Finally, a sizable subgroup of clients with GAD and comorbid MDD (N = 30) were enrolled and treated. Within- subjects effect sizes in both clinician-assessed and self-report measures of GAD severity, worry, trait anxiousness, and depression symptoms, and corresponding improvements in functionality and quality of life, were comparable to overall trial findings—thereby suggesting that MDD comorbidity did not interfere with treatment efficacy (Cohen's d's = 1.5 to 4.0). Furthermore, rumination and anhedonia also decreased (Cohen's d's = 1.5 to 2.0).

Preliminary Mechanisms Results

We argue that efforts to demonstrate the mechanisms by which one's treatment produces clinical improvement are best tied to *common* target mechanisms such as those that reflect the capacities described earlier (e.g., directed attention, emotional acceptance, cognitive distancing, cognitive change). Furthermore, target mechanism change should be demonstrated with biobehavioral marker assessments (e.g., behavioral tasks, functional magnetic resonance imaging [fMRI], psychophysiology) that have established reliability and validity in laboratory and analogue studies. This approach is aligned with the growing multidisciplinary field of intervention science and with NIMH priorities (e.g., RDoC; Craske, 2012), which seek to elucidate biobehavioral markers that are reliably dissociable in patient subgroups compared to healthy controls. Also, assessing common target mechanisms in various approaches may help us better understand patient characteristics that predict treatment success and failure (e.g., treatment matching, treatment optimization/augmentation; NIMH Council's treatment personalization initiative; Kraemer, Wilson, Fairburn, & Agras, 2002).

Commensurate with this viewpoint, ERT has been designed to target mechanisms of affective dysfunction that we have argued are central to refractory distress disorders. Thus, we have been interested in examining whether these treatment outcomes are the result of changes in purported motivational, regulatory, and contextual learning mechanisms. One promising preliminary finding is related to emotional conflict adaptation (Etkin & Schatzberg, 2011). A subset of our ERT clients (N = 15) completed the Etkin emotional conflict task (described earlier) at pretreatment and at the midpoint of ERT. Findings indicate that by midtreatment, clients improved in ability to shift their attention in the face of emotional conflict (pre- to midtreatment d = 0.74) to levels comparable to healthy controls (Etkin et al., 2010). Furthermore, clients who showed the greatest gains in conflict adaptation by midtreatment, showed the greatest pre- to posttreatment response in anxiety, anhedonic depression, and worry. These preliminary data are supportive of our hypotheses that ERT may, in part, exert its therapeutic impact through normalization of less elaborative emotion regulatory mechanisms such as ability to shift attention by adapting to conflict.

Although we have not yet tested changes in more elaborative regulatory mechanisms, we have examined responses to more complex emotional stimuli, which likely require more effort and elaboration to manage. Specifically, we assessed HRV during a fearful film paradigm at pre- and midtreatment. Eighteen clients were assessed during the neutral film, the fearful film, and, a recovery period. At pretreatment, these clients displayed a flattened response throughout the experimental period—suggesting reduced flexibility. At midtreatment, clients displayed a quadratic pattern of vagal withdrawal (i.e., reactivity) and vagal rebound ($d_{\text{pre to mid-tx}}$ = 0.81) reflecting a more normative response to these changing emotional contexts. Period-averaged HRV levels at midtreatment also increased to within 1 SD of levels in a healthy control sample. Furthermore, clients who showed the greatest increases in parasympathetic flexibility from pre- to midtreatment showed the greatest pre to posttreatment gains in diagnostic severity, anxiety, worry, anhedonic depression, impairment, and life quality. These data suggest that ERT normalizes emotional response patterns, and that this normalization plays a role in the therapeutic effects of ERT. However, it is unclear how clients were able to improve their response to the film, because spontaneous utilization of ERT-related or other strategies was not assessed. It will be important in future research to determine whether these improvements are indeed due to implementation of the skills learned in ERT.

Conclusions and Future Directions

Individuals with distress disorders (i.e., GAD, MDD) are commonly comorbid and appear to be characterized by higher order negative emotional factors (e.g., Krueger & Markon, 2006; Watson, 2005) that reflect activation of underlying motivational systems related to threat–safety and reward–loss (Campbell-Sills et al., 2004; Klenk et al., 2011; Woody & Rachman, 1994). Furthermore, they tend to perseverate (i.e., worry, ruminate) as a way to manage this motivationally relevant distress (Borkovec et al., 2004; Nolen-Hoeksema et al., 2008) and often utilize these self-conscious processes to the detriment of engaging new learning repertoires. ERT (Mennin & Fresco, 2009) integrates principles from traditional and contemporary CBT (e.g., skills training and exposure) with basic and translational findings from affect science to offer a blueprint for improving intervention by focusing on the motivational responses and corresponding regulatory characteristics of individuals with distress disorders. This emphasis on affect science permits identification of mechanisms of treatment in terms of core disruptions of normative cognitive, emotional, and motivational systems, which in turn helps generate more targeted solutions to help clients utilize adaptive ways to cope or compensate for these core deficits. In essence, contrasting a client's difficulties with what we understand as normative functioning allows us to generate theory-driven hypotheses that form that basis of our case conceptualization and treatment planning (e.g., Craske, 2012).

A summary of the conceptual model of ERT as presented in this chapter appears in Figure 28.1. However, ERT continues to be developed and tested. For up-to-date infor-

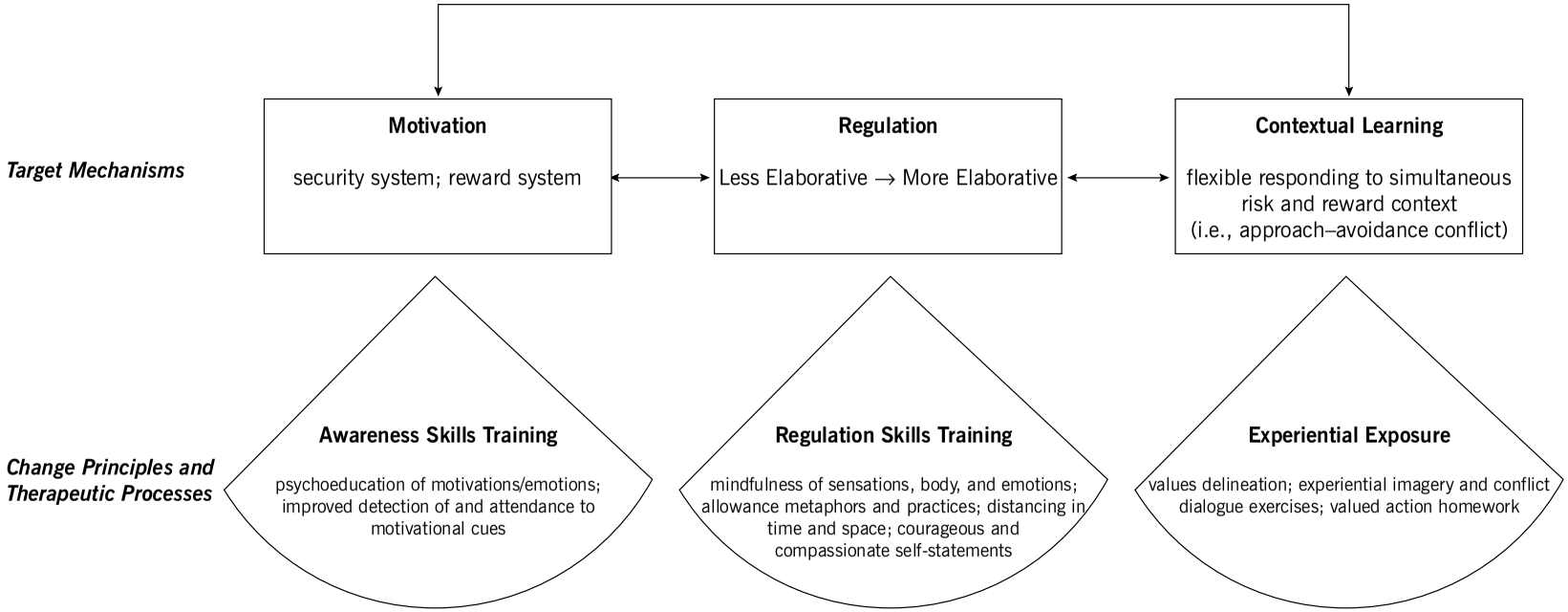

FIGURE 28.1. Conceptual model of target mechanisms, change principles, and therapeutic processes in emotion regulation therapy.

mation about ERT, please visit *www.emotionregulationtherapy.com*. Our emotion regulation model is based on current findings in affect science and the psychopathology of distress disorders. However, many of the central tenets of our model await further careful experimental inquiry using multimethod biobehavioral approaches. Nonetheless, preliminary data are supportive of our hypotheses that ERT may exert its therapeutic impact in part through normalization of emotional processes, and therefore be most effective for individuals with distress disorders in whom this system is most impaired. Building on these pilot findings on biobehavioral mechanisms of ERT, we are currently examining neural changes related to ERT utilizing fMRI procedures while administering paradigms examining motivational engagement and regulatory responses at less and more elaborate levels. Furthermore, we are currently developing a portable, computer-based "emotion regulation training" to target mechanisms of ERT in a briefer, more rapid, and cost-effective method of intervention, which may be particularly useful for certain populations and geographical locations in which standard treatment efforts are less feasible (Kazdin & Blase, 2011). Through this work, we are eager to continue testing motivational and regulatory mechanisms in the distress disorders and to demonstrate their role in targeted treatments such as ERT.

References

Aldao, A., & Mennin, D. S. (2012). Paradoxical cardiovascular effects of implementing adaptive emotion regulation strategies in generalized anxiety disorder. *Behaviour Research and Therapy, 50*, 122–130.

Aupperle, R. L., & Paulus, M. P. (2010). Neural systems underlying approach and avoidance in anxiety disorders. *Dialogues in Clinical Neuroscience, 12*, 517–531.

Badre, D., & D'Esposito, M. (2007). Functional magnetic resonance imaging evidence for a hierarchical organization of the prefrontal cortex. *Journal of Cognitive Neuroscience, 19*, 2082–2099.

Bar, M. (2009). A cognitive neuroscience hypothesis of mood and depression. *Trends in Cognitive Sciences, 13*, 456–463.

Barlow, D. H. (2002). *Anxiety and its disorders* (2nd ed.). New York: Guilford Press.

Barlow, D. H., Farchione, T. J., Fairholme, C. P., Ellard, K. K., Boisseau, C. L., Allen, L. B., et al. (2011). *The unified protocol for transdiagnostic treatment of emotional disorders: Therapist guide*. New York: Oxford University Press.

Barlow, D. H., Gorman, J. M., Shear, M. K., & Woods, S. W. (2000). Cognitive-behavioral therapy, imipramine, or their combination for panic disorder. *Journal of the American Medical Association, 283*, 2529–2536.

Beck, A. T., Rush, J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression: A treatment manual*. New York: Guilford Press.

Berridge, K. C., Robinson, T. E., & Aldridge, J. W. (2009). Dissecting components of reward: "Liking," "wanting," and learning. *Current Opinion in Pharmacology, 9*(1), 65–73.

Bogdan, R., & Pizzagalli, D. A. (2006). Acute stress reduces reward responsiveness: Implications for depression. *Biological Psychiatry, 60*, 1147–1154.

Borkovec, T. D., Alcaine, O., & Behar, E. (2004). Avoidance theory of worry and generalized anxiety disorder. In R. G. Heimberg, C. L. Turk, & D. S. Mennin (Eds.), *Generalized anxiety disorder: Advances in research and practice* (pp. 77–108). New York: Guilford Press.

Borkovec, T. D., & Ruscio, A. M. (2001). Psychotherapy for generalized anxiety disorder. *Journal of Clinical Psychiatry, 62*(Suppl. 11), 37–42.

Botvinick, M., Braver, T., Barch, D., Carter, C., & Cohen, J. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*, 624–652.

Bouton, M. E., Mineka, S., & Barlow, D. H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review, 108*, 4–32.

Brown, T. A. (2007). Temporal course and structural relationships among dimensions of temperament and DSM-IV anxiety and mood disorder constructs. *Journal of Abnormal Psychology, 116*(2), 313–328.

Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioral therapy: A review of meta-analyses. *Clinical Psychology Review, 26*, 17–31.

Campbell-Sills, L., Liverant, G. I., & Brown, T. A. (2004). Psychometric evaluation of the

behavioral inhibition/behavioral activation scales in a large sample of outpatients with anxiety and mood disorders. *Psychological Assessment, 16*, 244–254.

Carver, C. S., Avivi, Y. E., & Laurenceau, J.-P. (2008). Approach, avoidance, and emotional experiences. In A. Elliot (Ed.), *Handbook of approach and avoidance motivation* (pp. 385–397). New York: Psychology Press.

Clasen, P. C., Wells, T. T., Ellis, A. J., & Beevers, C. G. (2013). Attentional biases and the persistence of sad mood in major depressive disorder. *Journal of Abnormal Psychology, 122*(1), 74–85.

Craig, A. D. (2009). How do you feel—now?: The anterior insula and human awareness. *Nature Reviews Neuroscience, 10*, 59–70.

Craske, M. G. (2012). THE R-DOC Initiative: Science and practice. *Depression and Anxiety, 29*, 253–256.

Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy, 46*, 5–27.

Craske, M. G., & Vervliet, B. (2013). Extinction learning and its retrieval. In D. Hermans, B. Rimé, & B. Mesquita (Eds.), *Changing emotions* (pp. 53–59). Hove, UK: Psychology Press.

Cuijpers, P., van Straten, A., Andersson, G., & van Oppen, P. (2008). Psychotherapy for depression in adults: A meta-analysis of comparative outcome studies. *Journal of Consulting and Clinical Psychology, 76*, 909–922.

Delgado, M. R., Gillis, M. M., & Phelps, E. A. (2008). Regulating the expectation of reward via cognitive strategies. *Nature Neuroscience, 11*, 880–881.

Dobzhansky, T. (1970). *Genetics of the evolutionary process*. New York: Columbia University Press.

Dollard, J., & Miller, N. E. (1950). *Personality and psychotherapy*. New York: McGraw-Hill.

Dugas, M. J., & Robichaud, M. (2007). *Cognitive-behavioral treatment for generalized anxiety disorder: From science to practice*. New York: Routledge.

Elliott, R., Watson, J. C., Goldman, R., & Greenberg, L. (2004). *Learning emotion-focused therapy: The process–experiential approach to change*. Washington, DC: American Psychological Association.

Etkin, A., Prater, K. E., Hoeft, F., Menon, V., & Schatzberg, A. F. (2010). Failure of anterior cingulate activation and connectivity with the amygdala during implicit regulation of emotional processing in generalized anxiety disorder. *American Journal of Psychiatry, 167*, 545–554.

Etkin, A., & Schatzberg, A. F. (2011). Common abnormalities and disorder-specific compensation during implicit regulation of emotional processing in generalized anxiety and major depressive disorders. *American Journal of Psychiatry, 168*, 968–978.

Farabaugh, A. H., Bitran, S., Witte, J., Alpert, J., Chuzi, S., Clain, A. J., Baer, L., et al. (2010). Anxious depression and early changes in the HAMD-17 anxiety-somatization factor items and antidepressant treatment outcome. *International Clinical Psychopharmacology, 25*, 214–217.

Farb, N. A. S., Anderson, A. K., Mayberg, H., Bean, J., McKeon, D., & Segal, Z. V. (2010). Minding one's emotions: Mindfulness training alters the neural expression of sadness. *Emotion, 10*, 25–33.

Ferster, C. B. (1973). A functional analysis of depression. *American Psychologist, 28*, 857–870.

Forbes, E. E., Shaw, D. S., & Dahl, R. E. (2006). Alterations in reward-related decisions in boys with depressive and anxiety disorders. *Biological Psychiatry, 59*, 126S–126S.

Fresco, D. M., Moore, M., van Dulmen, M., Segal, Z., Ma, S., Teasdale, J., et al. (2007). Initial psychometric properties of the Experiences Questionnaire: Validation of a self-report measure of decentering. *Behavior Therapy, 38*, 234–246.

Friedman, R. S., & Forster, J. (2010). Implicit affective cues and attentional tuning: An integrative review. *Psychological Bulletin, 136*, 875–893.

Gilbert, P. (2009). *The compassionate mind: A new approach to life's challenges*. Oakland, CA: New Harbinger.

Gohm, C. L., & Clore, G. L. (2002). Four latent traits of emotional experience and their involvement in well-being, coping, and attributional style. *Cognition and Emotion, 16*, 495–518.

Gray, J. A., & McNaughton, N. (2000). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system* (2nd ed.). Oxford, UK: Oxford University Press.

Gross, J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*(3), 281–291.

Hayes, A. M., Feldman, G. C., Beevers, C. G., Laurenceau, J.-P., Cardaciotto, L., & Lewis-Smith, J. (2007). Discontinuities and cognitive changes in an exposure-based cognitive therapy for depression. *Journal of Consulting and Clinical Psychology, 75*, 409–421.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (2012). *Acceptance and commitment therapy: The process and practice of mindful change* (2nd ed.). New York: Guilford Press.

Higgins, E. T. (1997). Beyond pleasure and pain. *American Psychologist, 52*, 1280–1300.

Johnstone, T., van Reekum, C. M., Urry, H. L., Kalin, N. H., & Davidson, R. J. (2007). Failure to regulate: Counterproductive recruitment of top-down prefrontal–subcortical circuitry in major depression. *Journal of Neuroscience, 27*, 8877–8884.

Kabat-Zinn, J. (1990). *Full catastrophe living: Using the wisdom of your body and mind to face stress, pain, and illness*. New York: Delta Trade.

Kabat-Zinn, J. (1994). *Wherever you go, there you are*. New York: Hyperion Press.

Kalisch, R., Wiech, K., Critchley, H. D., Seymour, B., O'Doherty, J. P., Oakley, D. A., et al. (2005). Anxiety reduction through detachment: Subjective, physiological, and neural effects. *Journal of Cognitive Neuroscience, 17*, 874–883.

Kanter, J. W., Manos, R. C., Bowe, W. M., Baruch, D. E., Busch, A. M., & Rusch, L. C. (2010). What is behavioral activation?: A review of the empirical literature. *Clinical Psychology Review, 30*, 608–620.

Kazdin, A. E., & Blase, S. L. (2011). Rebooting psychotherapy research and practice to reduce the burden of mental illness. *Perspectives on Psychological Science, 6*, 21–37.

Kendler, K. S., Gardner, C. O., Gatz, M., & Pedersen, N. L. (2006). The sources of comorbidity between major depression and generalized anxiety disorder in a Swedish national twin sample. *Psychological Medicine, 37*, 453–462.

Kessler, R., Berglund, P., Demler, O., Jin, R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*, 593–602.

Klenk, M. M., Strauman, T. J., & Higgins, E. T. (2011). Regulatory focus and anxiety: A self-regulatory model of GAD–depression comorbidity. *Personality and Individual Differences, 50*, 935–943.

Kraemer, H., Wilson, G., Fairburn, C., & Agras, W. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry, 59*, 877–883.

Kross, E., & Ayduk, Ö. (2009). Boundary conditions and buffering effects: Does depressive symptomology moderate the effectiveness of self-distancing for facilitating adaptive emotional analysis? *Journal of Research in Personality, 43*(5), 923–927.

Krueger, R. F., & Markon, K. E. (2006). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology, 2*, 111–133.

Lanaj, K., Chang, C.-H., & Johnson, R. E. (2012). Regulatory focus and work-related outcomes: A meta-analysis. *Psychological Bulletin, 138*, 998–1034.

Lang, P. J., Davis, M., & Öhman, A. (2000). Fear and anxiety: Animal models and human cognitive psychophysiology. *Journal of Affective Disorders, 61*, 137–159.

LeDoux, J. E. (1996). *The emotional brain: The mysterious underpinnings of emotional life*. New York: Simon & Schuster.

Leary, M. R., Tate, E. B., Adams, C. E., Allen, A. B., & Hancock, J. (2007). Self-compassion and reactions to unpleasant self-relevant events: The implications of treating oneself kindly. *Journal of Personality and Social Psychology, 92*, 887–904.

Lewinsohn, P. M. (1974). A behavioral approach to depression. In R. M. Friedman & M. M. Katz (Eds.), *The psychology of depression: Contemporary theory and research* (pp. 157–185). Washington, DC: Winston-Wiley.

Linehan, M. (1993). *Skills training manual for treating borderline personality disorder*. New York: Guilford Press.

Lissek, S. (2012). Toward an account of clinical anxiety predicated on basic, neurally mapped mechanisms of Pavlovian fear-learning: The case for conditioned overgeneralization. *Depression and Anxiety, 29*, 257–263.

Lohr, J. M., Olatunji, B., & Sawchuk, C. (2007). A functional analysis of danger and safety signals in anxiety disorders. *Clinical Psychology Review, 27*, 114–126.

Mennin, D. S., Ellard, K. K., Fresco, D. M., & Gross, J. J. (2013). United we stand: Emphasizing commonalities across cognitive-behavioral

therapies within a broadening field of intervention science. *Behavior Therapy, 44*(2), 234–238.

Mennin, D. S., & Fresco, D. M. (2009). Emotion regulation as an integrative framework for understanding and treating psychopathology. In A. M. Kring & D. M. Sloan (Eds.), *Emotion regulation in psychopathology: A transdiagnostic approach to etiology and treatment* (pp. 356–379). New York: Guilford Press.

Mennin, D. S., & Fresco, D. M. (2011, November). *Emotion regulation therapy for complex and refractory presentations of anxiety and depression*. A spotlight presentation at the annual meeting of the Association for Behavioral and Cognitive Therapies, Toronto, Canada.

Mennin, D. S., Fresco, D. M., & Aldao, A. (2012, November). *Increasing vagal flexibility predicts improved clinical outcomes in emotion regulation therapy for GAD*. A paper delivered at the annual meeting of the Association for Behavioral and Cognitive Therapies, National Harbor, MD.

Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2005). Preliminary evidence for an emotion dysregulation model of generalized anxiety disorder. *Behaviour Research and Therapy, 43*, 1281–1310.

Mennin, D. S., Holaway, R. M., Fresco, D. M., Moore, M. T., & Heimberg, R. G. (2007). Delineating components of emotion and its dysregulation in anxiety and mood psychopathology. *Behavior Therapy, 38*(3), 284–302.

Mogg, K., & Bradley, B. P. (2005). Attentional bias in generalized anxiety disorder versus depressive disorder. *Cognitive Therapy and Research, 29*, 29–45.

Muraven, M. R., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin, 126*, 247–259.

Newman, M. G., & Llera, S. (2011). A novel theory of experiential avoidance in generalized anxiety disorder: A review and synthesis of research supporting a contrast avoidance model of worry. *Clinical Psychology Review, 31*, 371–382.

Newman, M. G., Przeworski, A., Fisher, A. J., & Borkovec, T. D. (2010). Diagnostic comorbidity in adults with generalized anxiety disorder: Impact of comorbidity on psychotherapy outcome and impact of psychotherapy on comorbid diagnoses. *Behavior Therapy, 41*, 59–72.

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3*, 400–424.

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14*, 1215–1229.

O'Doherty, J. P. (2004). Reward representations and reward-related learning in the human brain: Insights from neuroimaging. *Current Opinion in Neurobiology, 14*, 769–776.

Olatunji, B. O., Cisler, J. M., & Tolin, D. F. (2010). A meta-analysis of the influence of comorbidity on treatment outcome in the anxiety disorders. *Clinical Psychology Review, 30*, 642–654.

Porges, S. (2001). The polyvagal theory: Phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology, 42*, 123–146.

Posner, M. I., & Rothbart, M. K. (1992). Attentional regulation—from mechanism to culture. *International Journal of Psychology, 27*, 41–57.

Ray, R. D., Wilhelm, F. H., & Gross, J. J. (2008). All in the mind's eye?: Anger rumination and reappraisal. *Journal of Personality and Social Psychology, 94*, 133–145.

Roemer, L., & Orsillo, S. M. (2009). *Mindfulness- and acceptance-based behavioral therapies in practice*. New York: Guilford Press.

Rottenberg, J., & Gross, J. J. (2003). When emotion goes wrong: Realizing the promise of affective science. *Clinical Psychology Science and Practice, 10*, 227–232.

Schiller, D., & Delgado, M. R. (2010). Overlapping neural systems mediating extinction, reversal and regulation of fear. *Trends in Cognitive Sciences, 14*, 268–276.

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression: A new approach to preventing rela*pse. New York: Guilford Press.

Sheppes, G., & Gross, J. J. (2011). Is timing everything?: Temporal considerations in emotion regulation. *Personality and Social Psychology Review, 15*(4), 319–331.

Smallwood, J., & Schooler, J. W. (2006). The restless mind. *Psychological Bulletin, 132*, 946–958.

Sokol-Hessner, P., Camerer, C. F., & Phelps, E. A. (2013). Emotion regulation reduces loss aversion and decreases amygdala responses to losses. *Social Cognitive and Affective Neuroscience, 8*(3), 341–350.

Stein, M. B., & Paulus, M. P. (2009). Imbalance of approach and avoidance: The yin and yang of anxiety disorders. *Biological Psychiatry, 66*(12), 1072–1074.

Watkins, E., Teasdale, J. D., & Williams, R. M. (2000). Decentering and distraction reduce overgeneral autobiographical memory in depression. *Psychological Medicine, 30*, 911–920.

Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin, 134*, 163–206.

Watson, D. (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology, 114*(4), 522–536.

Westra, H., Arkowitz, H., & Dozois, D. (2009). Adding a motivational interviewing pretreatment to cognitive behavioral therapy for generalized anxiety disorder: A preliminary randomized controlled trial. *Journal of Anxiety Disorders, 23*, 1106–1117.

Whitmer, A. J., & Gotlib, I. H. (2012). Switching and backward inhibition in major depressive disorder: The role of rumination. *Journal of Abnormal Psychology, 121*(3), 570–578.

Wilson, K. G., & Murrell, A. R. (2004). Values work in acceptance and commitment therapy. In S. C. Hayes, V. M. Follette, & M. Linehan (Eds.), *Mindfulness and acceptance: Expanding the cognitive-behavioral tradition*. New York: Guilford Press.

Woody, S., & Rachman, S. (1994). Generalized anxiety disorder (GAD) as an unsuccessful search for safety. *Clinical Psychology Review, 14*, 745–753.

CHAPTER 29

Dialectical Behavior Therapy: An Intervention for Emotion Dysregulation

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Our aim in this chapter is to describe a set of emotion regulation skills developed within the context of dialectical behavior therapy (DBT; Linehan, 1993a, 1993b) and their potential to serve as a transdiagnostic intervention. DBT is a comprehensive cognitive-behavioral treatment originally developed for suicidal individuals and later expanded to suicidal individuals meeting criteria for borderline personality disorder (BPD). DBT has since been adapted to treat BPD with several comorbidities and other psychological disorders in which problems in emotion regulation lead to psychopathology. Data for the efficacy of DBT are extensive, including 43 clinical trials conducted across 21 independent research teams (for a review, see Neacsiu & Linehan, in press).

DBT conceptualizes difficulties with emotion regulation as a consequence of biosocial transactions: A biological sensitivity to emotions interacts with aversive or invalidating experiences during childhood and adolescence, and leads to neurobiological malfunction and to insufficient skills to manage the emotional system (Linehan, 1993a; Crowell, Beauchaine, & Linehan, 2009). Therefore, a primary focus in DBT is to teach clients how to regulate emotional responses actively.

In this chapter we present a model of emotion regulation that we teach in DBT. We argue that BPD is a disorder of pervasive emotion dysregulation, and also provide examples of how this model is relevant to people without BPD who have difficulties managing emotions. We then present how the DBT skills map onto the model of emotion regulation, and how they can be used systematically to change dysregulation. We conclude by describing the research needed to evaluate the model proposed.

Emotion and Emotion Dysregulation

The DBT Model of Emotion Regulation

Similar to many others (e.g., Ekman & Davidson, 1994), in DBT we consider emotions to be complex, brief, involuntary, patterned, full-system responses to internal and external stimuli. DBT emphasizes the importance of the evolutionary adaptive value of emotions (Tooby & Cosmides, 1990). Although emotional responses are viewed as systemic, we present them to clients as comprising six transacting subsystems that are practical in both understanding and learning to regulate emotions: (1) emotion vulnerability factors; (2) internal and/or external events that serve as emotional cues; (3) interpretations

of cues; (4) emotional response tendencies, including physiological responses, cognitive processing, experiential responses, and action urges; (5) nonverbal–verbal expressive responses and actions; and (6) aftereffects of the initial emotion, including secondary emotions (see Linehan, 1993a).

Although the DBT emotion model was developed to serve a clinical population, it is interesting to note the similarities it has to the modal model of emotions that evolved in basic science (Gross & Thompson, 2007). Briefly, in the modal model, emotions originate from person–situation transactions that are relevant to one's goals and values. Such a situation acts as a cue and draws the individual's attention, gives rise to an appraisal of the event, and leads to an emotional response. This response is associated with behavioral displays and action tendencies, and is malleable, in that the course of the emotion is not fixed when it starts (Gross & Thompson, 2007).

Both the DBT and the modal model include the importance of attending to a cue within a relevant context, highlight appraisals as potentially influencing the course of the emotion and present how emotions directly affect context. Nevertheless, DBT places more emphasis on difficulties regulating the emotional response *after* it has already been initiated, especially when it is past the point at which it could be suppressed. Therefore, one difference between the models is that in DBT the emotional response is broken into experiential and expressive responses. Research supporting the importance of this distinction for clinical populations suggests that processes occurring after the emotional firing are key for psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010).

An additional difference is the inclusion of emotion vulnerability factors. The construct of *emotion vulnerability* refers to the effects of distal and proximal prior events on the initiation, course, and intensity of emotional responses. For example, individuals diagnosed with BPD who meet diagnostic criteria for co-occurring posttraumatic stress disorder (PTSD; a distal vulnerability factor) report significantly higher emotion dysregulation (Harned, Rizvi, & Linehan, 2010). Similarly, sufficient sleep (a proximal vulnerability factor) leads to less emotional intensity when compared to lack of sleep (Gujar, Yoo, Hu, & Walker, 2011).

In this chapter, we reorganize Linehan's original model (1993a) to be applicable to a wider range of disorders and to use terminology consistent with basic research models (Figure 29.1; Table 29.1). Briefly, in this model, emotions start within the context of a situation, where a cue grabs the individual's attention. The cue is appraised or interpreted, which triggers an emotional response that comprises biological–experiential changes (including urges or response tendencies) and expressions–actions changes (including body language, facial expression, and actions). All components are affected by proximal and distal emotion vulnerability

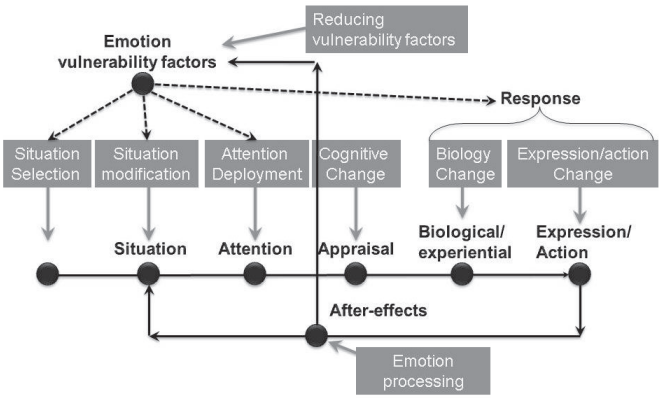

FIGURE 29.1. DBT extended model of emotion regulation. Adapted to be consistent with Gross and Thompson (2007).

TABLE 29.1. Emotion Regulation Tasks and Corresponding DBT Skills

Emotion components	Regulation strategies	DBT skills
A. Emotion Vulnerability Factors	Managing Vulnerability Factors (Biological and Contextual)	Change Biological Sensitivity (PLEASE skills) **A**ccumulate Positives **B**uild Mastery **C**ope Ahead by Covert Rehearsal [Mindfulness Skills]
B. Situation (emotional cue)	Situation Selection Situation Modification	Problem Solving Interpersonal Effectiveness Skills [Mindfulness Skills]
C. Attention	Attention Deployment	Distract Crisis Survival Skills [Mindfulness Skills]
D. Appraisal	Cognitive Change	Check the Facts Reality Acceptance [Mindfulness Skills]
E. Biological/ Experiential Response	Biological Change	Change Physiology (TIP skills) Self-Soothe Half-Smile/Willing Hands [Mindfulness Skills]
F. Expression/ Action Response	Expression and Action Change	Opposite Action [Mindfulness Skills]
G. Emotional After-Effects (including emotional awareness)	Emotional Processing	Identify and Label Emotions [Mindfulness Skills]

Note. See text for explanation of acronyms.

factors. The emotional response is followed by aftereffects, including secondary emotions.

To give an example: A depressed woman has a fight with her partner over house chores, followed by a night of poor sleep. Within the context of these *emotion vulnerability* factors, she walks into the kitchen and a pile of dirty dishes [situation] in the sink captures her *attention*. She *appraises* the situation by thinking "The dishes are dirty"; "I should have washed them"; and "I didn't so I'm a terrible wife." The emotion continues, with her heart beating faster, her body slumping lower; she has the urge to go to bed and hide under the covers [biological response–action urge] and she starts crying [expression–action response]. Her attention narrows [aftereffect] and, as she walks around the house, she sees the clothes [situation] she did not iron [appraisal], which refires her emotion of shame. She goes to bed and covers her head with the blanket [action response] and subsequently becomes angry with herself [secondary emotion].

Figure 29.1 and Table 29.1 present types of strategies that can be used to change each emotion component. Like Davidson (1998) we contend that emotion regulation can be both automatic and effortful, and that regulatory processes are an integral part of emotional responding.

Pervasive Emotion Dysregulation

Emotion dysregulation is the inability, even when one's best efforts are applied, to change *in a desired way* emotional cues, experiences, actions, verbal responses, and/or nonverbal expressions under normative conditions. Characteristics of emotion dysregulation include an excess of aversive emotional experiences, an inability to regulate intense physiological arousal, problems turning attention away from stimuli, cognitive distortions and failures in information

processing, insufficient control of impulsive behaviors related to strong emotions, difficulties organizing and coordinating activities to achieve non-mood-dependent goals when emotionally aroused, and a tendency to "freeze" or dissociate under very high stress (Ray et al., 2006). *Pervasive emotion dysregulation* refers to an inability to regulate emotions that occurs across a wide range of emotions and situational contexts.

BPD: A Disorder of Pervasive Emotion Dysregulation

The Disorder

BPD is a severe mental disorder with a serious dysregulation of the emotion system at its core. Clients show a characteristic pattern of instability in emotion regulation, impulse control, interpersonal relationship, and self-image. The often severe functional impairment leads to substantial treatment utilization and a mortality rate by suicide of almost 10%, which is 50% higher than the rate in the general population (American Psychiatric Association, 2001). BPD affects approximately 3% of the general population, up to 10% of outpatients treated for mental disorders, and up to 20% of inpatients (Trull, Jahng, Tomko, Wood, & Sher, 2010). Because of the severity of the disturbance and the intensive treatment use, clients with BPD constitute a disproportionately large subset of psychiatric patients, consuming considerably more mental health resources than most other psychiatric groups (Soeteman, Hakkaart-van Roijen, Verheul, & Busschbach, 2008).

BPD as a Disorder of Emotion Regulation

Based on clinical experience, Linehan (1993a) proposed that pervasive emotion dysregulation in BPD is caused by an interplay between biological (e.g., genetic, intrauterine factors; trauma to the biological system) vulnerability and aversive sociobiographical experiences. According to this biosocial theory, a child born with heightened biological sensitivity to emotional cues encounters emotionally aversive experiences (e.g., interpersonal violence, social rejection, emotional neglect, invalidation) and consequently develops biological and psychological alterations of the emotion regulation system (Linehan, 1993a; Distel et al., 2011). The neurobiological alterations in the emotion circuitry manifest in adulthood as heightened emotional sensitivity (low threshold for recognition of/response to emotional stimuli), heightened reactivity (high amplitude of emotional responses), and a slow return to baseline after emotion induction (Linehan, 1993a; Crowell et al., 2009). The psychological alterations involve maladaptive or insufficient learning in how to understand, label, regulate, or tolerate emotional responses effectively. A BPD diagnosis is hypothesized to emerge from such biological alterations, coupled with insufficient knowledge about emotion regulation.

Existing evidence provides emerging support for this theory. First, 60% of clients with BPD report sexual abuse and severe interpersonal violence during childhood (Hernandez, Arntz, Gaviria, Labad, & Gutiérrez-Zotes, 2012; Bornovalova et al., 2013), which leads to significantly higher suicidality and emotion dysregulation (Harned et al., 2010). Second, animal research shows that traumatization during early life stages leads to morphological alterations of the central frontolimbic system and to behavioral and epigenetic modifications (Pryce & Feldon, 2003; Cirulli et al., 2009). Third, preliminary data connect genetic factors with the development of borderline features (Distel et al., 2009).

Neurobiological Dysfunction in BPD

Numerous studies have tested biological alterations in the emotion circuitry in BPD, with the majority of studies assessing reactivity and return to baseline. Findings are extensive and somewhat mixed; next, we present some highlights of this body of literature.

Functional and structural data support enhanced reactivity and slow return to baseline in BPD. In this population, volume reduction of the amygdala (Nunes et al., 2009) was correlated with amygdala hyperreactivity to emotional stimuli (e.g., Herpertz et al., 2001; Niedtfeld et al., 2012). This heightened amygdala activation was more prominent in BPD and took longer to return to baseline when compared to clini-

cal and nonclinical controls (Hazlett et al., 2012). In addition to amygdala dysfunction, volume reductions in brain areas hypothesized to serve emotion regulation functions were also found (Tebartz van Elst et al., 2003; Minzenberg, Fan, New, Tang, & Siever, 2008). Recent studies also report reduced neural connectivity between such brain areas and the amygdala at baseline (New et al., 2007) or during emotional distress (Niedtfeld et al., 2012). Furthermore, research indicates a reduced activation of prefrontal areas after emotional induction (Minzenberg, Fan, New, Tang, & Siever, 2007; Schulze et al., 2011). These findings suggest that in BPD amygdala hyperactivity in the presence of emotional stimuli takes longer to return to baseline, partly because of insufficient modulation from brain centers responsible for emotion regulation.

Self-report and psychophysiological findings are less clear with regard to heightened reactivity in individuals diagnosed with BPD (for a detailed review, see Rosenthal et al., 2008). Across several samples, people diagnosed with BPD self-report being more reactive, having more negative emotions, and experiencing more emotional instability than non-BPD controls (Rosenthal et al., 2008; Lobbestael & Arntz, 2010). Additional studies using heart rate as a psychophysiological measures of distress (Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2010; Reitz et al., 2012), or electrophysiological recordings (Marissen, Meuleman, & Franken, 2010) also found greater emotional reactivity for participants with BPD. Nevertheless, studies using different psychophysiological indices of distress (i.e., skin conductance response) failed to show enhanced reactivity (e.g., Kuo & Linehan, 2009; Rosenthal et al., 2008). Thus, additional research is needed to better understand the mixed findings on reactivity.

Self-report and psychophysiological data support a prolonged return to baseline. Applying ambulatory assessments under daily life conditions, Stiglmayr, Shapiro, Stieglitz, Limberger, and Bohus (2001) reported significantly longer intervals of activated aversive emotions but no nonspecific arousal for people with BPD compared to healthy controls. In addition, in a laboratory study participants diagnosed with BPD evidenced prolonged return to baseline after induced anger (Jacob et al., 2008) and stress (Reitz et al., 2012).

Emotion Dysregulation in BPD

It is important to highlight that the construct of emotion dysregulation is independent from neurobiological alterations in the emotion circuitry that are thought to underlie BPD. Having increased sensitivity, reactivity and a slow return to baseline may make it more difficult for individuals to regulate emotions. At the same time, persistent use of dysfunctional regulation strategies may lead to continued biological alteration. Whether neurobiological alterations precede dysregulation, or vice versa, remains an empirical question. Nevertheless, both constructs are crucial for understanding difficulties with emotions as presented in DBT.

We propose that individuals with BPD have pervasive emotion dysregulation. In support, below we summarize evidence suggesting that individuals diagnosed with BPD have problems with each set of emotion regulation strategies described in the DBT model. Difficulties with reducing emotion vulnerability are highlighted by findings suggesting dysregulated sleep patterns in BPD individuals (Schredl et al., 2012), high prevalence of abuse history and substance use disorders (Trull et al., 2010; Distel et al., 2012), as well as chronic health problems and poor lifestyle choices (Frankenburg & Zanarini, 2004).

Self-inflicted injuries (including suicide attempts) and most other dysfunctional behaviors (i.e., suicide threats, impulsive behaviors, dissociation) are hypothesized to be maladaptive problem-solving strategies (Reitz et al., 2012), an escape mechanism (Chapman, Gratz, & Brown, 2006), or a way to communicate distress (Koerner & Linehan, 1997). These behaviors may suggest problems with regulating the biological and expressive components of the emotion, or with selecting effective situation modification strategies. Problems with emotion-induced dissociation have also consistently been shown and suggest impairments in the attention regulation component (Ebner-Priemer et al., 2009). In addition, research has documented problems with cognitive flexibility (Ruocco, 2005), cognitive change (Selby & Joiner, 2009), emotional awareness (Levine, Marzialli, & Hood, 1997), and aftereffects (Korfine & Hooley, 2000),

suggesting difficulties with regulation of all the remaining emotion components.

The DBT Model and Other Disorders

Emotional dysregulation has been reported to underlie etiological and maintenance mechanisms for a large number of mental health problems (Kring & Sloan, 2010). Literature reviews have demonstrated that mood disorders, anxiety disorders, substance use disorders (SUDs), eating disorders, schizophrenia, and even psychotic disorders are directly linked to emotion dysregulation (Cisler, Olatunji, Feldner, & Forsyth, 2010; Harrison, Sullivan, Tchanturia, & Treasure, 2009; Kring & Werner, 2004; Thorberg, Young, Sullivan, & Lyvers, 2009).

Individuals diagnosed with a variety of Axis I disorders also appear to reveal emotion dysregulation patterns. Increased reactivity when compared to controls has been connected to generalized anxiety disorder (GAD; Mennin, Heimberg, Turk, & Fresco, 2005), substance dependence (Thorberg et al., 2009), social anxiety disorder (SAD), and specific phobias (Etkin & Wager, 2007). Furthermore, the transaction between neurobiological dysfunction (e.g., anxiety sensitivity) and lack of skills to manage it is similar to the etiological theories presented in panic disorder (Barlow, Allen, & Choate, 2004), GAD (Mennin et al., 2005), and some specific phobias (Cisler et al., 2010). It can therefore be hypothesized that in cases where emotion dysregulation is involved, reactivity to emotional cues coupled with insufficient regulation strategies may result in psychopathology. Thus, emotion dysregulation may be a transdiagnostic phenomenon.

People diagnosed with BPD have difficulties with regulating all of the emotion subsystems described in the DBT model. Similar difficulties can be found in many additional Axis I disorders, as described in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5). Difficulties managing emotion vulnerability factors are common in mood and anxiety disorders and in SUDs (e.g., Monti & Monti, 2000; Wong, Brower, Fitzgerald, & Zucker, 2004). Individuals diagnosed with anxiety and mood disorders also make negative interpretations (e.g., catastrophizing) that lead to dysregulated emotions, and use maladaptive situation selection and situation modification strategies (e.g., avoidance, use of safety cues; Kring & Werner, 2004; Aldao et al., 2010). Experimental evidence also makes a strong link between depression and anxiety, and difficulties regulating attention in emotional contexts. For example, depressed individuals have a reduced capacity to sustain positive emotions (Heller et al., 2009) and more difficulties finding attentional distracters in the context of a distressing emotion (Koole, 2009) when compared with controls. Similarly, when presented with social threat stimuli, participants with SAD report more negative emotions than do controls, suggesting attention deployment and/or cognitive change dysregulation (Goldin, Manber, Hakimi, Canli, & Gross, 2009).

Finally, difficulties with emotion processing have been linked to PTSD (Frewen, Dozois, Neufeld, & Lanius, 2012).

DBT Emotion Regulation Skills

Rationale

Linehan (1993a) included in DBT a set of concrete skills translated from behavioral research and other evidence-based treatments, aimed to address emotion dysregulation in BPD. It was therefore hypothesized that increases in skills use led to improvements in emotion regulation, which in turn led to positive outcomes in treated populations. Empirical findings indeed suggest that use of DBT skills explains changes in depression, anger regulation, and suicidal behavior across BPD treatments (Neacsiu, Rizvi, & Linehan, 2010). Therefore, behavioral skills are likely a potent mechanism of change for emotion dysregulation.

In addition to the five sets of regulatory processes proposed by Gross and Thompson (2007), DBT targets five additional processes: managing emotion vulnerability factors, biological change, expression and action change, and emotional processing (at the point of emotional aftereffects). We next present the skills that directly map onto the DBT model of emotions, organizing them according to regulation process to which they primarily relate, although the functions

of each set of skills can be applied across many of the regulation processes (see Table 29.1). This is particularly the case for mindfulness skills, which can be viewed as critical at every juncture in the emotion regulation process.

Strategies for Managing Emotion Vulnerability Factors

DBT teaches clients to decrease emotion vulnerability factors by increasing happiness and resilience ("building a life worth living") through a set of skills that target biological homeostasis and influence emotional reactivity. The *PLEASE* skills target treating *P*hysical i*L*lness (Anderson, Hackett, & House, 2004), balancing nutrition and *E*ating (Smith, Williamson, Bray, & Ryan, 1999), staying off nonprescribed mood-*A*ltering drugs, getting sufficient but not excessive *S*leep (Gujar et al., 2011), and getting adequate *E*xercise (Cox, Thomas, Hinton, & Donahue, 2004).

In addition, DBT promotes resilience by teaching skills for *accumulating positive life events* and for *building* a sense of generalized *mastery*. Increasing the number of pleasurable events in one's life is one approach to increasing positive emotions. In the short term, this involves increasing daily positive experiences. In the long term, it means working on goals related to important life values, so that pleasant events will occur more often. Building mastery is achieved by engaging in activities that increase a sense of competence and self-efficacy. The focus is very similar to activity and mastery scheduling in psychotherapy for depression (Martell, Addis, & Jacobson, 2001). Both skills have been shown to predict decreased vulnerability to negative emotional states (Joiner, Lewinsohn, & Seeley, 2002; de Beurs et al., 2005).

Coping ahead (Linehan, in press) is an additional skill that promotes contextual resiliency. Individuals learn to use imaginal exposure and rehearsal to cope successfully with a difficult situation ahead of time. For people who are prone to dysregulation, coping ahead via covert rehearsal can be helpful in building the coping skills necessary for problem solving (see Fourkas, Avenanti, Urgesi, & Aglioti, 2006). Thus, this skill is likely to increase peoples' appraisal of their own ability to cope with an emotional event, effectively increasing a sense of mastery and self-efficacy.

Situation Selection–Modification Strategies

Situation selection and modification are two classes of emotion regulation strategies through which emotions are modulated via stimulus control (i.e., avoiding or modifying situations that generate unwanted emotions). To promote effective situation selection skills, DBT teaches how to generate a list of pros and cons to guide a course of action. To improve situation modification, DBT teaches a simple set of *problem-solving* skills (Linehan, in press) aimed at changing or developing strategies for eliminating, reducing, or avoiding emotionally problematic situations. The focus here is on defining those situations that cue unwanted emotions, then applying standard problem-solving steps, such as those outline by D'Zurilla and Nezu (1999) and others.

Because many problems are interpersonal (and even if they are not may require interpersonal interactions to solve), DBT also includes a set of *interpersonal effectiveness skills*. These skills focus on how to obtain a wanted objective without hurting the interpersonal relationship or one's own self-respect. Coping ahead as a way to practice these skills and to manage intense emotional arousal before it happens is often a helpful addition to problem solving.

DBT also includes a set of mindfulness skills that emphasize observing, describing, and participating in the present moment effectively and without judgment. These skills may also promote adaptive situation selection by nonjudgmentally expanding awareness regarding situations that in the past have evoked emotional experience. This awareness is hypothesized to increase sensitivity to the current contingencies in the environment, allowing the opportunity for new learning. Thus, by seeing reality "as it is" (i.e., being in the present moment without historical filters), mindfulness may enhance the ability of an individual to decide what situations to avoid, when to attempt to problem-solve, or when to cope ahead.

Attentional Deployment Strategies

Mindfulness skills are often used in DBT to promote attentional control, which can reduce problems with attentional deployment. Mindfulness involves learning to *control the focus of attention*, not the object to which one attends (e.g., observing a thought as a thought or emotion as emotion, without attempting to change the thought or emotion). Being able to disengage from emotional stimuli may reduce the tendency to experience negative affect (Ellenbogen, Schwartzman, Stewart, & Walker, 2002), and redeploying attention has been postulated to lead to a "flexibility of attention" (Teasdale, Segal, & Williams, 1995) needed for successful attention modulation in emotional contexts. Thus, mindfulness may help modulate emotional experience by enhancing the practioner's ability to turn his or her attention away from that which is not useful (or effective) and attend to what is (Lynch, Rosenthal, et al., 2006).

Cognitive Change Strategies

DBT focuses on analyzing and correcting situation appraisals by *checking the facts* (Linehan, in press). These skills focus on discriminating assumptions, interpretations, ruminative thoughts, and worries from the actual observed facts of situations. Support for this set of skills comes from a number of studies comparing different reappraisal strategies, including nonappraisal control conditions, following presentation of emotional cues (e.g., Lazarus & Abramovitz, 1962).

An additional set of strategies (*reality acceptance*) targets changing one's appraisal of emotions as experiences that cannot be tolerated or experienced willingly. Emotion acceptance, when compared to emotion suppression, or a control, has been shown to result in less subjective anxiety or avoidance in clients diagnosed with panic disorder undergoing a carbon dioxide challenge (Levitt, Brown, Orsillo, & Barlow, 2004). In addition, coaching in an acceptance mindset, compared to coaching in a control-your-emotions mindset or a placebo condition, significantly increased the amount of time a subject was willing to spend in a cold pressor task (Hayes et al., 1999). With respect to emotions, DBT reality acceptance skills ("turning the mind" toward acceptance, radical acceptance, and willingness over willfulness) focus on *radical* (meaning full and complete) acceptance of the current emotion and willingness to experience even aversive emotions.

Mindfulness skills may also alter situation appraisal by reducing literal belief in emotional appraisals. Mindfulness teaches individuals to observe appraisals as only thoughts that are not necessarily literally true. This is hypothesized to increase sensitivity to the current contingencies in the environment, allowing the opportunity for new learning. In this context, mindfulness in DBT would *not* be predicted to reduce the frequency of distressing thoughts but instead to decrease the influence these thoughts have on subsequent behavior and emotions.

Biological–Experiential Change Strategies

In DBT, an important part of the biological component of an emotion is the *action tendency*, or urge, to act in a specific manner. DBT provides a range of *distress tolerance skills* whose aim is to inhibit acting on maladaptive urges that interfere with long-term emotion regulation. These skills are also designed to down-regulate the extreme physiological arousal that often accompanies intense emotions. The function of these skills is to impact high arousal quickly, without requiring a high level of cognitive processing to complete. Grouped under the term TIP skills (Linehan, in press) these skills target activation of the parasympathetic nervous system.

The first skill (*T*emperature change with ice water) has to do with using cold, icy water on the face to trigger the human dive reflex (which is typically elicited to aid survival when falling into a frozen lake). This reflex can be triggered by a combination of breath holding and face immersion in cold water. The physiological response that follows involves both branches of the autonomic nervous system and reduces emotional arousal for a short period of time (Hurwitz & Furedy, 1986).

Intense exercise is also recommended if arousal is very high. Most important here is the intensity of the exercise. Cox and col-

leagues (2004) compared intensity of bouts of exercise and found that while intensity of exercise conditions did not differ in state anxiety immediately after exercise, a significant difference favoring *the most intense exercise* condition over the control condition emerged at 30 minutes postexercise.

Additional distress tolerance strategies are *P*aced breathing and *P*rogressive relaxation, soothing one of the five senses, adopting an opened posture with palms facing the ceiling (willing hands), or adopting a serene facial expression (half smile).

Expression and Action Change Strategies

Changing expression and action components of emotions implies preventing emotional actions, or acting in a way that opposes or is inconsistent with the emotion. The DBT skill of *opposite action* is based on the idea that not only is changing action tendencies essential for reducing emotional disorders, but also that deliberate actions opposite to those associated with unwanted emotions can effectively change emotions as well as action tendencies. Others have made this same point (e.g., Barlow, 1988, p. 313). Izard (1977) stated that treatment for anxiety disorders involves "the individual learn[ing] to act his way into a new feeling" (cited in Barlow, 1988, p. 410).

Opposite action in DBT applies principles of exposure-based treatments for anxiety disorders across the entire domain of emotions. All exposure-based interventions include this one common element: Individuals have to approach the object/situation that is fearful, thus acting counter to (and inhibiting) their prominent urges to avoid. Effective treatments for anger also require individuals to act counter to the urges associated with anger (attack physically or verbally) by leaving the situation. Anger interventions also focus on taking the opposite perspective, and shifting from aggression and blame to gentleness and forgiveness (e.g., Tafrate, Kassinove, & Dundin, 2002). A number of researchers have observed that effective therapies for depression also share a common thread: They activate behavior. For example cognitive therapy (Beck, Rush, Shaw, & Emery, 1979) and behavioral activation (BA; Martell et al., 2001) require that the individual galvanize him- or herself to engage in activities that result in a sense of mastery or pleasure. This engagement runs counter to the depression urge to withdraw and shut down.

Opposite action "all the way" (Linehan, in press) targets changing the entire range of physical responses that accompany action, including visceral responses, body postures, facial expressions, and movements. A large literature has demonstrated that the activation of specific physical states activates the other facets of the corresponding emotion responses, whether via the face (e.g., Duclos & Laird, 2001), posture (Stepper & Strack, 1993) or respiration. To the contrary, there is ample empirical evidence that modulating one's physical state alters one's emotional state (Philippot, Baeyenes, Douilliez, & Francart, 2004). Also implied by "all the way" are emotion-linked thought patterns and verbal responses. The idea here is to act contrary to an emotion, not to mask or hide emotions.

In part, opposite action is hypothesized to work by influencing classically conditioned emotional responses (Lynch, Chapman, Rosenthal, Kuo, & Linehan, 2006). Opposite action may also create sensory feedback from facial muscles and skin that can be transformed directly into emotional experience without cognitive mediation (Izard, 1977). Finally, self-perception of expressive behavior, action, and appraisals regarding proprioceptive sensations has been proposed to influence subjective emotional experience (Laird, 1974). Opposite action may influence emotion by changing the perception of the emotional event. Thus, behavior that is the opposite of the automatic response or action urge of an emotion is intended to alter the meaning of the emotional event automatically and without conscious effort (Lynch, Rosenthal, et al., 2006). In essence, people conclude that they feel safe because they are "acting as if" all is safe.

Strategies for Changing Emotional Aftereffects

Aftereffects of emotions, which include changes in attention, memory, and reasoning, are fairly well established (see Dolan, 2002, for a review). These aftereffects can increase the probability that the emotion

will reoccur. Interrupting the cycle can be enhanced if the individual notices and identifies a current, ongoing emotion, which can then guide application of relevant change strategies.

Therefore, DBT included a skill (*Observe and Describe Emotions*) through which increased awareness of the emotional experience is promoted. This skill is supported by research showing that processing emotional experience with greater specificity has advantages for improved emotion regulation over emotional processing that is overgeneral or nonspecific (e.g., Williams, Stiles, & Shapiro, 1999). Indeed, recent research has demonstrated that priming individuals with overgeneral emotional memories results in more intense emotional experience compared to priming specific emotional memories or a control condition (Schaefer et al., 2003). In addition, experimentally manipulated social anxiety has been shown to be reduced by observing and describing specifically the fear producing cues, in contrast to general impressions regarding cues that resulted in higher fear (Philippot, Burgos, Verhasselt, & Baeyens, 2002).

Drawing from the work of many, including both Shaver (e.g., Shaver, Schwartz, Kirson, & O'Connor, 1987) and Hupka (e.g., Hupka, Lenton, & Hutchinson, 1999), Linehan (in press) expanded the original list of six emotions to a taxonomy of 10 basic emotions: anger, disgust, envy, fear, jealousy, joy, love, sadness, shame, and guilt. For each emotion the following characteristics are listed: (1) family of emotion names associated with the basic emotion, (2) typical prompting events (cues), (3) interpretations or appraisals, (4) biological changes and experiences, (5) expressions and actions, (6) aftereffects, and (7) secondary emotions associated with each family of emotions. Using the taxonomy, clients are coached in learning to observe and describe their emotions relative to various events.

Evidence for DBT Skills as a Treatment for Emotion Dysregulation

Problems with regulating each component of the emotion system can be connected with BPD and with other disorders. As we argued in the previous section, there are DBT skills to target each of the types of emotion dysregulation our model presented. Therefore, DBT skills training is a promising candidate for treating emotion dysregulation in BPD and other Axis I disorders.

Treatment trials, albeit fraught with methodological flaws, offer some support that DBT skills training is effective in reducing emotion dysregulation in various clinical presentations. A DBT skills training intervention improved pre- to posttreatment ratings of depression in abuse victims (Iverson, Shenk, & Fruzzetti, 2009), and depression and anger in vocational rehabilitation clients (Koons et al., 2006). When compared to treatment as usual (TAU) or a wait-list condition, DBT skills training decreased depression in treatment-resistant depressed individuals (Harley, Sprich, Safren, Jacobo, & Fava, 2008) and self-reported anger in a forensic sample (Evershed et al., 2003). DBT skills training was also superior to standard group therapy in improving depression, anger and affect instability in a BPD sample (Soler et al., 2009).

Therefore, although originally developed to be part of a comprehensive intervention, skills training by itself may be the mechanism through which change occurs in a variety of populations with emotion regulation difficulties. Indeed, skills training has been linked to the reduction of emotion dysregulation indices (Neacsiu et al., 2010) and emotion dysregulation has, in turn, been related to a variety of mental health problems (Kring & Sloan, 2010). Although this research area is in its infancy and more findings are needed, the evidence suggests that DBT skills are a promising intervention for emotion dysregulation across psychopathology.

Directions for Future Research

Psychopathology Research

Construct Validity of Emotion Regulation

We have refined the construct of emotion regulation as applicable to psychopathology and presented a testable model. Although we have defined *dysregulation* as dysfunction at either level in the emotion generation–regulation process, we do not know whether one or more "tipping" points differentiate

normative difficulties regulating extreme emotional arousal versus non-normative difficulties that predict serious emotional disturbance. In addition, how difficulties as emphasized by this model vary with each mental disorder is also not yet clear.

Construct Validity of Pervasive Emotion Dysregulation

We have proposed the construct of pervasive emotion dysregulation and conceptualized it as a combination of a tendency to high emotionality across a wide array of both positive and negative emotions, together with an inability to regulate intense emotion-linked responses. The validity of this construct has not been evaluated, nor are there measures of the construct. The high incidence of comorbidity across emotional disorders suggests that the construct may be a useful one. Research is needed both to validate and identify the parameters of the construct. We further have proposed BPD as a model of pervasive emotion dysregulation. Research designed specifically to evaluate this contention, particularly research comparing BPD to other emotional disorders, is needed. Research on BPD and emotions outside of anxiety, depression, and anger is also needed to support or refute the pervasiveness of emotion dysregulation in this disorder.

Neurobiological Dysfunction of the Emotion Circuitry in BPD

We have defined neurobiological dysfunction of the emotion system in BPD as sensitivity to emotional stimuli; intense reactions to such stimuli; and a slow, delayed return to an emotional baseline. First, more research is needed to assess whether individuals with BPD do have a heightened sensitivity to emotional stimuli. Second, although it appears evident that the intensity of all emotions is enhanced in BPD, the empirical evidence that clients with BPD are reactive to emotion-eliciting cues is mixed. Additional research clarifying the inconsistent findings on emotional reactivity in BPD is sorely needed. Furthermore, it is unclear whether mode of stimulation (visual, auditory, somatic, etc.) makes a difference.

We have highlighted clear evidence for both structural and functional alterations in the frontolimbic circuits of clients with BPD. However, it remains unclear whether these findings are specific to clients with BPD or characteristic of individuals with emotion regulation difficulties in general. It is also unclear how neurobiological alterations interact with pervasive emotion dysregulation, especially in the case of social emotions such as shame or guilt. There is only beginning research on restitution of these neurobiological alterations after successful treatment.

Emotion Dysregulation as a Transdiagnostic Phenomenon

As we have highlighted, emotion dysregulation and alterations of the biological architecture of the emotion system are issues not entirely unique to BPD. Therefore, future research should further examine the relationship between problems with emotions and other disorders. Furthermore, much of the research assessing emotion dysregulation in BPD has compared people with BPD to healthy controls (for a review, see Rosenthal et al., 2008). There is a great need for research examining the specificity of emotion dysregulation in BPD, by comparing people with BPD to those with other mental disorders. This could help refine nuances of how emotion dysregulation manifests in psychopathology and how it can be more effectively targeted.

Intervention Research

As noted previously, emerging data indicate that the skills training component of DBT is a successful stand-alone intervention for emotion dysregulation in a variety of clinical samples. What we do not know yet is whether we can use DBT skills training as a transdiagnostic treatment for emotion dysregulation.

In addition, we do not know whether some DBT skills are more useful than others, nor are there data regarding the role of competence of skills application (i.e., whether application of the "right skill at the right time" is important). It is also not clear which skills are the right skills for various situations. Given the propensity for emotional avoidance in many emotionally disturbed individuals, it is extremely important to find out

when to teach clients to distract themselves from unwanted emotions and cues and when to expose themselves to emotions and emotional stimuli. In DBT we make a distinction between moderate and extreme emotional responses. *Extreme emotional responses* are defined as those accompanied by cognitive processing that is so compromised that skills requiring high use of cognitive resources (e.g., problem solving, checking the facts) are unlikely to be successful. With extreme responses, skills that more directly impact somatic arousal (e.g., deep breathing, using ice water) or attention (e.g., distraction) are recommended. Data verifying the wisdom of these recommendations are sorely lacking. This is particularly important in light of the increasing use of mindfulness-based treatment interventions, which teach individuals to notice and accept ongoing emotional responses. The question might be reframed as follows: When is mindfulness of current emotions (a DBT skill) more or less important?

Although there is a fair amount of basic research supporting the specific skills taught in DBT, there is little evidence on their individual effectiveness as treatment interventions in clinical populations. The systematic examination of the DBT skills, both individually and in combination, is an essential first step in improving treatment for deregulated individuals. This is particularly important for the skill of opposite action. Linehan (1993a) has suggested that opposite action will be effective across a wide range of both dysfunctional positive (e.g., loving the wrong person) and negative emotions. One study found promising results for opposite action with shame (Rizvi & Linehan, 2006), but other emotions have not been studied explicitly. Thus, although DBT has been thoroughly evaluated in efficacy studies, there has been substantially less emphasis on the treatment mechanisms of change, and future research must work to narrow this gap.

Conclusion

To sum up, we propose a framework for emotion dysregulation that is applicable to BPD and in theory can be extended to other psychological disorders. We argue that emotion dysregulation is prevalent beyond BPD and invite research to support our model in other disorders in which problems with emotions have been identified. Furthermore, we offer a set of skills aimed at addressing directly common components of dysregulation and illustrate how such skills have been used with individuals diagnosed with BPD. We propose additional ways in which intervention research, as directly relevant to emotion dysregulation, can advance our knowledge and help make treatments for this problem more effective.

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References

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*, 217–237.

American Psychiatric Association. (2001). Practice guideline for the treatment of patients with borderline personality disorder. *American Journal of Psychiatry, 158*(Suppl. 10), 1–52.

Anderson, C. S., Hackett, M. L., & House, A. O. (2004). Interventions for preventing depression after stroke. *Cochrane Database Systems Review, 2*, CD003689.

Barlow, D. H. (1988). *Anxiety and its disorders: The nature and treatment of anxiety and panic*. New York: Guilford Press.

Barlow, D. H., Allen, L. B., & Choate, M. L. (2004). Toward a unified treatment for emotional disorders. *Behavior Therapy, 35*, 205–230.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.

Bornovalova, M. A., Huibregtse, B. M., Hicks, B. M., Keyes, M., McGue, M., & Iacono, W. (2013). Tests of a direct effect of childhood abuse on adult borderline personality disorder traits: A longitudinal discordant twin design. *Journal of Abnormal Psychology*, *122*(1), 180–194.

Chapman, A. L., Gratz, K. L., & Brown, M.

Z. (2006). Solving the puzzle of deliberate self-harm: The experiential avoidance model. *Behaviour Research and Therapy, 44*, 371–394.

Cirulli, F., Francia, N., Berry, A., Aloe, L., Alleva, E., & Suomi, S. J. (2009). Early life stress as a risk factor for mental health: Role of neurotrophins from rodents to non-human primates. *Neuroscience and Biobehavioral Review, 33*, 573–585.

Cisler, J. M., Olatunji, B. O., Feldner, M. T., & Forsyth, J. P. (2010). Emotion regulation and the anxiety disorders: An integrative review. *Journal of Psychopathology and Behavioral Assessment, 32*, 68–82.

Cox, R. H., Thomas, T. R., Hinton, P. S., & Donahue, O. M. (2004). Effects of acute 60 and 80% VO2max bouts of aerobic exercise on state anxiety of women of different age groups across time. *Research Quarterly for Exercise and Sport, 75*, 165–175.

Crowell, S. E., Beauchaine, T. P., & Linehan, M. M. (2009). A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. *Psychological Bulletin, 135*, 495–510.

Davidson, R. J. (1998). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology, 35*, 607–614.

de Beurs, E., Comijs, H., Twisk, J. W., Sonnenberg, C., Beekman, A. T., & Deeg, D. (2005). Stability and change of emotional functioning in late life: Modelling of vulnerability profiles. *Journal of Affective Disorders, 84*, 53–62.

Distel, M. A., Middeldorp, C. M., Trull, T. J., Derom, C. A., Willemsen, G., & Boomsma, D. I. (2011). Life events and borderline personality features: The influence of gene–environment interaction and gene–environment correlation. *Psychological Medicine, 4*(4), 849–860.

Distel, M. A., Trull, T. J., de Moor, M. M., Vink, J. M., Geels, L. M., et al. (2012). Borderline personality traits and substance use: Genetic factors underlie the association with smoking and ever use of cannabis, but not with high alcohol consumption. *Journal of Personality Disorders, 26*(6), 867–879.

Distel, M. A., Trull, T. J., Willemsen, G., Vink, J. M., Derom, C. A., Lynskey, M., et al. (2009). The five-factor model of personality and borderline personality disorder: A genetic analysis of comorbidity. *Biological Psychiatry, 66*(12), 1131–1138.

Dolan, R. J. (2002). Emotion, cognition, and behavior. *Science, 298*, 1191–1194.

Duclos, S. E., & Laird, J. D. (2001). The deliberate control of emotional experience through control of expressions. *Cognition and Emotion, 15*, 27–56.

D'Zurilla, T. J., & Nezu, A. M. (1999). *Problem-solving therapy: A social competence approach to clinical intervention* (2nd ed.). New York: Springer.

Ebner-Priemer, U. W., Mauchnik, J., Kleindienst, N., Schmahl, C., Peper, M., Rosenthal, Z., et al. (2009). Emotional learning during dissociative states in borderline personality disorder. *Journal of Psychiatry and Neuroscience, 34*(3), 214–222.

Ekman, P., & Davidson, R. J. (1994). *The nature of emotion*. New York: Oxford University Press.

Ellenbogen, M. A., Schwartzman, A. E., Stewart, J., & Walker, C. D. (2002). Stress and selective attention: The interplay of mood, cortisol levels, and emotional information processing. *Psychophysiology, 39*, 723–732.

Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry, 164*, 1476–1488.

Evershed, S., Tennant, A., Boomer, D., Rees, A., Barkham, M., & Watson, A. (2003). Practice-based outcomes of dialectical behaviour therapy (DBT) targeting anger and violence, with male forensic patients: A pragmatic and non-contemporaneous comparison. *Criminal Behavior and Mental Health, 13*, 198–213.

Fourkas, A. D., Avenanti, A., Urgesi, C., & Aglioti, S. M. (2006). Corticospinal facilitation during first and third person imagery. *Experimental Brain Research, 168*(1–2), 143–151.

Frankenburg, F. R., & Zanarini, M. C. (2004). The association between borderline personality disorder and chronic medical illnesses, poor health-related lifestyle choices, and costly forms of health care utilization. *Journal of Clinical Psychiatry, 65*(12), 1660–1665.

Frewen, P. A., Dozois, D. A., Neufeld, R. J., & Lanius, R. A. (2012). Disturbances of emotional awareness and expression in posttraumatic stress disorder: Meta-mood, emotion regulation, mindfulness, and interference of emotional expressiveness. *Psychological Trauma: Theory, Research, Practice, and Policy, 4*(2), 152–161.

Goldin, P. R., Manber, T., Hakimi, S., Canli, T., & Gross, J. J. (2009). Neural bases of social anxiety disorder: Emotional reactivity and cognitive regulation during social and physical threat. *Archives of General Psychiatry, 66*, 170–180.

Gratz, K. L., Rosenthal, M. Z., Tull, M. T., Lejuez, C. W., & Gunderson, J. G. (2010). An experimental investigation of emotional reactivity and delayed emotional recovery in borderline personality disorder: The role of shame. *Comprehensive Psychiatry, 51*(3), 275–285.

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York: Guilford Press.

Gujar, N., Yoo, S., Hu, P., & Walker, M. P. (2011). Sleep deprivation amplifies reactivity of brain reward networks, biasing the appraisal of positive emotional experiences. *Journal of Neuroscience, 31*(12), 4466–4474.

Harley, R., Sprich, S., Safren, S., Jacobo, M., & Fava, M. (2008). Adaptation of dialectical behavior therapy skills training group for treatment-resistant depression. *Journal of Nervous and Mental Disease, 196*(2), 136–143.

Harned, M. S., Rizvi, S. L., & Linehan, M. M. (2010). Impact of co-occurring posttraumatic stress disorder on suicidal women with borderline personality disorder. *American Journal of Psychiatry, 167*(10), 1210–1217.

Harrison, A., Sullivan, S., Tchanturia, K., & Treasure, J. (2009). Emotion recognition and regulation in anorexia nervosa. *Clinical Psychology and Psychotherapy, 16*, 348–356.

Hayes, S. C., Bissett, R. T., Korn, Z., Zettle, R. D., Rosenfarb, I. S., Cooper, L. D., et al. (1999). The impact of acceptance versus control rationales on pain tolerance. *Psychologcial Reports, 49*, 33–47.

Hazlett, E. A., Zhanga, J., New, A. S., Zelmanovaa, Y., Goldstein, K. E. M., Haznedar, M. M., et al. (2012). Potentiated amygdala response to repeated emotional pictures in borderline personality disorder. *Biological Psychiatry, 72*(6), 448–456.

Heller, A. S., Johnstone, T., Shackman, A. J., Light, S. N., Peterson, M. J., Kolden, G. G., et al. (2009). Reduced capacity to sustain positive emotion in major depression reflects diminished maintenance of fronto-striatal brain activation. *Proceedings of the National Academy of Sciences USA, 106*, 22445–22450.

Hernandez, A., Arntz, A., Gaviria, A. M., Labad, A., & Gutiérrez-Zotes, J. A. (2012). Relationships between childhood maltreatment, parenting style, and borderline personality disorder criteria. *Journal of Personality Disorders, 26*(5), 727–736.

Herpertz, S. C., Dietrich, T. M., Wenning, B., Krings, T., Erberich, S. G., Willmes, K., et al. (2001). Evidence of abnormal amygdala functioning in borderline personality disorder: A functional MRI study. *Biological Psychiatry, 50*, 292–298.

Hupka, R. B., Lenton, A. P., & Hutchinson, K. A. (1999). Universal development of emotion categories in natural language. *Journal of Personality and Social Psychology, 77*, 247–278.

Hurwitz, B. E., & Furedy, J. J. (1986). The human dive reflex—an experimental, topographical and physiological analysis. *Physiology and Behavior, 36*, 287–294.

Iverson, K. M., Shenk, C., & Fruzzetti, A. E. (2009). Dialectical behavior therapy for women victims of domestic abuse: A pilot study. *Professional Psychology: Research and Practice, 40*, 242–248.

Izard, C. E. (1977). Distress—Anguish, grief, and depression. In C. Izard (Ed.), *Human emotions* (pp. 285–288). New York: Plenum Press.

Jacob, G. A., Guenzler, C., Zimmermann, S., Scheel, C. N., Rüsch, N., Leonhart, R., et al. (2008). Time course of anger and other emotions in women with borderline personality disorder: A preliminary study. *Journal of Behavior Therapy and Experimental Psychiatry, 39*(3), 391–402.

Joiner, T. E., Jr., Lewinsohn, P. M., & Seeley, J. R. (2002). The core of loneliness: Lack of pleasurable engagement—more so than painful disconnection—predicts social impairment, depression onset, and recovery from depressive disorders among adolescents. *Journal of Personality Assessment, 79*, 472–491.

Koerner, K., & Linehan, M. M. (1997). Case formulation in dialectical behavior therapy for borderline personality disorder. In T. Eells (Ed.), *Handbook of psychotherapy case formulation* (pp. 340–367). New York: Guilford Press.

Koole, S. (2009). The psychology of emotion regulation: An integrative review. *Cognition and Emotion, 23*, 4–41.

Koons, C. R., Chapman, A. L., Betts, B. B., O'Rourke, B., Morse, N., & Robins, C. J. (2006). Dialectical behavior therapy adapted

for the vocational rehabilitation of significantly disabled mentally ill adults. *Cognitive and Behavioral Practice, 13*, 146–156.

Korfine, L., & Hooley, J. M. (2000). Directed forgetting of emotional stimuli in borderline personality disorder. *Journal of Abnormal Psychology, 109*(2), 214–221.

Kring, A. M., & Sloan, D. M. (2010). *Emotion regulation and psychopathology: A transdiagnostic approach to etiology and treatment.* New York: Guilford Press.

Kring, A. M., & Werner, K. H. (2004). Emotion regulation and psychopathology. In P. Philippot & R. S. Feldman (Eds.), *The regulation of emotion* (pp. 359–408). Mahwah, NJ: Erlbaum.

Kuo, J. R., & Linehan, M. M. (2009). Disentangling emotion processes in borderline personality disorder: Physiological and self-reported assessment of biological vulnerability, baseline intensity, and reactivity to emotionally evocative stimuli. *Journal of Abnormal Psychology, 118*(3), 531–544.

Laird, J. D. (1974). Self-attribution of emotion: The effects of expressive behavior on the quality of emotional experience. *Journal of Personality and Social Psychology, 29*, 475–486.

Lazarus, A. A., & Abramovitz, A. (1962). The use of "emotive imagery" in the treatment of children's phobias. *Journal of Mental Science, 108*, 191–195.

Levine, D., Marziali, E., & Hood, J. (1997). Emotion processing in borderline personality disorders. *Journal of Nervous and Mental Disease, 185*(4), 240–246.

Levitt, J. T., Brown, T. A., Orsillo, S. M., & Barlow, D. H. (2004). The effects of acceptance versus suppression of emotion on subjective and psychophysiological response to carbon dioxide challenge in patients with panic disorder. *Behavior Therapy, 25*, 747–766.

Linehan, M. M. (1993a). *Skills training manual for treating borderline personality disorder.* New York: Guilford Press.

Linehan, M. M. (1993b). *Cognitive-behavioral treatment of borderline personality disorder.* New York: Guilford Press.

Linehan, M. M. (in press). *Skills training for dialectical behavior therapy.* New York: Guilford Press.

Lobbestael, J., & Arntz, A. (2010). Emotional, cognitive and physiological correlates of abuse-related stress in borderline and antisocial personality disorder. *Behaviour Research and Therapy, 48*(2), 116–124.

Lynch, T. R., Chapman, A. L., Rosenthal, M. Z., Kuo, J. R., & Linehan, M. M. (2006) Mechanisms of change in dialectical behavior therapy: Theoretical and empirical observations. *Journal of Clinical Psychology, 62*, 459–480.

Lynch, T. R., Rosenthal, M. Z., Kosson, D., Cheavens, J. S., Lejuez, C. W., & Blair, R. J. R. (2006). Heightened sensitivity to facial expressions of emotion in borderline personality disorder. *Emotion, 6*, 647–655.

Marissen, M. E., Meuleman, L., & Franken, I. A. (2010). Altered emotional information processing in borderline personality disorder: An electrophysiological study. *Psychiatry Research: Neuroimaging, 181*(3), 226–232.

Martell, C. R., Addis, M., & Jacobson, N. S. (2001). *Depression in context: Strategies for guided action.* New York: Norton.

Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2005). Preliminary evidence for an emotion dysregulation model of generalized anxiety disorder. *Behaviour Research and Therapy, 43*, 1281–1310.

Minzenberg, M. J., Fan, J., New, A. S., Tang, C. Y., & Siever, L. J. (2007). Fronto-limbic dysfunction in response to facial emotion in borderline personality disorder: An event-related fMRI study. *Psychiatry Research, 155*(3), 231–243.

Minzenberg, M. J., Fan, J., New, A. S., Tang, C. Y., & Siever, L. J. (2008). Frontolimbic structural changes in borderline personality disorder. *Journal of Psychiatric Research, 42*, 727–733.

Monti, J. M., & Monti, D. (2000). Sleep disturbance in generalized anxiety disorder and its treatment. *Sleep Medicine Reviews, 4*, 263–276.

Neacsiu, A. D., Rizvi, S. L., & Linehan, M. M. (2010). Dialectical behavior therapy skills use as a mediator and outcome of treatment for borderline personality disorder. *Behaviour Research and Therapy, 48*(9), 832–839.

Neacsiu, A. D., & Linehan, M. M. (in press). Dialectical behavior therapy for borderline personality disorder. In D. Barlow (Ed.), *Clinical handbook of psychological disorders* (5th ed.). New York: Guilford Press.

New, A. S., Hazlett, E. A., Buchsbaum, M. S., Goodman, M., Mitelman, S. A., Newmark, R., et al. (2007). Amygdala–prefrontal disconnection in borderline personality disorder. *Neuropsychopharmacology, 32*, 1629–1640.

Niedtfeld, I., Kirsch, P., Schulze, L., Herpertz, S. C., Bohus, M., & Schmahl, C. (2012). Func-

tional connectivity of pain mediated affect regulation in borderline personality disorder. *PLoS ONE, 7*(3), e33293.

Nunes, P. M., Wenzel, A., Borges, K. T., Porto, C. R., Caminha, R. M., & de Oliveira., I. R. (2009). Volumes of the hippocampus and amygdala in patients with borderline personality disorder: A meta-analysis. *Journal of Personality Disorders, 23*, 333–345.

Philippot, P., Baeyenes, C., Douilliez, C., & Francart, B. (2004). Cognitive regulation of emotion: Application to clinical disorders. In P. Philippot & R. S. Feldman (Eds.), *The regulation of emotion* (pp. 71–100). New York: Erlbaum.

Philippot, P., Burgos, A. I., Verhasselt, S., & Baeyens, C. (2002). *Specifying emotional information: Modulation of emotional intensity via executive processes*. Paper presented at the 2002 International Society for Research on Emotion Conference, La Cuenca, Spain.

Pryce, C. R., & Feldon, J. (2003). Long-term neurobehavioural impact of the postnatal environment in rats: Manipulations, effects and mediating mechanisms. *Neuroscience and Biobehavioral Review, 27*, 57–71.

Ray, W. J., Odenwald, M., Neuner, F., Schauer, M., Ruf, M., Wienbruch, C., et al. (2006). Decoupling neural networks from reality: Dissociative experiences in torture victims are reflected in abnormal brain waves in left frontal cortex. *Psychological Science, 17*(3), 825–829.

Reitz, S., Krause-Utz, A., Pogatzki-Zahn, E. M., Ebner-Priemer, U., Bohus, M., & Schmahl, C. (2012). Stress regulation and incision in borderline personality disorder—a pilot study modeling cutting behavior. *Journal of Personality Disorders, 26*(4), 605–615.

Rizvi, S. L., & Linehan, M. M. (2006). The treatment of maladaptive shame in borderline personality disorder: A pilot study of "Opposite Action." *Cognitive and Behavioral Practice, 12*, 437–447.

Rosenthal, M. Z., Gratz, K. L., Kosson, D. S., Cheavens, J. S., Lejuez, C. W., & Lynch, T. R. (2008). Borderline personality disorder and emotional responding: A review of the research literature. *Clinical Psychology Review, 28*, 75–91.

Ruocco, A. C. (2005). The neuropsychology of borderline personality disorder: A meta-analysis and review. *Psychiatry Resource, 137*(3), 191–202.

Schaefer, A., Collette, F., Philippot, P., van der, L. M., Laureys, S., Delfiore, G., et al. (2003). Neural correlates of "hot" and "cold" emotional processing: A multilevel approach to the functional anatomy of emotion. *NeuroImage, 18*, 938–949.

Schredl, M., Paul, F., Reinhard, I., Ebner-Priemer, U., Schmahl, C., & Bohus, M. (2012). Sleep and dreaming in patients with borderline personality disorder: A polysomnographic study. *Psychiatry Research, 200*(2–3), 430–436.

Schulze, L., Domes, G., Krüger, A., Berger, C., Fleischer, M., Prehn, K., et al. (2011). Neuronal correlates of cognitive reappraisal in borderline patients with affective instability. *Biological Psychiatry, 69*(6), 564–573.

Selby, E. A., & Joiner, T. E. (2009). Cascades of emotion: The emergence of borderline personality disorder from emotional and behavioral dysregulation. *Review of General Psychology, 13*, 219–229.

Shaver, P., Schwartz, J., Kirson, D., & O'Connor, C. (1987). Emotion knowledge: Further exploration of a prototype approach. *Journal of Personality and Social Psychology, 52*, 1061–1086.

Smith, C. F., Williamson, D. A., Bray, G. A., & Ryan, D. H. (1999). Flexible vs. rigid dieting strategies: Relationship with adverse behavioral outcomes. *Appetite, 32*, 295–305.

Soeteman, D. I., Hakkaart-van Roijen, L., Verheul, R., & Busschbach, J. J. (2008). The economic burden of personality disorders in mental health care. *Journal of Clinical Psychiatry, 69*(2), 259–265.

Soler, J., Pascual, J. C., Tiana, T., Cebria, A., Barrachina, J., Campins, M. J., et al. (2009). Dialectical behaviour therapy skills training compared to standard group therapy in borderline personality disorder: A 3-month randomised controlled clinical trial. *Behaviour Research and Therapy, 47*, 353–358.

Stepper, S., & Strack, F. (1993). Proprioceptive determinants of emotional and nonemotional feelings. *Journal of Personality and Social Psychology, 64*, 211–220.

Stiglmayr, C. E., Shapiro, D. A., Stieglitz, R. D., Limberger, M. F., & Bohus, M. (2001). Experience of aversive tension and dissociation in female patients with borderline personality disorder—a controlled study. *Journal Psychiatric Research, 35*, 111–118.

Tafrate, R. C., Kassinove, H., & Dundin, L. (2002). Anger episodes in high- and low-trait-anger community adults. *Journal of Clinical Psychology, 58*, 1573–1590.

Teasdale, J. D., Segal, Z., & Williams, J. M. (1995). How does cognitive therapy prevent

depressive relapse and why should attentional control (mindfulness) training help? *Behaviour Research and Therapy, 33*, 25–39.

Tebartz van Elst, L., Hesslinger, B., Thiel, T., Geiger, E., Haegele, K., Lemieux, L., et al. (2003). Frontolimbic brain abnormalities in patients with borderline personality disorder: A volumetric magnetic resonance imaging study. *Biological Psychiatry, 54*, 163–171.

Thorberg, F. A., Young, R. M., Sullivan, K. A., & Lyvers, M. (2009). Alexithymia and alcohol use disorders: A critical review. *Addictive Behaviors, 34*, 237–245.

Tooby, J., & Cosmides, L. (1990). The past explains the present: Emotional adaptations and the structure of ancestral environment. *Ethology and Sociobiology, 11*, 375–424.

Trull, T. J., Jahng, S., Tomko, R. L., Wood, P. K., & Sher, K. J. (2010). Revised NESARC personality disorder diagnoses: Gender, prevalence, and comorbidity with substance dependence disorders. *Journal of Personality Disorders, 24*(4), 412–426.

Williams, J. B. W., Stiles, W. B., & Shapiro, D. A. (1999). Cognitive mechanisms in the avoidance of painful and dangerous thoughts: Elaborating the assimilation model. *Cognitive Therapy and Research, 23*, 285–306.

Wong, M. M., Brower, K. J., Fitzgerald, H. E., & Zucker, R. A. (2004). Sleep problems in early childhood and early onset of alcohol and other drug use in adolescence. *Alcoholism: Clinical and Experimental Research, 28*(4), 578–587.

CHAPTER 30

Regulation of Emotion through Modification of Attention

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Emotions tend to be triggered by situations and generally operate to facilitate adaptive functioning (Werner & Gross, 2010). However, the nature and intensity of emotional experience is not determined by situational factors alone. As clearly evidenced by the extensive work reviewed in this handbook, a range of psychological operations can moderate emotional responses to environmental circumstances. Collectively these have been termed *emotion regulation* processes. Gross and others have emphasized that emotion regulation results from a heterogeneous array of processes exerting their influence at differing points in the emotion generation continuum (Gross, 1998; Gross & Thompson, 2007). Antecedent-focused components operate prior to the generation of emotion, and response-focused components operate subsequent to emotion generation, while other emotion regulation processes exert a direct influence on emotion generation itself. The combined impact of these emotion regulation processes shapes the nature of emotional experience, with individual differences in emotional regulation giving rise to variation in emotional disposition. Thus, emotional resilience has been attributed to the effective use of adaptive emotion regulation (Troy & Mauss, 2011), while heightened vulnerability to negative emotions and to emotional pathology have been attributed to maladaptive emotional regulation (Moses & Barlow, 2006). Such accounts carry the implication that a better understanding of the specific processes that functionally contribute to emotion regulation, coupled with the ability to systematically influence these processes, could potentially enhance such regulation in ways that ameliorate emotional vulnerability and dysfunction.

Since variation in emotion regulation gives rise to individual differences in emotional disposition, it follows that information-processing biases theoretically implicated in the determination of emotional disposition represent plausible emotion regulation mechanisms. Numerous influential models have proposed that biased attentional selectivity contributes both to dispositional emotional vulnerability and emotional pathology (cf. Mathews & MacLeod, 2005). Hence, it is unsurprising that selective attention has been widely implicated in models of emotion regulation (Gross & Barrett, 2011). Kring and Werner (2004) contend that selective attentional deployment is a critical emotion regulation process that causally contributes both to variability in normal emotional experience and to emotional dysfunction. Thompson and Goodman (2010) echo this view, arguing that certain types of attentional selectivity contribute to good emotional regulation and therefore to emo-

tional resilience, while other patterns of attentional deployment compromise emotional regulation in ways that give rise to emotional pathology. Many other theorists have similarly proposed that attentional bias to negative information makes a maladaptive contribution to emotion regulation that elevates emotional vulnerability and the risk of emotional pathology (White, Helfinstein, Reeb-Sutherland, Degnan, & Fox, 2009), while attentional avoidance of negative information serves to enhance emotional resilience (Troy & Mauss, 2011).

This idea that selective attentional response to negative information makes a causal contribution to emotional regulation, and therefore functionally influences emotional vulnerability and dysfunction, is our focus in this chapter. Here we examine how use of cognitive technologies that have proven capable of systematically training attentional change has served to illuminate the causal role of selective attentional deployment in the regulation of emotion. We also review evidence that the enhancement of emotion regulation, through the application of such attentional bias modification procedures, can yield tangible benefits for people experiencing emotional difficulties. Before describing the attentional bias modification approach, we first briefly summarize the evidence that attentional preference for negative information represents a reliable characteristic of emotional vulnerability and dysfunction. This also enables us to communicate how the types of cognitive–experimental tasks previously employed to assess attentional bias recently have been adapted to create attentional bias modification procedures.

Assessing the Association between Attentional Bias and Emotion Regulation

Some investigators have sought to assess attention simply by asking people to introspectively reflect upon and self-report the manner in which their attention operates (e.g., Derryberry & Reed, 2002). However, the limitations of relying on verbal reports to assess cognitive processes have been well documented (cf. Nisbett & Wilson, 1977). In response to these limitations, many researchers investigating whether individual differences in emotion regulation are systematically associated with variation in selective attentional responding to negative information have drawn upon cognitive–experimental methodologies, to appraise more objectively the attentional processes of interest (Mathews & MacLeod, 2005). The resulting assessment procedures infer attentional selectivity not from self-report measures but from performance measures, such as response latencies to make particular decisions, that necessarily will be influenced in particular ways by underlying patterns of attentional selectivity. Several such attentional assessment tasks have been widely used in studies designed to establish whether biased attention to negative information is characteristic of people who display evidence of poor emotion regulation. We now briefly describe these assessment techniques and illustrate how each has lent support to the veracity of this hypothesis.

One of the earliest methodologies developed to objectively assess the association between selective attentional responding to negative information and emotional vulnerability is the emotional Stroop task. In this assessment approach, the participant must rapidly name the color of emotionally toned words displayed in differing ink colors, while ignoring their semantic content. The degree to which color naming is disproportionately slow on negative words compared to neutral words is taken as a measure of attentional bias to negative information, as it suggests particular difficulty ignoring the negative content of these words. Individuals who display heightened emotional vulnerability, or who suffer from emotional dysfunction, commonly show this pattern of emotional Stroop performance (e.g., Williams, Mathews, & MacLeod, 1996), leading to the conclusion that variation in the capacity to regulate emotion is indeed related to differential attentional responding to negative information (Ashley & Swick, 2009). Another technique commonly used to assess the association between selective attentional response to negative information and individual difference in emotion regulation is the visual search task. In this approach, participants are exposed to an array of stimuli and are required to locate a specified target stimulus as quickly as possible. An index of

attentional bias to negative information is provided by their relative speeding to detect emotionally negative targets compared to neutral or positive targets. It has repeatedly been found that participants with heightened emotional vulnerability or dysfunction display greater evidence of such speeding (c.f. Cisler & Koster, 2010). Investigators employing this visual search approach have concluded that variation in attentional responding to emotional information is systematically related to variation in the ability to regulate emotion effectively (Leclerc & Kensinger, 2008).

Probably the most widely used method of assessing biased attention to negative information is the attentional probe task (MacLeod, Mathews, & Tata, 1986). In this task, the participant is briefly exposed to stimulus pairs whose members differ in emotional valence. These stimuli can be words or images, and most commonly one member of each pair is emotionally negative, while the other is neutral in emotional tone. Immediately following stimulus offset, a small visual probe is presented in the locus of one the previously displayed stimuli, and the participant is required to rapidly execute a discriminatory response to this probe. It is assumed that the probe will be processed fastest when it appears in the locus where participants already are attending. Hence, the degree to which this discrimination response is speeded when the probe appears in the locus of the negative information, rather than the locus of neutral information, provides a measure of attentional bias toward negative information. A reliable finding from studies using this task is that emotionally vulnerable individuals, and those suffering from emotional pathology, display an attentional bias to negative information (c.f. Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007). Once again, this has led theorists to conclude that an attentional bias to negative information is characteristic of deficient emotion regulation (e.g., Bradley, Mogg, White, Groom, & de Bono, 1999).

While the weight of evidence leaves little room for doubt that impaired emotion regulation, as evidenced by heightened emotional vulnerability or the presence of emotional pathology, is reliably characterized by an attentional bias favoring negative information, this does not permit the conclusion that such attentional deployment makes a direct functional contribution to emotion regulation. This association could instead reflect the influence of emotion regulation on attentional selectivity. Alternatively, it could be that attentional selectivity and emotion regulation represent the independent consequence of some third individual-difference variable. To determine whether selective attention exerts a causal influence on emotion regulation, it is necessary to systematically alter attentional selectivity. Adoption of the attentional bias modification approach has served both to confirm, and to capitalize on, the contribution of attentional selectivity to emotion regulation.

The Attentional Bias Modification Approach

The most powerful way to evaluate the hypothesis that patterns of selective attentional response to emotional information make a functional contribution to individual differences in emotion regulation is to systematically manipulate such attentional selectivity, in order to test the prediction that the modification of attentional bias will alter emotion regulation, as revealed by changes in emotional reactivity and dysfunction. Confirmation of this prediction would lend powerful support to the contention that attentional bias is causally involved in emotion regulation. It also would introduce the possibility that dysfunctional patterns of emotional symptomatology, resulting from deficiencies in emotion regulation, might be therapeutically attenuated by clinical interventions that appropriately exploit such attentional bias modification procedures.

A number of early studies designed to appraise the possibility that emotion regulation may be influenced by altering attentional selectivity have employed only verbal instruction as the intended method of attentional manipulation. Participants instructed to alter their attentional style not uncommonly report that this alters their emotional experience (Richards & Gross, 2006). However, while such findings are certainly encouraging, reliance on verbal instruction to modify attentional selectivity has a number of significant limitations

(MacLeod & Bucks, 2011). For one thing, such an approach at best could only influence attentional processes amenable to volitional control, and it is well established that the patterns of attentional bias that characterize emotional vulnerability and dysfunction commonly operate automatically within the cognitive system (Mathews & MacLeod, 2005). Relatedly, while participants may report that they comply with such attentional instructions, in the absence of objective attentional measures it is difficult to exclude the potential influence of demand effects. Hence, it would be highly desirable to induce attentional change in a manner that does not assume the targeted attentional process to be under voluntary control, and to objectively verify the efficacy of this attentional manipulation without relying on self-report measures of attentional selectivity.

Recent years have witnessed the development and implementation of new cognitive technologies designed to achieve these objectives by redesigning cognitive–experimental tasks previously employed to assess patterns of attentional bias relevant to emotion regulation, in ways that transform them into procedures capable of instead training change in such attentional selectivity (Mathews & MacLeod, 2002). Collectively referred to as cognitive bias modification for attention (CBM-A), or more simply as attentional bias modification (ABM) tasks, this general approach has involved introducing systematic contingencies into the original assessment tasks, such that task performance will be enhanced by the acquisition of a biased attentional response to a target category of emotional information. Following extensive practice in the execution of such tasks, subsequent performance on conventional attentional bias assessment procedures has served to confirm that these ABM procedures can systematically alter selective attentional responding to emotional information.

MacLeod, Rutherford, Campbell, Ebsworthy, and Holker (2002) developed the earliest and most widely used ABM task by transforming the attentional probe task into a training procedure. The assessment version of this task presents probes with equal frequency in the screen locations where either negative or neutral members of a stimulus word pair have just been presented. In their ABM version of the task, however, MacLeod et al. either presented all probes in the loci where the negative words appeared, in order to induce attentional vigilance for negative information (attend-negative training), or in the loci where the neutral words appeared, in order to induce attentional avoidance of negative information (avoid-negative training). Across several studies, MacLeod and colleagues showed that following the delivery of 576 such ABM trials, participants who received these alternative training conditions came to differ as intended in the pattern of attentional bias they subsequently displayed on the conventional assessment version of the probe task.

Dandeneau and Baldwin (2004) developed an ABM variant of the visual search procedure previously employed to assess patterns of attentional selectivity associated with individual differences in emotion regulation. Their attentional training version was designed to enhance attentional vigilance for positive information, while inducing attentional avoidance of negative information by having participants repeatedly search for a single emotionally positive target image (a smiling face) in a matrix of emotionally negative distracter images (frowning faces). Following exposure to 112 trials of this ABM procedure, participants evidenced a change in attentional bias consistent with the training objective, as assessed using the emotional Stroop task. Once again, this effect has been replicated across subsequent studies (Dandeneau, Baldwin, Baccus, Sakellaropoulo, & Pruessner, 2007).

Because the attentional impact of ABM procedures has been demonstrated using performance-based rather than self-report measures of attentional selectivity, there are good grounds for confidence that they induce genuine attentional change. Further support for the authenticity of their attentional impact comes from the breadth of attentional measures that are influenced by these bias modification procedures. Thus, for example, ABM tasks alter attentional selectivity as assessed using eye movement measures (Wadlinger & Isaacowitz, 2008), and they also modulate event-related potential (ERP) indices of attentional selectivity (Eldar & Bar-Haim, 2010). Using functional magnetic resonance imaging (fMRI), Browning, Holmes, Murphy, Goodwin, and

Harmer (2010) have demonstrated that the attentional change elicited by ABM procedures is mediated by altered patterns of activation in the lateral frontal cortex.

This powerful new capacity to directly modify selective attentional responding to emotional information has enabled researchers not only to evaluate the hypothesis that such attentional bias causally contributes to emotion regulation but also to exploit therapeutically the capacity of these ABM procedures to enhance emotion regulation (cf. Bar-Haim, 2010; Hertel & Mathews, 2011; MacLeod & Mathews, 2012). In reviewing this work, we focus first on laboratory-based research that has employed single-session ABM, with the principal aim of illuminating the functional nature of the association between attentional bias and emotion regulation. We next consider work that has delivered extended ABM, with the aim of enhancing emotion regulation in ways that can reduce emotional vulnerability and dysfunction in real-world settings.

Single-Session ABM Studies: Determining the Causal Contribution of Attentional Bias to Emotion Regulation

Studies employing single-session ABM procedures have examined the impact of transiently induced change in attentional responding to emotional information on emotional experience within controlled laboratory settings. In some cases, investigators have studied the effect of such ABM procedures on normal emotional reactions to contrived stress. In others, the focus has been on the capacity of ABM to attenuate dysfunctional emotional symptoms in participants selected because of their disposition to experience particular types of emotional difficulties, such as worry, obsessive concerns, or social anxiety. The following sections review the outcomes of these investigations.

Impact of Single-Session ABM on Affective Reactivity to Laboratory-Based Mood Induction

In two early studies, MacLeod et al. (2002) employed a single session of the probe-based ABM task to successfully induce a differential attentional response to negative verbal stimuli in students who obtained midrange scores on a measure of emotional vulnerability. These participants were subsequently exposed to a stressful anagram task, and their emotion regulation capability was determined by assessing the degree to which this elicited anxious and depressed mood. While the anagram stressor increased levels of anxiety and depression across all participants, the magnitude of this dysphoric emotional response was significantly attenuated for the participants who had just completed ABM in the avoid-negative rather than the attend-negative training condition. Furthermore, the degree to which the ABM procedure influenced the emotional impact of this stressor was a direct function of the degree to which it influenced selective attentional responding to negative stimuli. These findings confirm that selective attentional avoidance of negative information can enhance down-regulation of dysphoric responses to stressful experiences, which is a characteristic of good emotion regulation.

Using a single session of their visual search ABM variant, Dandeneau and Baldwin (2009) have provided further support for the idea that attentional selectivity causally influences emotion regulation. Unselected workers at an adult education center completed a single session of this ABM task either in a condition designed to induce attentional vigilance for socially positive information (smiling faces) and avoidance of socially negative information (frowning faces), or in a control condition designed to leave attentional selectivity unaltered. A subsequent attentional probe assessment task confirmed that participants given the ABM training, but not those receiving the control condition, came to display attentional avoidance of information related to social rejection. The former participants also evidenced enhanced emotion regulation when exposed to a variety of stressor tasks. For example, they felt less subjective rejection than did control participants following a simulated interaction in which they were treated coldly, and this was particularly evident in participants who initially had scored low in self-esteem. Participants who had received the ABM training also reported less rejection-related thoughts when attempting to complete a subsequent insoluble anagram task.

Furthermore, failure on this task exerted a less negative impact on self-esteem in participants who received the ABM than was the case for those in the control condition. Dandeneau and Baldwin reasonably concluded from these findings that selective attentional responding to emotional information makes a direct functional contribution to the self-regulation of emotional experience.

These aforementioned studies indicate that attentional bias can causally influence emotion regulation in participants whose emotional experiences fall within the normal range. Researchers have gone on to investigate whether a single session of ABM can transiently ameliorate dysfunctional emotion in participants selected on the basis of preexisting emotional problems.

Impact of Single-Session ABM on Negative Thought Intrusions in Worriers

Hayes, Hirsch, and Mathews (2010) recruited volunteers who defined themselves as high worriers, and who scored high on a conventional worry questionnaire. Half of these participants were given an ABM procedure that delivered the probe ABM task and a novel dichotic ABM task, both configured to induce attentional avoidance of negative information. The remaining participants were given control versions of these tasks without any training contingency. When attentional bias was subsequently measured, it was found that participants receiving ABM differed significantly from the control participants in terms of attentional bias, and now demonstrated the intended avoidance of negative information. A thought-sampling procedure was then employed to assess negative thought intrusions before and after a final worry induction. Although there was no significant difference between the groups in terms of negative thought intrusions before the worry induction, the control participants, but not the experimental participants, evidenced an increase in such intrusions following this worry manipulation. The finding that participants who received avoid-negative ABM experienced less negative thought intrusions than did those who received the no-training control procedure verifies that attentional bias can make a casual contribution to worry.

In an extension of this work, using worriers selected in the same manner, Hirsch et al. (2011) compared the impact of two variants of ABM. Both employed verbal stimuli, but one was designed to modify the degree to which attention is captured by negative information, while the other was designed to instead modify the degree to which attention is held by negative information. These two procedures altered attentional selectivity to an equivalent degree, but only the former ABM variant influenced subsequently assessed negative thought intrusions. On the basis of these findings, Hirsch et al. argue that heightened attentional engagement with negative information may contribute more than impaired attentional disengagement from such information to the evocation of worry-related negative thought intrusion. This study illustrates the capacity for carefully designed implementations of the ABM approach to illuminate which aspects of attentional bias make the greatest contributions to dysfunctional symptoms reflecting impaired emotion regulation.

Impact of Single-Session ABM on Behavioral Avoidance in Subclinical Obsessive–Compulsive Disorder

The studies reported so far have assessed the impact of ABM only on self-report measures of emotion. However, the modification of attentional bias to negative information also has been shown to influence behavioral indices of dysfunctional emotion. Najmi and Amir (2010) recruited individuals who reported excessive concern about germs, dirt, or contamination, and scored high on a conventional measure of obsessive–compulsive symptomatology. Half these participants received a single session of probe-based ABM, delivered in the avoid-negative training condition, while the other half received this task in a control condition that did not include any training contingencies. The emotionally negative information presented in these tasks was words chosen on the basis of their relevance to contamination-related concerns. As intended, the former group, but not the latter, came to exhibit attentional avoidance of this contamination-related negative information. A behavioral approach task was then given, which required participants to take as many steps as possible

toward a contamination-related stimulus (e.g., potting soil mixed with cat hair and dead crickets). Those who had received the avoid-negative ABM training exhibited greater levels of behavioral approach than did those who instead received the control condition. Moreover, Najmi and Amir were able to show that the degree to which this ABM training increased behavioral approach was mediated by the degree to which it reduced attentional bias to the negative information. Thus, selective attentional responding to negative information makes a contribution to not only the regulation of emotion itself but also the regulation of emotionally relevant patterns of behavior.

Impact of Single-Session ABM on Affective, Somatic, and Behavioral Symptoms of Social Anxiety

Amir, Weber, Beard, Bomyea, and Taylor (2008) have shown that ABM also influences emotionally relevant patterns of behavior in socially anxious people with a fear of public speaking. Included in the study were individuals who responded to an advertisement seeking volunteers who had difficulty giving speeches, and who scored high on a questionnaire measure of social anxiety. They were exposed to a single-session attentional probe procedure that presented faces displaying either negative emotion (disgust) or neutral expressions, delivered in either the avoid-negative ABM condition or a no-training control condition. As intended, the former participants alone displayed a significant reduction in attention to the socially relevant negative information, coming to show less attention to such negative information than did control participants. When subsequently required to give a short speech, the participants who had received avoid-negative ABM were rated as performing better than control participants. They also reported lower levels of anxiety during speech presentation than did control participants. The superior speech performance exhibited by the participants who received avoid-negative ABM was statistically mediated by the emotional impact of this ABM procedure. This suggests that the induced attentional avoidance of negative information adaptively regulated emotional response to the speech stressor, with beneficial behavioral consequences for speech performance.

Using a somewhat more complex design, which involved having participants engage in physical exercise during the ABM sessions, Julian, Beard, Schmidt, Powers, and Smits (2012) recently failed to replicate Amir et al.'s (2008) finding that ABM designed to reduce attention to negative information served to attenuate emotional reactivity to a subsequent speech stressor. However, it should be emphasized that Julian et al.'s (2012) ABM procedure actually failed to modify selective attentional responding to negative information. Hence, the results of this study do not challenge the hypothesis that selective attentional deployment influences emotion regulation. Other investigators who have succeeded in modifying this attentional bias have been able to verify Amir et al.'s (2008) original conclusion that such attentional bias modification influences both subjective and behavioral measures of socially anxious participants' stress response to a speech task. Heeren, Lievens, and Philippot et al. (2011) delivered a range of attentional training variants to socially anxious participants and found that an ABM procedure designed specifically to increase attentional disengagement from faces displaying negative expressions was most effective in enhancing independently rated speech performance, while also reducing self-reported anxiety. Thus, the pattern of attentional selectivity that most contributes to the adaptive regulation of social anxiety may involve the ready disengagement of attention from socially relevant negative information.

The weight of evidence from single-session ABM studies firmly favors the conclusion that selective attentional responding to emotional information can make a causal contribution to emotion regulation. Single sessions of ABM can moderate the intensity of normal affective reactions to laboratory stressors, and attenuate dysfunctional emotional symptoms such as excessive worry, elevated social anxiety, and undue concerns over contamination within the laboratory setting. Such findings offer empirical support to the hypothesis that attentional selectivity contributes to the regulation of emotion. They also raise the prospect that appropriately designed extensions of single-session ABM procedures may be capable of enhanc-

ing emotion regulation within real-world settings in ways that may deliver practical therapeutic benefits. In the next section, we consider ABM studies that have directly evaluated this exciting possibility.

Extended-Delivery ABM Studies: The Therapeutic Enhancement of Emotion Regulation in Real-World Settings

While the results of single-session ABM studies have confirmed that selective attentional responding to emotional information can influence emotion regulation within the laboratory, this does not mean that attentional bias necessarily contributes to real-world emotion regulation. Nor do these finding permit the conclusion that ABM has the capacity to enhance emotion regulation in ways that can reduce dysfunctional emotional experience outside the experimental setting. Both of these issues have been addressed, however, by studies designed to evaluate whether repeated exposure to ABM procedures can enhance regulation of emotional reactions to naturalistic stressors, and can improve emotion regulation to an extent that ameliorates the clinical symptoms of emotional pathology in the real-world.

Impact of Extended ABM on Affective Reactivity to Real-Life Stressors

See, MacLeod, and Bridle (2009) employed an online procedure to deliver 10-minute sessions of a probe-based ABM task on each of 15 consecutive days, to high school graduates approaching the stressor of migrating overseas to commence tertiary education. Half received this task in the avoid-negative training condition, while the other half received it in a control condition containing no attentional training contingency. The former participants, unlike the latter, displayed a reduction in attention to negative information across the duration of the study. Emotional measures taken immediately after participants subsequently experienced the stress of migration were compared with measures taken prior to the commencement of the study. As anticipated, this real-world stress event substantially increased state anxiety. However, the magnitude of this negative emotional reaction was attenuated in participants who had received the avoid-negative ABM, compared to control participants. Furthermore, a measure of trait anxiety, reflecting general disposition to experience anxiety symptoms, also was attenuated by the ABM procedure. The degree to which ABM reduced attention to negative information statistically mediated the degree to which it reduced trait anxiety, which in turn mediated its impact on emotional reactivity to the stressful transition event. Thus, the reduction of attention to negative information through extended exposure to ABM served to enhance the regulation of emotion within the naturalistic setting in ways that decreased negative emotional reactivity to a real-world stressor.

Similar conclusions are invited by the findings of Dandeneau et al. (2007), who examined the impact of 5 consecutive days of their visual search ABM task on self-report and physiological indices of negative emotional experience in participants who experienced stressful real-world situations. Compared to those who received a no-training control procedure, students who completed this extended ABM in the condition designed to inhibit attention to negative and increase attention to positive information reacted to a subsequent school examination with lower levels of subjective stress and anxiety. When telemarketers, employed within a stressful work environment, received this 5-day ABM procedure, they reported an increase in self-esteem and decrease in subjective stress, compared to colleagues given a no-training control procedure. They also evidenced a reduction in physiological signs of stress, were rated as more confident by their supervisors, and even showed higher levels of sales performance. Such findings strongly support the involvement of attentional selectivity in real-world emotion regulation and demonstrate that attentional bias modification procedures can enhance the regulation of normal emotional experience in ways that benefit people in stressful real-world situations.

Impact of Extended ABM on Pathological Worry

The extended delivery of ABM also has been shown to attenuate dysfunctional emo-

tional symptoms experienced outside the laboratory by people who report worrying excessively. Hazen, Vasey, and Schmidt (2009) recruited participants who exhibited extreme levels of worry, and across a 2-week period completed five sessions, each lasting for 30 minutes, of either probe-based ABM in the avoid-negative condition or a control task that contained no training contingency. The attentional vigilance for negative information initially shown by these worriers was reversed by the avoid-negative ABM. Participants who received this ABM, unlike control participants, also displayed a significant reduction in worry, anxiety, and depression. Hazen et al. contend, on the basis of their findings, that the enhancement of emotion regulation using attentional training may make a therapeutic contribution to the treatment of generalized anxiety disorder (GAD), in which pathological worry is the hallmark symptom.

Subsequent research has lent empirical weight to this contention. Amir, Beard, Burns, and Bomyea (2009) directly assessed whether ABM produced clinically significant benefits when given to treatment-seeking individuals meeting diagnostic criteria for GAD. All patients completed an attentional probe task twice weekly across a 4-week period. For one group, this task was given in the avoid-negative ABM condition, configured to induce attentional avoidance of negative information, while another group received a control condition that contained no training contingency. Clinical symptomatology was assessed before and after this 4-week period, using both questionnaire measures and diagnostic interview. Patients who received avoid-negative ABM procedure displayed the expected decrease in attention to negative information, which was not shown by patients in the control condition, resulting, as intended, in a post-training group difference in selective attentional responding to negative information. Across the 4 weeks of the study, patients in the control condition evidenced no decline in either questionnaire measures or interview-based measures of symptomatology that assessed worry, anxiety, and depression. In contrast, patients who received the avoid-negative ABM procedure demonstrated reliable reductions in all these symptom measures. The clinical significance of the change was such that, after this 4-week period, only 50% of the participants who had received avoid-negative ABM continued to meet diagnostic criteria for GAD, compared with 87% of those in the control condition. Amir et al. concluded that biased attentional responding to negative information functionally contributes to the emotional pathology observed in GAD. Moreover, on the basis of their findings, they advocate the use of ABM as a clinical tool in the treatment of this emotional disorder.

Impact of Extended ABM on Social Anxiety Dysfunction

There is compelling evidence that extended exposure to ABM can enhance emotional functioning in people experiencing problematic levels of social anxiety. After recruiting participants who scored high on a questionnaire measure of social anxiety, Li, Tan, Qian, and Liu (2008) gave them seven consecutive daily sessions that presented either probe-based ABM in the avoid-negative condition, or a control version of this procedure that contained no training condition. Only those who received the avoid-negative ABM came to display reduced attention to negative information, and only they evidenced a significant reduction in Social Interaction Anxiety Scale (SIAS) scores. Li et al.'s findings suggest that the regulation of real-world social anxiety can be improved by the modification of attentional bias in nonclinical participants. Later work carried out with clinical samples indicates that such ABM procedures also can therapeutically alleviate the clinical symptoms of patients with psychological disorders that involve excessive levels of social anxiety.

Amir, Beard, Taylor, et al. (2009) examined whether ABM could enhance emotional functioning in patients meeting diagnostic criteria for generalized social phobia (GSP). Patients who received eight sessions of avoid-negative ABM across a 4-week period evidenced reduced levels of attention to socially relevant negative facial expressions, which was not the case for those who instead received a no-training control version of the procedure. Over these 4 weeks, the former patients, relative to the latter, also exhibited disproportionate reductions across all measures of social anxiety, which

included the Liebowitz Social Anxiety Scale (LSAS), the Social Phobia and Anxiety Inventory (SPAI) and the Sheehan Disability Scale (SDS). At the end of this period, only 50% of the individuals who had received the avoid-negative ABM training continued to meet diagnostic criteria, compared to 86% of those who received the control condition. This improvement in social anxiety symptoms experienced by the patients given ABM was maintained at 4-month follow-up.

Carlbring et al. (2012) recently failed to replicate Amir, Beard, Taylor, et al. (2009) findings when delivering ABM online to Internet-recruited volunteers who met diagnostic criteria for social anxiety disorder (SAD). However, it is important to note that this intended ABM procedure failed to induce any reduction in selective attention to negative information. Hence, while Carlbring et al.'s study raises questions concerning the most effective methods of bringing about attentional change, its negative findings do not challenge Amir, Beard, Taylor, et al.'s (2009) conclusion that attentional bias causally contributes to impaired emotion regulation in SAD. Moreover, converging support for the conclusion that ABM can alleviate such clinical dysfunction has been independently obtained by other investigators. For example, Schmidt, Richey, Buckner, and Timpano (2009) evaluated the impact of avoid-negative ABM on the clinical symptoms of patients diagnosed with generalized social anxiety disorder (GSAD), using the same experimental design as Amir, Beard, Taylor, et al. (2009). Patients who received eight such ABM sessions across a 4-week period demonstrated reduced levels of emotional dysfunction compared to participants who received the no-training control condition on all measures of social anxiety, which included the SPAI, the LSAS, and the Brief Social Phobia Scale. At the end of this 4-week period only 28% of those who received the avoid-negative ABM continued to meet diagnostic criteria for GSAD, compared to 75% of participants in the control condition. Again, these gains were largely maintained at 4-month follow-up.

Heeren, Reese, McNally, and Philippot (2012) have shown that extended exposure to avoid-negative ABM reduces the negative emotional symptoms experienced by patients with social anxiety dysfunction in naturalistic environments and enhances their capacity to regulate their emotional reaction to experimentally delivered social stress. Patients diagnosed with GSAD were given four daily sessions of ABM training in either the avoid-negative condition or a no-training control version of the task. Attentional bias to negative information was significantly attenuated at a posttraining assessment point for the former participants relative to the latter. Participants receiving ABM also exhibited a disproportionate decline in social anxiety, as assessed by the LSAS and the Fear of Negative Evaluation Scale (FNE). Prior to, and after, the 4 days of attentional training participants were exposed to a speech task, in which they delivered a 2-minute oral presentation, and the impact of this stressor on self-report, behavioral, and physiological measures of emotion was assessed. Participants who received the avoid-negative ABM displayed a disproportionate reduction, relative to control participants, in the degree to which this stressor evoked negative emotion at the posttraining assessment point. Those who had received ABM reported experiencing less distress than did control participants during the speech task. They also performed the task with less evidence of negative emotion, as assessed by behavioral ratings and galvanic skin response (GSR).

Impact of Extended ABM on Depression

An attentional bias to negative information is generally considered a more robust characteristic of anxiety than depression, raising uncertainty concerning whether such attentional selectivity contributes to the regulation of depression. Nevertheless, a few ABM studies suggest that this is the case, at least when depression is not severe. For example, Wells and Beevers (2010) recruited undergraduate students whose questionnaire scores indicated mild to moderate levels of depression. Across a 2-week period they received four sessions of either probe-based ABM in the avoid-negative training condition or a control probe task that contained no training contingency. Across this period, the former participants alone displayed a reduction in attention to negative information, and by the end of the 2 weeks they were displaying significantly lower levels

of attention to negative information than was the case for control participants. Likewise, only the participants who received the avoid-negative ABM showed a reduction in depressive symptoms across these 2 weeks, and at the end of the period their depression scores were significantly lower than those of control participants. This ABM-induced difference in depression levels remained evident at a follow-up assessment 2 weeks later. The influence of the training procedure on depressive symptomatology was statistically mediated by its impact on attentional bias.

These findings suggest that attentional selectivity causally contributes to the regulation of depression, as well as anxiety, raising the possibility that ABM may potentially yield benefits in the treatment of depressive dysfunction. It would be premature, however, to draw conclusions concerning the responsiveness of clinical depression to this type of attentional manipulation. Baert, De Raedt, Schacht, and Koster (2010) reported finding no therapeutic impact of a novel probe-based ABM procedure they delivered, across 10 daily sessions, to students reporting severe depression, and to patients receiving concurrent conventional treatment for clinical depression. It must be noted, however, that the probe-based procedure employed by these researchers not only differed in significant ways from the ABM task adopted by most other researchers (e.g., presenting only single stimuli rather than stimulus pairs) but also prove ineffective in altering selective attentional responding to negative emotional information. We concur with the authors that their failure to induce differential attentional selectivity using this intended bias modification procedure begs a cautious interpretation of the accompanying failure to influence emotional symptomatology in severely depressed students and clinically depressed patients. It remains to be seen whether the successful reduction of attention to negative information can serve to therapeutically attenuate clinically significant depression.

Impact of Extended ABM on Dysfunctional Affect in Children and Adolescents

Across recent years, growing concern over emotional dysfunction in childhood and adolescence has led researchers to recognize the importance of better understanding the factors that contribute to emotion regulation in young people and identifying ways of enhancing this. ABM studies suggest that selective attentional responding to negative information causally influences emotion regulation in children as well as adults, and provide grounds for optimism that ABM procedures can be used to enhance emotion regulation in younger populations. Early support for the hypothesis that attentional selectivity contributes to emotion regulation in children was provided by Eldar, Ricon, and Bar-Haim (2008). These researchers' recruited children ages 7–12 years, whose questionnaire scores placed them midrange in terms of emotional vulnerability. Across two sessions, these children completed a probe-based ABM procedure in either an avoid-negative or an attend-negative training condition. Following this, they were exposed to a stressor, which involved their being videotaped while trying to solve a difficult puzzle. Children who had been given the differing ABM procedures exhibited discrepant emotional reactivity to this stressor. Those who had received the attend-negative ABM experienced a pronounced elevation of negative emotion following exposure to the stressor, relative to a prestressor baseline mood measure. In contrast, children who had received the avoid-negative ABM showed no elevation of negative emotion in response to the stressor. Thus, altering attentional responding to negative information influenced the regulation of the emotional reactions to stress exhibited by the children. In addition to confirming the role of attentional selectivity in childhood emotion regulation, these findings highlight the potential value of ABM as a technique for enhancing such regulation in children with dysfunctional emotional symptomatology.

More recently, two published randomized controlled trials further strengthen the conclusion that improving emotion regulation through direct modification of attentional selectivity can therapeutically alleviate dysfunctional emotional experience in anxious children. The first of these was carried out by Bar-Haim, Morag, and Glickman (2011) on a sample of 10-year-old children who scored high on a questionnaire instrument screening for childhood emotional disor-

ders. These children were randomly assigned to receive either a probe-based ABM avoid-threat training, or a control version of the task containing no training contingency, on two occasions separated by 4–6 days. Pre-training attentional bias was assessed prior to the first ABM session, while posttraining attentional bias was assessed 5–8 days after the second ABM session. At this latter assessment point, participants were exposed to the puzzle stressor employed by Eldar et al. (2008) to ascertain whether ABM influenced regulation of their emotional reactivity to stress. The avoid-negative ABM training changed attentional selectivity as intended, with the children who received this training displaying reduced attention to negative information compared to control children. It also enhanced emotion regulation during performance of the stressor task. While children in the control condition displayed a significant elevation of negative emotion in response to the final stressor, no elevation of negative affect was elicited by this stressor in children who had undergone avoid-negative ABM training. Hence, despite their initially high levels of emotional vulnerability, these children became more resilient to stress when they acquired the ability to attentionally avoid-negative information, indicating that this style of attentional selectivity improves emotion regulation in younger populations also.

Eldar et al. (2012) recently conducted the first randomized controlled trial evaluating the impact of ABM on children with formally diagnosed pediatric emotional disorders. Their sample comprised treatment-seeking children, ages 8–14 years, recruited from a hospital-based child anxiety clinic. All met diagnostic criteria for at least one anxiety disorder (i.e., separation anxiety disorder, social phobia, specific phobia, or GAD), with 75% meeting diagnostic criteria for more than one such disorder. These clinically anxious children received four weekly sessions of either probe-based ABM in the avoid-negative condition or a control version of the task containing no training contingency. Children who received the avoid-negative ABM, but not those who received the control condition, demonstrated a significant decline in both the number of anxiety symptoms recorded at clinical interview and the rated severity of these symptoms. Well over twice as many of children who received avoid-negative ABM, as those who received the control condition, showed full remission. Specifically, 33.3% no longer met diagnostic criteria for any anxiety disorder after receiving the 4 weeks of avoid-negative ABM, compared to only 13.3% of those in the control condition. Hence, for children who suffer from emotional dysfunction, as for adults, modification of attentional selectivity to increase avoidance of negative information serves to improve emotion regulation, as evidenced by the significant reduction of dysfunctional emotional experience.

Future Directions for ABM Approaches to Emotion Regulation

The studies reviewed in this chapter have demonstrated that ABM procedures can serve to attenuate dysfunctional emotional symptoms. It may seem paradoxical that ABM-induced attentional avoidance of negative information can be therapeutically beneficial given that many models implicate avoidance in the etiology of psychopathology. However, such accounts often pertain to behavioral rather than to cognitive avoidance (see Barlow, 2002). Furthermore, with respect to cognitive avoidance, it is important to distinguish effortful attempts to suppress negative thoughts from the type of attentional avoidance induced by ABM. The former appears to be emotionally counterproductive, probably because effortful thought suppression commonly fails to produce the intended pattern of cognitive avoidance and can instead paradoxically increase the very thoughts one tries to suppress (Wegner, Schneider, Carter, & White, 1987). In contrast, ABM procedures designed to reduce attention to negative information without effort or intention have proven successful in inducing attentional avoidance of such information, with consequent emotional benefits.

The conclusion that ABM procedures can effectively modify attentional response to negative information and therapeutically alleviate dysfunctional emotion has been bolstered by the outcomes of recent meta-analyses. One such meta-analysis by Hallion and Ruscio (2011), which did not distinguish between bias modification procedures

designed to alter attentional vs. interpretive selectivity, revealed that cognitive bias modification exerts a medium-size effect on both cognitive bias (g = 0.49) and clinical symptomatology (g = 0.39). Other meta-analyses that have focused specifically on the modification of attentional bias (Hakamata et al., 2010; Beard, Sawyer, & Hofmann, 2012) have concluded that ABM exerts a large effect on attention bias (g = 1.06–1.15), and a medium-to-large effect on clinical symptomatology (g = 0.48–0.77). Meta-analysis also has identified some clinically relevant variables that may moderate the magnitude of both effects. For example, Beard et al. report that ABM alters attentional bias to the greatest degree in participants who show highest initial levels of negative emotional symptomatology. Bias modification appears to influence anxiety more than depression (Hallion & Ruscio, 2011). Hakamata et al. (2010) found that ABM has a greater impact on anxious disposition than on anxious mood state per se, and that this emotional impact is nominally greater in anxiety patients than in nonclinical participants. This overall pattern lends weight to the potential value of ABM in the treatment of emotional dysfunction, particularly in the case of anxiety disorders.

Having established that attentional avoidance of negative information can successfully be induced by ABM procedures, and serves to increase emotional resilience and alleviate dysfunction emotional experience, future research can now build on this firm foundation in a number of important ways. We close this chapter by considering several directions that we believe will prove to be particularly profitable.

Improving ABM Techniques to Better Enhance Emotion Regulation

The degree to which emotion is influenced by ABM procedures is largely determined by the extent of change in negative attentional bias that they induce (Hakamata et al., 2010). Therefore, an obvious way potentially to increase the beneficial impact of ABM techniques on emotion regulation will be to improve their capacity to modify selective attentional responding to negative information. This endeavor will likely involve the future development of new CBM procedures, in ways guided by deeper understanding of the mechanisms through which existing ABM tasks exert their influence. Additionally, the capacity of existing ABM tasks to modify attentional selectivity effectively may be further enhanced by refining the manner in which they are delivered. We briefly consider, in turn, each of these potential approaches to the future improvement of ABM techniques.

One important issue will be to establish whether ABM tasks exert a highly specific effect on attentional bias alone, or whether their emotional benefits reflect change in more general cognitive control systems, such as working memory. Consistent with this latter possibility, Hirsch, Hayes, and Mathews (2009) reported that their cognitive bias modification procedure not only reduced the selective process of negative information but also improved working memory. Klumpp and Amir (2010) argued, on the basis of their findings, that the impact of ABM tasks on attentional bias may be mediated by a general improvement in cognitive control. Whether any such training induces improvement in working memory, directly influences emotion regulation, or instead facilitates adaptive change in attentional bias, must be determined by future research. However, such considerations suggest that developing ABM tasks in ways that maximize their capacity to improve working memory, or perhaps supplementing the delivery of ABM tasks with training procedures designed to directly improve working memory (Owens, Koster, & Derakshan, 2013), may augment their capacity to enhance emotion regulation.

The refinement of ABM procedures also will likely benefit from better understanding of the particular juncture(s) in the emotion regulation continuum where ABM exerts its effect(s). Gross and Thompson (2007) propose that the influence of selective attentional deployment on emotion regulation occurs at the emotion generation stage. The finding that ABM influences emotional reactivity to stressors is consistent with the possibility that it affects the generation of negative emotion. However, ABM also may exert an influence subsequent to initial emotion generation, at the response-focused stage of emotional regulation, by operating to prolong or curtail negative emotional

experience. This, too, could contribute to its observed impact on emotional reactivity measures, which typically involve the assessment of emotional experience at least several minutes after commencing exposure to an emotion-generating stressor. Indeed, it may be that modifying certain aspects of attentional selectivity, such as initial attentional engagement with negative information, could influence the emotion-generation stage, while modifying other aspects of attentional selectivity, such as the degree to which negative information holds attention over time, instead could influence the longevity of generated emotions (Hirsch et al., 2011). Determining where alternative ABM variants exert their influence on emotion regulation will require fine-grained assessment of the temporal dynamics of the resulting change in emotional experience, and physiological measures such as GSR and heart rate variability may prove useful in this regard. Future research of this type could potentially lead to the development of new ABM batteries designed either to exert a broader influence across multiple stages of emotion regulation or selectively target particular stages of emotional regulation, perhaps in an individually tailored manner.

While the creation of new of attentional training procedures, guided by better understanding of the mechanisms through which ABM influences emotion regulation, will likely contribute to future progress, it also seems probable that the efficacy of existing ABM tasks could be improved by identifying the most effective delivery formats. Most obviously, increasing the number and length of ABM sessions may amplify the resulting attentional change, and it has been suggested that such change may be more enduring if ABM sessions are widely distributed across time rather than grouped in close temporal proximity (Hertel & Mathews, 2011). Another interesting possibility is that the impact of ABM procedures on attentional bias may be affected by nature of instructions given to participants. Researchers commonly have assumed that ABM alters attention implicitly, and so typically have not explicitly communicated either the training contingency or intention to participants (MacLeod & Mathews, 2012). Consistent with this assumption, participants often are unable to subsequently report the training contingency, or to identify the training condition to which they have been exposed (Beard, 2011). However, this represents only weak evidence that ABM influences attentional selectivity implicitly. Furthermore, even if attentional change is trained implicitly by existing ABM approaches, the possibility remains that explicit training may more effectively induce therapeutic change in attentional bias. Hence, we suggest that future investigators should empirically contrast the impact of implicit and explicit versions of ABM, which differ with respect to whether they provide participants training contingency information and directly instruct practice in the target pattern of attentional selectivity. Future research of this type will identify which approach yields the greatest emotional benefit, and in doing so will also illuminate whether implicit or explicit attentional change more strongly governs the impact of ABM on emotional regulation.

Enhancing the power of ABM procedures must involve not only increasing the size and longevity of the attentional effects they evoke but also ensuring good generalization of this training. Generalization is revealed by the transfer of training effects beyond the original training task. A distinction has been drawn between "far-transfer" and "near-transfer" where, respectively, training effects are observed under conditions that differ either greatly, or only slightly, from the initial training experience (Hertel & Mathews, 2011). Most ABM studies have confirmed near-transfer of training by showing that the attentional impact of ABM can be detected on new stimulus materials not employed in the training task (e.g., MacLeod et al., 2002; Hirsch et al., 2011). However, the practical value of ABM clearly depends on also ensuring the far-transfer of training. Given that the effect of ABM can be detected on emotional assessment tasks very different in nature from the original training procedures, it seems clear such far-transfer can occur (MacLeod & Mathews, 2012). Further evidence of far-transfer comes from studies demonstrating that ABM-induced training effects can be detected across quite different measures of attentional selectivity (e.g., Dandeneau & Baldwin, 2004), and also on measures of interpretive selectivity (e.g., White, Suway, Pine, Bar-Haim, & Fox,

2011). Nevertheless, occasional transfer failures (e.g., Bockstaele, Koster, Verschuere, Crombez, & De Houwer, 2012) underscore the importance of identifying how best to optimize far-transfer. It has been proposed that the prospect of ABM training generalizing to new contexts and to new tasks might best be ensured by delivering ABM training sessions across a variety of differing contexts, and varying the task procedures and parameters employed within them to induce attentional change (MacLeod, Koster, & Fox, 2009). As yet, this proposal has not been formally tested, so it remains to be seen whether these innovations would indeed increase such generalization.

While the purpose of ABM in clinical studies conducted to date has been to induce general attentional avoidance of negative information, such a pattern of selectivity may not be optimal across all contexts. For example, there will likely be advantages associated with attending to threat cues in situations where the risk of genuine danger is high. Maximal well-being may result from attentional vigilance to negative information in high-risk contexts, and attentional avoidance of such information in low-risk contexts. It would therefore be appropriate for future researchers to investigate whether ABM can be delivered in ways that induce situation-specific patterns of attentional selectivity, such that the optimal attentional response to negative information becomes operational within each of these differing types of context. While undoubtedly ambitious, the viability of this objective is supported by recent demonstrations that when alternative patterns of implicit processing each are trained within a differing situational context, reinstatement of either training context favors expression of the specific pattern of cognition originally trained within that same context (e.g., D'Angelo, Milliken, Jimenez, & Lupianez, 2013).

Extending the Application of ABM to Other Forms of Self-Regulation

Because this review describes research motivated by the hypothesis that attentional bias to negative information underpins emotional vulnerability and dysfunction, the focus has been on ABM procedures designed specifically to modify attentional response to negative information. However, the ABM approach could potentially be employed to enhance other forms of self-regulation that plausibly are affected by attentional selectivity. For example, the hypothesis that heightened positive affectivity reflects an attentional bias toward positive information (Taylor, Bomyea, & Amir, 2011), suggests that ABM designed to modify attentional responding to positive information could influence positive emotional reactivity to happy events. Grafton, Ang, and MacLeod (2012) recently confirmed this by demonstrating that participants exposed to an ABM procedure that increased attention to positive words, compared to those given an ABM procedure that reduced attention to such words, displayed disproportionately intense positive emotional reactions to a subsequent success experience.

The potential therapeutic benefits of ABM need not be restricted to the regulation of emotional experience. In many types of psychological dysfunction that involve biased patterns of attentional selectivity, the primary symptom that could benefit from better regulation is not emotion. There is growing evidence that ABM procedures may prove beneficial in enhancing these other clinically relevant aspects of self-regulation. For example, people with chronic pain syndrome display an attentional bias toward pain-related information, and theorists contend that this pattern of attentional selectivity makes a functional contribution to their heightened perceptions of pain (Eccleston & Crombez, 1999). A long-standing aim of psychological interventions for this condition has been to enhance self-regulation of pain sensation. Recent work examining whether ABM can contribute to the regulation of pain experience has yielded promising outcomes. For example, Sharpe et al. (2010) gave participants either a conventional relaxation manipulation or a probe-based ABM procedure designed to induce attentional avoidance of pain-related information, before exposing them to a cold pressor task designed to induce painful sensation. Those who received the attentional training reported later onset of pain than did those given the relaxation procedure. Such findings should encourage future research-

ers to investigate whether the use of ABM to directly manipulate selective attentional responding to pain-relevant information can enhance the regulation of pain sensation in ways that yield therapeutic benefits for people who suffer from this clinical condition.

Another clinical disorder that involves impaired self-regulation, but in which negative emotional experience is incidental to the symptoms of primary concern, is substance dependency. Addictions are characterized by biased patterns of attentional selectivity that favor information related to the abused substance, and theorists contend that this bias contributes to associated craving and consumption (Wiers et al., 2007). This has led some investigators to examine whether ABM variants designed to reduce attention to such information can serve to attenuate craving and/or regulate consumption, and positive findings have been reported for both tobacco dependency and alcohol abuse. After exposure to a single-session of probe-based ABM, configured to either increase or decrease attention to smoking cues, Attwood, O'Sullivan, Leonards, Mackintosh, and Munafo (2008) found that male smokers in the latter group reported reduced craving for tobacco, and this reduction in craving was predicted by the magnitude of the induced attention change. Fadardi and Cox (2009) gave a sample of problem drinkers four weekly sessions of ABM, configured to reduce attention to alcohol-related information. This successfully modified attentional selectivity as intended and led also to a significant decline in alcohol consumption, which was maintained at 3-month follow-up.

Thus, it would appear that the modification of attentional bias carries the potential to enhance other forms of self-regulation, beyond the direct regulation of emotional experience, so future research could profitably seek to extend the application of the ABM approach across a wider range of such conditions. Of course, given that distressing conditions such as chronic pain and substance dependency contribute to negative emotional experience, ABM procedures that increase the capacity to regulate pain, addictive symptomatology, and other maladaptive aspects of experience and behavior also will likely indirectly benefit emotion regulation itself.

Refining the Clinical Utility of ABM in the Remediation of Dysfunctional Emotion Regulation

Given the wealth of evidence that supports the capacity of ABM approaches to influence emotion regulation beneficially, it is no surprise that investigators now are seeking to exploit its therapeutic potential in the treatment of emotional dysfunction and other psychological disorders that involve deficient self-regulation. This approach now has passed the proof-of-concept stage as a viable means of therapeutically attenuating clinically relevant symptoms, warranting its further development as a practical tool that can be employed in the clinical setting. Already, several small-scale studies have confirmed that ABM configured to induce attentional avoidance of negative information can be employed effectively within conventional psychology clinics to treat patients suffering from anxiety disorders and/or clinical depression (Brosan, Hoppitt, Shelfer, Sillence, & Mackintosh, 2011; Beard, Weisberg, & Amir, 2011).

The more widespread adoption of ABM within the clinical setting will partly depend upon patients' perceived acceptability of the approach. Hence, it is reassuring that patient feedback has generally been favorable. Following delivery of their 12-session ABM program to socially anxious 10- to 17-year-olds, Rozenman, Weersing, and Amir (2011) had recipients and their parents rate the acceptability of this intervention procedure. Responses ranged from *acceptable* to *excellent*, with mean participant and parent ratings of 4.2 and 4.5, respectively, on a 5-point scale. Beard (2011) likewise reported that primary care patients receiving ABM were generally quite satisfied with the procedure on average, rating its acceptability at level 3 on 4-point scale. Beard also noted that dropout rates from treatment trials delivering such bias modification have typically been low, ranging from 0 to 8%, suggesting that recipients find the procedures acceptable. Nevertheless, qualitative interviews carried out by Beard and her colleagues (2011) revealed that some recipients experience ABM as overly repetitive. Hence, one type of refinement to ABM that may increase patient acceptability could involve the intro-

duction of features that sustain interest and motivation. For example, this might involve delivering ABM in shorter blocks and providing performance feedback, so that participants can strive to improve across blocks. Beard (2011) also reports that some patients express a desire to better understand how ABM could help with their emotional difficulties. Therefore, another refinement that might enhance patient acceptance would be provision of a rationale that communicates how this procedure can contribute to the regulation of negative emotion.

One feature of ABM that Beard's interview data (2011) identified as being particularly highly valued by patients is the flexibility and convenience with which it can be accessed by recipients, due to its computer-based method of delivery. Recent years have witnessed the growing use of computer technology to facilitate the transport of conventional psychological interventions into the community (Marks & Cavanagh, 2009). Given its amenability to Internet delivery (e.g., See et al., 2009), ABM could readily be integrated into such forms of intervention. Remotely accessed computerized treatments that include ABM components already are beginning emerge. For example, Amir and Taylor (2012) recently evaluated the efficacy of a home-based treatment that provided treatment-seeking GAD patients with both ABM and computerized cognitive-behavioral therapy (CBT), accessed online as often as desired across a 6-week period. Patients who completed this intervention evidenced reduced attention to negative information and displayed significant reductions in both self-reported and clinician-rated symptoms of anxiety, worry, and depression. Symptom change was related to the magnitude of the observed reduction in attentional bias. At completion of the 6-week intervention, 79% of participants no longer met diagnostic criteria for GAD. This study was not designed to distinguish the contributions made by the CBT and ABM components of the online package. It will be important for future investigators to directly compare the efficacy of CBT and ABM procedures, and also to determine whether the combination of these two types of cognitive intervention can deliver greater therapeutic benefit than does either approach alone. Nevertheless, we agree with Amir and Taylor that the incorporation of ABM into computerized, home-based treatments for emotional dysfunction, represents a promising future method of effectively exploiting its demonstrated capacity to enhance emotion regulation.

Closing Comments

The use of ABM procedures to test hypotheses concerning the contributions of attentional selectivity to emotion regulation and therapeutically enhance such regulation in people experiencing emotional dysfunction is still a young area of research. Well over 70% of all the research published in this field has appeared only within the past 3 or 4 years (MacLeod & Mathews, 2012). Nevertheless, this early work already has clearly established that biased attentional responding to negative information does causally contribute to emotion regulation, affecting emotional reactivity to stress and influencing the symptoms of emotional dysfunction. The research reviewed in this chapter also has convincingly demonstrated that the use of ABM procedures to reduce attention to negative information can improve emotion regulation. Inducing such attention change serves to attenuate the degree to which stress elicits dysphoric emotional responses and also to alleviate the negative emotional symptoms associated with subclinical or clinical manifestations of emotional pathology. By drawing upon increasingly sophisticated contemporary approaches to attentional bias assessment, designed to assess progressively more precise dimensions of attention, it should be possible to develop novel training variants that target highly specific aspects of selective attention for modification. Hence, we anticipate that future ABM research will illuminate the ways in which particular facets of attention selectivity functionally contribute to different features of emotion regulation. Furthermore, if these evolving ABM technologies become more widely embedded in the types of computerized treatment programs now being used to deliver psychological interventions, we also can expect that they will make a growing future contribution to the effective remediation of emotional dysfunction.

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References

Amir, N., Beard, C., Burns, M., & Bomyea, J. (2009). Attention modification program in individuals with generalized anxiety disorder. *Journal of Abnormal Psychology, 118*(1), 28–33.

Amir, N., Beard, C., Taylor, C. T., Klumpp, H., Elias, J., Burns, M., et al. (2009). Attention training in individuals with generalized social phobia: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 77*(5), 961–973.

Amir, N., Weber, G., Beard, C., Bomyea, J., & Taylor, C. T. (2008). The effect of a single-session attention modification program on response to a public-speaking challenge in socially anxious individuals. *Journal of Abnormal Psychology, 117*(4), 860–868.

Amir, T., & Taylor, C. (2012). Combining computerized home-based treatments for generalized anxiety disorder: An attention modification program and cognitive behavioral therapy. *Behavior Therapy, 43*, 546–559.

Ashley, V., & Swick, D. (2009). Consequences of emotional stimuli: Age differences on pure and mixed blocks of the emotional Stroop. *Behavioral and Brain Functions, 5*(14).

Attwood, A. S., O'Sullivan, H., Leonards, U., Mackintosh, B., & Munafo, M. R. (2008). Attentional bias training and cue reactivity in cigarette smokers. *Addiction, 103*(11), 1875–1882.

Baert, S., De Raedt, R., Schacht, R., & Koster, E. H. (2010). Attentional bias training in depression: Therapeutic effects depend on depression severity. *Journal of Behavior Therapy and Experimental Psychiatry, 41*(3), 265–274.

Bar-Haim, Y. (2010). Attention bias modification (ADM): A novel treatment for anxiety disorders. *Journal of Child Psychology and Psychiatry, 51*(8), 859–870.

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*, 1–24.

Bar-Haim, Y., Morag, I., & Glickman, S. (2011). Training anxious children to disengage attention from threat: A randomized controlled trial. *Journal of Child Psychology and Psychiatry, 52*(8), 861–869.

Barlow, D. (2002). *Anxiety and its disorders: The nature and treatment of anxiety and panic* (2nd ed.). New York: Guilford Press.

Beard, C. (2011). Cognitive bias modification as a treatment for anxiety: Current evidence and future directions. *Expert Review of Neurotherapeutics, 11*(2), 299–311.

Beard, C., Sawyer, A., & Hofmann, S. (2012). Efficacy of attention bias modification using threat and appetitive stimuli: A meta-analytic review. *Behavior Therapy, 43*, 724–740.

Beard, C., Weisberg, R. B., & Amir, N. (2011). Combined cognitive bias modification treatment for social anxiety disorder: A pilot trial. *Depression and Anxiety, 28*(11), 981–988.

Bockstaele, V., Koster, E., Verschuere, B., Crombez, G., & de Houwer, J. (2012). Limited transfer of threat bias following attentional retraining. *Journal of Behavior Therapy and Experimental Psychiatry, 43*, 794–800.

Bockstaele, V., Verschuere, B., Koster, E. H., Tibboel, H., De Houwer, J., & Crombez, G. (2011). *Journal of Behavioral Therapy and Experimental Psychiatry, 42*(2), 211–218.

Bradley, B. P., Mogg, K., White, J., Groom, C., & de Bono, J. (1999). Attentional bias for emotional faces in generalized anxiety disorder. *British Journal of Clinical Psychology, 38*, 267–278.

Brosan, L., Hoppitt, L., Shelfer, L., Sillence, A., & Mackintosh, B. (2011). Cognitive bias modification for attention and interpretation reduces trait and state anxiety in anxious patients referred to an out-patient service: Results from a pilot study. *Journal of Behavior Therapy and Experimental Psychiatry, 42*(3), 258–264.

Browning, M., Holmes, E. A., Murphy, S. E., Goodwin, G. M., & Harmer, C. J. (2010). Lateral prefrontal cortex mediates the cognitive modification of attentional bias. *Biological Psychiatry, 67*(10), 919–925.

Carlbring, P., Apelstrand, M., Sehlin, H., Amir, A., Rousseau, A., Hofmann, et al. (2012). Internet-delivered attention bias modifiction training in individuals with social anxiety disorder: A double blind randomized control trial. *BMC Psychatry, 12*, 66.

Cisler, J., & Koster, E. H. (2010). Mechanisms of attentional biases towards threat in anxiety disorders: An integrative review. *Clinical Psychology Review, 30*, 203–216.

Dandeneau, S. D., & Baldwin, M. W. (2004). The inhibition of socially rejecting information among people with high versus low self-esteem: The role of attentional bias and the effects of bias reduction training. *Journal of Social and Clinical Psychology, 23*(4), 584–602.

Dandeneau, S. D., & Baldwin, M. W. (2009). The buffering effects of rejection-inhibiting attentional training on social and performance threat among adult students. *Contemporary Educational Psychology, 34*(1), 42–50.

Dandeneau, S. D., Baldwin, M. W., Baccus, J. R., Sakellaropoulo, M., & Pruessner, J. C. (2007). Cutting stress off at the pass: Reducing vigilance and responsiveness to social threat by manipulating attention. *Journal of Personality and Social Psychology, 93*(4), 651–666.

D'Angelo, M., Milliken, B., Jimenez, L., & Lupianez, J. (2013). Implementing flexibility in automaticity: Evidence from context-specific implicit sequence learning. *Consciousness and Cognition, 22*(1), 64–81.

Derryberry, D., & Reed, M. A. (2002). Anxiety-related attentional biases and their regulation by attentional control. *Journal of Abnormal Psychology, 111*(2), 225–236.

Eccleston, C., & Crombez, G. (1999). Pain demands attention: A cognitive-affective model of the interruptive function of pain. *Psychological Bulletin, 125*(3), 356–366.

Eldar, S., Apter, A., Lotan, D., Edgar, K. P., Naim, R., Fox, N. A., & Bar-Haim, Y. (2012). Attention bias modification treatment for pediatric anxiety disorders: A randomized controlled trial. *American Journal of Psychiatry, 169*, 213–220.

Eldar, S., & Bar-Haim, Y. (2010). Neural plasticity in response to attention training in anxiety. *Psychological Medicine, 40*(4), 667–677.

Eldar, S., Ricon, T., & Bar-Haim, Y. (2008). Plasticity in attention: Implications for stress response in children. *Behaviour Research and Therapy, 46*(4), 450–461.

Fadardi, J. S., & Cox, W. M. (2009). Reversing the sequence: Reducing alcohol consumption by overcoming alcohol attentional bias. *Drug and Alcohol Dependence, 101*(3), 137–145.

Grafton, B., Ang, C., & MacLeod, C. (2012). Always look on the bright side of life: The attentional basis of positive affectivity. *European Journal of Personality, 26*, 133–144.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*, 271–299.

Gross, J. J., & Barrett, L. F. (2011). Emotion generation and emotion regulation: One or two depends on your point of view. *Emotion Review, 3*, 8–16.

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York: Guilford Press.

Hakamata, Y., Lissek, S., Bar-Haim, Y., Britton, J. C., Fox, N. A., Leibenluft, E., et al. (2010). Attention bias modification treatment: A meta-analysis toward the establishment of novel treatment for anxiety. *Biological Psychiatry, 68*(11), 982–990.

Hallion, L., & Ruscio, A. (2011). A meta-analysis of the effect of cognitive bias modification on anxiety and depression. *Psychological Bulletin, 6*, 940–958.

Hayes, S., Hirsch, C. R., & Mathews, A. (2010). Facilitating a benign attentional bias reduces negative thought intrusions. *Journal of Abnormal Psychology, 119*(1), 235–240.

Hazen, R. A., Vasey, M. W., & Schmidt, N. B. (2009). Attentional retraining: A randomized clinical trial for pathological worry. *Journal of Psychiatric Research, 43*(6), 627–633.

Heeren, A., Lievens, L., & Philippot, P. (2011). How does attention training work in social phobia: Disengagement from threat or re-engagement to non-threat? *Journal of Anxiety Disorders, 25*, 1108–1115.

Heeren, A., Reese, H. E., McNally, R. J., & Philippot, P. (2012). Attention training toward and away from threat in social phobia: Effects on subjective, behavioral, and physiological measures of anxiety. *Behaviour Research and Therapy, 50*, 30–39.

Hertel, P. T., & Mathews, A. (2011). Cognitive bias modification: Past, perspective, current findings, and future applications. *Perspectives in Psychological Science, 6*(6), 521–536.

Hirsch, C., Hayes, S., & Mathews, A. (2009). Looking on the bright side: Accessing benign meanings reduces worry. *Journal of Abnormal Psychology, 118*, 44–54.

Hirsch, C., MacLeod, C., Mathews, A., Sandher, O., Siyani, A., & Hayes, S. (2011). The contribution of attentional bias to worry: Distinguishing the roles of selective engagement and

disengagement. *Journal of Anxiety Disorders, 25*, 272–277.

Julian, K., Beard, C., Schmidt, N. B., Powers, M. B., & Smits, J. A. J. (2012). Attention training to reduce attention bias and social stressor reactivity: An attempt to replicate and extend previous findings. *Behavior Research and Therapy, 50*, 350–358.

Klumpp, H., & Amir, N. (2010). Preliminary study of attention training to threat and neutral faces on anxious reactivity to a social stressor in social anxiety. *Cognitive Therapy and Research, 34*, 263–271.

Kring, A. M., & Werner, K. H. (2004). Emotion regulation and psychopathology. In P. Philippot & R. S. Feldman (Eds.), *The regulation of emotion* (pp. 359–385). Oxford, UK: Psychology Press.

Leclerc, C. M., & Kensinger, E. A. (2008). Effects of age on detection of emotional information. *Psychology and Aging, 23*(1), 209–215.

Li, S., Tan, J., Qian, M., & Liu, X. (2008). Continual training of attentional bias in social anxiety. *Behaviour Research and Therapy, 46*(8), 905–912.

MacLeod, C., & Bucks, R. S. (2011). Emotion regulation and the cognitive-experimental approach to emotional dysfunction. *Emotion Review, 3*(1), 62–73.

MacLeod, C., Koster, E., & Fox, E. (2009). Whither cognitive bias modification research?: Commentary on the special section articles. *Journal of Abnormal Psychology, 118*, 89–99.

MacLeod, C., & Mathews, A. (2012). Cognitive bias modification approaches to anxiety. *Annual Review of Clinical Psychology, 8*, 189–217.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95*, 15–20.

MacLeod, C., Soong, L. Y., Rutherford, E. M., & Campbell, L. W. (2007). Internet-delivered assessment and manipulation of anxiety-linked attentional bias: Validation of a free-access attentional probe software package. *Behavior Research Methods, 39*, 533–538.

MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective attention and emotional vulnerability: Assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of Abnormal Psychology, 111*, 107–123.

Mathews, A., & MacLeod, C. (2002). Induced processing biases have causal effects on anxiety. *Cognition and Emotion, 16*, 331–354.

Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annual Review of Clinical Psychology, 1*, 167–195.

Marks, I., & Cavanagh, K. (2009). Computer-aided psychological treatments: Evolving issues. *Annual Review of Clinical Psychology, 5*, 121–141.

Moses, E. B., & Barlow, D. H. (2006). A new unified treatment approach for emotional disorders based on emotion science. *Current Directions in Psychological Science, 15*(3), 146–150.

Najmi, S., & Amir, N. (2010). The effect of attention training on a behavioral test of contamination fears in individuals with subclinical obsessive–compulsive symptoms. *Journal of Abnormal Psychology, 119*(1), 136–142.

Nisbett, R. E., & Wilson, T. D. (1977). Telling more than we can know: Verbal reports on mental processes. *Psychological Review, 84*, 231–259.

Owens, M., Koster, E., & Derakshan, N. (2013). Improving attention control in dysphoria through cognitive training: Transfer effects on working memory capacity and filtering efficiency. *Psychophysiology, 50*(3), 297–307.

Reese, H. E., McNally, R. J., Najmi, S., & Amir, N. (2010). Attention training for reducing spider fear in spider-fearful individuals. *Journal of Anxiety Disorders, 24*, 1–8.

Richards, J. M., & Gross, J. J. (2006). Personality and emotional memory: How regulating emotion impairs memory for emotional events. *Journal of Research in Personality, 40*, 631–651.

Rozenman, M., Weersing, V., & Amir, N. (2011). A case series of attention modification in clinically anxious youths. *Behaviour Research and Therapy, 49*(5), 324–330.

Schmidt, N. B., Richey, J., Buckner, J. D., & Timpano, K. R. (2009). Attention training for generalized social anxiety disorder. *Journal of Abnormal Psychology, 118*(1), 5–14.

See, J., MacLeod, C., & Bridle, R. (2009). The reduction of anxiety vulnerability through the modification of attentional bias: A real-world study using a home-based cognitive bias modification procedure. *Journal of Abnormal Psychology, 118*(1), 65–75.

Sharpe, L., Perry, K. N., Rodgers, P., Dear, B. F., Nicholas, M. K., & Refshauge, K. (2010). A comparison of the effect of attentionl training

and relaxation responses to pain. *Pain, 150*(3), 469–476.

Taylor, C., Bomyea, J., & Amir, N. (2011). Malleability of attentional bias for positive emotional information and anxiety vulnerability. *Emotion, 11*, 127–138.

Thompson, R. A., & Goodman, M. (2010). Development of emotion regulation: More than meets the eye. In A. Kring & D. Sloan (Eds.), *Emotion regulation and psychopathology* (pp. 38–58). New York: Guilford Press.

Troy, A. S., & Mauss, I. B. (2011). Resilience in the face of stress: Emotion regulation as a protective factor. In S. M. Southwick, B. T. Litz, D. Charney, & M. J. Friedman (Eds.), *Resilience and mental health: Challenges across the lifespan* (pp. 30–44). New York: Cambridge University Press.

Wadlinger, H. A., & Isaacowitz, D. M. (2008). Looking happy: The experimental manipulation of a positive visual attention bias. *Emotion, 8*(1), 121–126.

Wegner, D. M., Schneider, D. J., Carter S. R., & White, T. L. (1987). Paradoxical effects of thought suppression. *Journal of Personality and Social Psychology, 53*, 5–13.

Wells, T. T., & Beevers, C. G. (2010). Biased attention and dysphoria: Manipulating selective attention reduces subsequent depressive symptoms. *Cognition and Emotion, 24*(4), 719–728.

Werner, K., & Gross, J. J. (2010). Emotion regulation and psychopathology: A conceptual framework. In A. Kring & D. Sloan (Eds.), *Emotion regulation and psychopathology* (pp. 13–37). New York: Guilford Press.

White, L. K., Helfinstein, S. M., Reeb-Sutherland, B. C., Degnan, K. A., & Fox, N. A. (2009). Role of attention in the regulation of fear and anxiety. *Developmental Neuroscience, 31*, 309–317.

White, L. K., Suway, J. G., Pine, D. S., Bar-Haim, Y., & Fox, N. A. (2011). Cascading effects: The influence of attention bias to threat on the interpretation of ambiguous information. *Behaviour Research and Therapy, 49*(4), 244–251.

Wiers, R., Bartholow, B., van den Wildenberg, E., Thush, C., Engels, R., Sher, K., et al. (2007). Automatic and controlled processes and the development of addictive behaviors in adolescents: A review and a model. *Pharmacology Biochemistry and Behavior, 86*(2), 263–283.

Williams, M. G., Mathews, A., & MacLeod, C. (1996). The emotional Stroop task and psychopathology. *Psychological Bulletin, 120*(1), 3–24.

CHAPTER 31

Affect Regulation Training

Matthias Berking
Jeanine Schwarz

Deficits in emotion regulation skills are a putative risk and maintaining factor in various forms of psychopathology (Berking & Wupperman, 2012; Hofmann, Sawyer, Fang, & Asnaani, 2012; Werner & Gross, 2010). The defining criteria for many disorders listed in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013) include undesired emotions, such as sadness, anxiety, or anger, that exceed the intensity and/or duration of what is considered adaptive. For other disorders, core symptoms can be conceptualized as dysfunctional attempts to avoid or down-regulate undesired emotions. For example, bingeing in eating disorders and substance use in alcohol dependence are often seen as dysfunctional attempts to cope with states of sadness, anger, boredom, or other forms of emotional anguish (Berking et al., 2011; Dingemans, Martijn, Jansen, & Furth, 2009).

In a similar fashion, the development and maintenance of depressive thinking can be conceptualized as dysfunctional emotion regulation. Appraising a situation as aversive, uncontrollable, and unlikely to change over time (Teasdale & Barnard, 1993) may help to reduce the pressure actively to solve personal problems (because working on unattainable goals would be pointless) and provide protection against hurtful disappointments (because hope is an antecedent of disappointment). In a situation in which an individual is under high pressure to solve numerous problems but neither feels able to do so nor to bear another disappointment, a depressogenic type of thinking that reduces the pressure associated with unsolved problems and/or the pain associated with unfulfilled hopes may become an irresistible temptation. Giving in to this type of thinking may result in a small and short-lived reduction of negative affect. In turn, this type of thinking is reinforced and more likely to occur again in similar situations in the future. Eventually, this process may lead into a clinically significant depression. Adaptive affect regulation skills present an alternative way of coping with undesired affective states that may prevent such depressogenic cognitive processes or facilitate disengagement from such processes (Berking & Whitley, 2013).

Given the increasing amount of evidence favoring such theories across various mental disorders, enhancing emotion regulation skills can be considered a promising transdiagnostic treatment target (Berking, Wupperman, et al., 2008; Mennin & Fresco, 2009; Moses & Barlow, 2006). Others have also argued that undifferentiated stress responses (Bogdan & Pizzagalli, 2006), negative mood states (Van Rijsbergen, Bockting, Berking, Koeter, & Schene, 2012), motivational

impulses (Grawe, 2006), and even appraisal processes, which are defined through a strong intrinsic affective component (e.g., "feelings" of hopelessness; Gibb, Beevers, Andover, & Holleran, 2006; Teasdale & Barnard, 1993), constitute a serious health risk when the individual is unable to cope successfully with these phenomena. Based on the feasibility of applying "emotion" regulation skills to these other affective states, we propose that the same arguments that indicate the importance of emotion regulation skills also apply to undesired affective states in general (i.e., emotions, stress responses, moods, and urges; Gross, this volume). Thus, the potential of targeting emotion regulation skills in treatment can be hypothesized to generalize to affect regulation skills in general.

Almost all psychotherapeutic treatments implicitly or explicitly work to improve affect regulation skills in one way or another. However, in some treatments (e.g., person-centered psychotherapy, psychodynamic treatments) general emotion regulation skills are not explicitly addressed and directly targeted but are assumed to be enhanced through the application of strategies considered to stimulate the healing process in general (e.g., personal growth, self-understanding, insight into unconscious motives). In other treatments, such as cognitive-behavioral therapy (CBT), these efforts often focus on affective states that are thought to be strongly related to the client's symptoms. For example, clients suffering from anxiety are primarily taught skills to cope successfully with anxiety. Although patients are usually encouraged to apply the acquired skills to other emotions, specific attempts to (1) systematically train patients to apply acquired coping skills in the context of other challenging affective states or (2) to teach and practice additional skills that are not relevant in the context of anxiety but are relevant for successful coping with other undesired affective states (e.g., distraction as a strategy to reduce anger; engagement in positive activities to reduce dysphoric mood and feelings of hopelessness) are rare. If these additional undesired affective states are relevant for the maintenance of the anxiety disorder, focusing only on anxiety-managing skills may not be sufficient to solve the problem effectively. On such occasions (e.g., if anger, dysphoric mood, and/or hopelessness significantly reduce the client's motivation and capacity to confront feared stimuli and/or to draw helpful conclusions from such exercises or habituate to feared stimuli; e.g., Foa, Riggs, Massie, & Yarczower, 1995), a systematic focus on general affect regulation skills might be warranted.

Moreover, many patients suffer from more than one mental disorder (Kessler, Chiu, Demler, & Walters, 2005). Particularly in highly comorbid patients, "transaffective" deficits in adaptive coping skills (e.g., the inability to accept various undesired affective states if they cannot be modified) might be at the core of many of their problems. For these patients, a systematic focus on affect regulation skills in general might be a more efficient way of treatment than a compilation of various disorder-specific manuals. Minimally, an additional focus on strengthening affect regulation skills in addition to providing disorder-specific treatments should be considered when there is evidence that general affect regulation deficits have contributed to various psychopathological symptoms. Thus, there is a need for interventions focusing on *general* affect regulation skills that may complement disorder-focused interventions.

Treatments such as emotion regulation therapy (ERT; Mennin & Fresco, this volume), the emotion regulation module used in dialectic behavior therapy (DBT; Neacsiu, Bohus, & Linehan, this volume), and some mindfulness-based treatments (Farb, Anderson, Irving, & Segal, this volume; Wupperman et al., 2012) are based on a similar conceptualization of mental disorders as affect regulation training (ART) and, consequently, also address affect regulation skills in a more general fashion. The main difference between these treatments and ART is that ART has from its beginning focused on communalities across various mental disorders with regard to deficits in general emotion regulation skills. To improve such skills, ART systematically integrates techniques from various psychotherapeutic approaches, such as CBT (e.g., Beck, 1995), compassion-based therapy (Gilbert, 2011; Weissman & Weissman, 1996), DBT (Neacsiu et al., this volume), emotion-focused therapy (EFT; Greenberg, 2004), ERT (Mennin & Fresco, this volume), mindfulness-based interven-

tions (Farb et al., this volume), neuropsychotherapeutic translational approaches (Grawe, 2006), principles used in problem-solving therapies (D'Zurilla & Nezu, 2010), and strength-focused interventions (Duckworth, Steen, & Seligman, 2005; Grawe, 2002), into a highly standardized and transdiagnostically oriented training program. This program can be utilized (1) as a stand-alone intervention (when working with patients suffering from less severe or subthreshold mental disorders, with individuals at risk for developing mental health problems, or with healthy individuals who simply desire to enhance their well-being) or (2) as an adjunctive intervention complementing any empirically validated treatment of mental disorders whenever a stronger focus on enhancing general affect regulation skills is desired. Although ART certainly shares some theoretical assumptions with many of the approaches just described and also uses some of the interventions applied in these approaches, we believe that the uniqueness and advantage of ART lies with its comprehensive approach of incorporating specific strategies, interventions, and exercises into a consistent training program that explicitly and exclusively focuses on enhancing general affect regulation skills—regardless of the patient's particular disorder(s) (for details see Berking & Whitley, 2013).

Inspired by Klaus Grawe's (2006) translational ideas on the development of mental disorders, as well as the lack of transdiagnostic interventions to enhance general affect regulation skills at the time, we began to develop ART in 2004. Since then, the training has become quite popular in the German-speaking parts of Europe, where it is known as *Training Emotionaler Kompetenzen* (TEK; Berking, 2010; *www.tekonline.info*). The training was primarily developed as a group-based intervention (four to eight participants), but it can also be used in individual therapy. Although the program has only been evaluated in adults, its application for adolescents is practicable after adapting the procedures and materials to the needs and capacities of this age group. The training, which takes at least 18 hours to complete, can be partitioned in various ways (e.g., two 1.5-hour sessions or one 3-hour session per week over a 6-week period). From our experience, however, participants should have at least 4 to 6 weeks time to practice the skills-building exercises. ART can be delivered by an ART-trained mental health professional, such as a licensed psychotherapist with a master's degree in medicine or psychology. Other, nonlicensed mental health professionals can also be trained to administer ART, albeit not to individuals suffering from acute mental disorders. Subsequently, we outline the theoretical and empirical background of ART before we provide practical explanations of how the relevant skills are delivered to participants. At the end of the chapter, we review empirical evidence for the effectiveness of ART and suggest future directions for our research.

The ART Model of Adaptive Affect Regulation

The ART model of adaptive affect regulation was developed by synthesizing (1) established affect regulation theories (e.g., Gottman & DeClaire, 1997; Grawe, 2002, 2006; Gross, 1998; Larsen, 2000; Lazarus, 1991; Leahy, 2002; Saarni, 1999; Salovey & Mayer, 1990), (2) findings from studies on the association between affect regulation and psychopathology (Berking & Wupperman, 2012), and (3) our extensive clinical experience. The purpose of this development was to provide a normative (i.e., prescriptive) model explaining common failures in affect regulation and directions for interventions to enhance affect regulation. In the resulting model, adaptive affect regulation is conceptualized as a situation-dependent interaction between several affect regulation skills. Figure 31.1 illustrates an overview of the model. The skills included in the model are described below.

1. *The ability to consciously perceive and be aware of affective states.* The ability to consciously perceive and be aware of affective states is the basis of every effortful attempt to regulate affective states. Effortful and conscious regulation may only play a small role in the daily routine of regulation in a healthy individual, because implicit or highly automatized processes are likely to dominate the regulation process as long as these processes are able to maintain the individual's affect in the comfort zone (Koole &

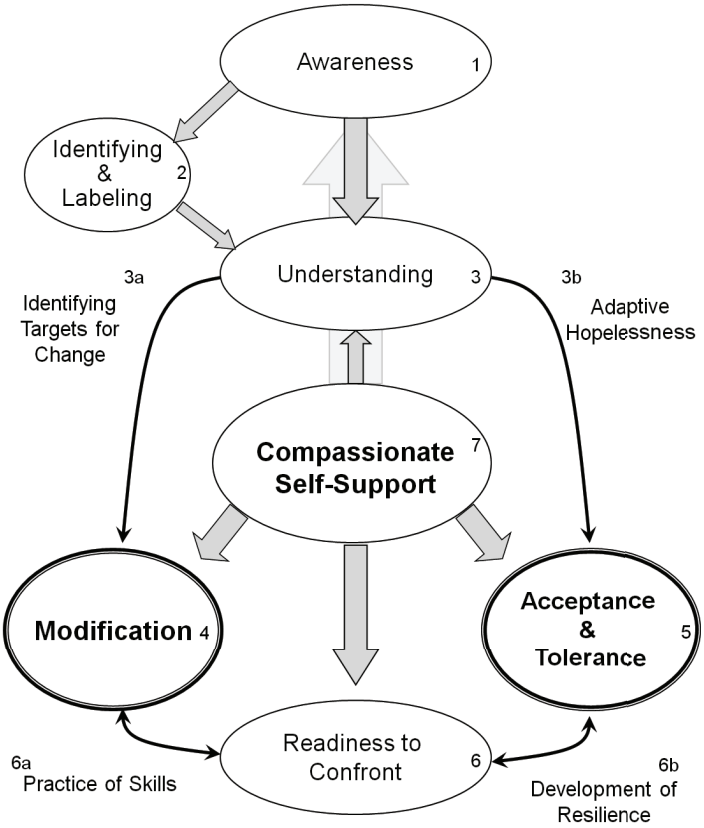

FIGURE 31.1. The ART model of adaptive affect regulation.

Rothermund, 2011; Gyurak & Etkin, this volume). However, if undesired affective states cannot be down-regulated through implicit processes, conscious information processing will be activated as a precious resource reserved for solving problems in the self-management process (Mauss & Tamir, this volume). Becoming aware of affective states may facilitate the use of affect regulation skills requiring cognitive resources (e.g., evaluating the degree of undesirability of the emotion, labeling the emotion, identifying its causes) and hence foster adaptive regulation. Conversely, the inability to become aware of one's emotions impedes the utilization of cognitive problem-solving capacities, thus intensifying and prolonging the unwanted affective state (e.g., Lane et al., 1996; Swart, Kortekaas, & Aleman, 2009).

2. *The ability to identify and correctly label affective states*. Identifying and labeling affective states refers to matching emotional experiences with the appropriate semantic categories (e.g., "This feeling I am experiencing is anger"). The availability of a differentiated system of cognitive representations of affective states and the ability to correctly assign an experience to a specific category helps the individual to build and use knowledge about this state, which is often helpful for adaptive regulation. For example, recognizing an emotion as anger provides information about the nature and purpose of the emotion (e.g., enhancing assertive behavior; fighting for one's rights), its potential risks (cueing aggression leading to sanctions through others) and benefits (getting what one wants, even if one has to fight for it), as well as information on regulation strategies that are likely to be effective (potentially effective: distraction, relaxation, reappraisal and compassion; likely to be ineffective: prolonged exposure). Recognizing the emotion facilitates adaptive cog-

nitive processing. Thus, this ability should foster effective affect regulation and hence mental health and well-being (Feldman-Barrett, Gross, Christensen, & Benvenuto, 2001).

3. *The ability to identify relevant maintaining factors for current affective states.* Identifying relevant maintaining factors refers to development of an inner working model to explain factors that initially cued the current affective state and, more importantly, to identify the factors that maintain the emotional state at that moment. In general, psychological treatment for mental health problems should (a) normalize the client's experiences, (b) help to explain the occurrence of the problem, and (c) provide direction for successful coping. With an additional focus on affective states, the model should include relevant and changeable factors such as the internal or external stimuli or situation cueing the emotion, appraisal of the stimuli or situation, as well as desires, beliefs, and goals relevant for the appraisal. Understanding factors that initially elicit and maintain an affective state helps to (a) give meaning to an aversive experience, thereby making it easier to bear; (b) clarify whether the emotion can or cannot be changed (thus enhancing the acceptance of an emotion when necessary); and (c) identify targets for effective modification of the affective state.

4. *The ability to modify affective states actively.* To modify affective states actively is to effectively change the quality, intensity, and/or duration of an affective state into a desired direction with the help of strategies that have no unwanted, long-term side effects. This ability provides one of the most effective means for preventing the use of dysfunctional strategies (i.e., strategies that have unwanted side effects). Moreover, effective modification skills provide an empirical basis for the expectation of being able to modify unwanted affective states whenever necessary. Such an emotional self-efficacy is likely to reduce both anxiety about future aversive events and avoidance tendencies toward affective states. As a result, this expectation reduces general anxiety and the willingness to experience emotionally challenging situations, thus facilitating further skills building. Evidence for this hypothesis comes from studies demonstrating that an individual's expectation of successfully alleviating negative mood states is significantly associated with mental health (Catanzaro & Greenwood, 1994). In this context, it is noteworthy that ART skills should be understood as categories that include various subskills. This is particularly relevant for the ability to modify affective states. For example, most of the long list of positive activities used in behavioral therapy for depression can be seen as modification skills. It is also noteworthy that the effectiveness of specific modification skills differs across individuals and situations, and is likely to be moderated by numerous individual and contextual factors (Bonanno, Papa, Lalande, Westphal, & Coifman, 2004).

5. *The ability to accept and tolerate negative affective states when necessary.* Acceptance and tolerance of unwanted affective states is a crucial skill whenever affective states cannot be changed or when changing them would involve too high a cost (e.g., in terms of time and energy). Although an optimistic perspective about one's abilities to modify affective states has clear advantages, acknowledging that affective states are often very difficult to modify, or that they sometimes cannot be modified, is equally advantageous (Hayes, Strosahl, & Wilson, 1999). Modifying undesired affective states is often difficult—particularly for patients suffering from mental disorders—and hence may threaten the need for control. To end the aversive affective state and/or to restore a sense of control, the individual may engage in dysfunctional strategies (e.g., suppression) that help to avoid the aversive experience but likely lead to unwanted long-term outcomes (e.g., Feldner, Zvolensky, Stickle, Bonn-Miller, & Leen-Feldner, 2006; Levitt, Brown, Orsillo, & Barlow, 2004). Conversely, the availability to accept, bear, or tolerate aversive affective states reduces this need and hence the risk of engaging in maladaptive affect regulation (e.g., Wupperman, Neumann, & Axelrod, 2008).

6. *The ability to approach and confront situations that may cue negative affective states.* Approaching and confronting situations that may cue negative affective states is often necessary to accomplish personally relevant goals (Hayes et al., 1999). More-

over, confronting these situations provides an opportunity to improve existing affect regulation skills and to develop new ones (arrows 6a and 6b in Figure 31.1). Willingly encountering occasional situations that cue negative affective states to attain important goals when necessary, ensures that one's affect regulation skills are practiced on a regular basis and will be available and effective in times of emotional anguish. Moreover, as this ability facilitates goal attainment, it helps to satisfy relevant needs and thus reduces negative affect in the long run.

7. *The ability to support oneself compassionately in distressing situations*. All of the skills just described have the potential to increase emotional suffering in the short term. For example, consciously perceiving unwanted affective states is usually associated with an increase in the perceived intensity of the emotional suffering. Identifying momentary feelings such as stress, fear, anger, sadness, dysphoria, shame, and guilt activates semantic concepts that likely to cue further negative feelings. Working to understand undesired affective states can lead to the processing of problems and the acknowledgment of one's inabilities to solve them, which is likely to cue further deterioration of mood. Efforts to modify undesired affective states are often unsuccessful, or at least not as successful as expected or desired, hence cueing disappointment, frustration, and eventually despair. Attempts to accept and tolerate an emotion are often associated with feelings of helplessness and hopelessness, and unrealistic fears of catastrophic consequences assumed to occur when negative affective states are no longer suppressed. Moreover, confronting situations that cue negative affective states to attain important goals, by definition, cues negative affective states. As illustrated by these examples, the process of applying adaptive affect regulation skills could lead to deterioration of mood and trigger the use of dysfunctional strategies. Therefore, self-soothing or self-encouraging strategies are needed as part of the regulation process to prevent one's mood from deteriorating. Unfortunately, many individuals with mental disorders have a tendency to be strongly self-critical of perceived failures. In the process of affect regulation, this self-critical tendency is likely to manifest when individuals perceive that they have failed to regulate their emotions successfully. Such a critique usually cues further (secondary) negative affective states (see Greenberg, 2004) that impede efforts to cope with the undesired primary affective states. Conversely, a compassionate stance toward oneself can help reduce the risk of being caught up in such a vicious cycle (Gilbert, 2011).

In addition to the list of skills and assumptions about why these skills are important and how they interact, the ART model includes the hypothesis that modification and acceptance/tolerance skills are the only skills in the model that are ultimately relevant for mental health. All other skills are not considered relevant by themselves but only to the extent that they facilitate the successful application of modification or acceptance/tolerance. This idea is based on research on rumination (e.g., Morrow & Nolen-Hoeksema, 1990) and our clinical observation that many clients obviously experience their affective states consciously, analyze them, and work to explain them but are unable to benefit from these activities. On the contrary, excessive use of these strategies seemed to be partly responsible for maintaining the mental disorder. Empirical studies on the ART model, reviewed in the next section, provide further preliminary support for this assumption.

Empirical Evidence for the ART Model of Affect Regulation

After we conceptualized the ART model, we developed and validated the Emotion Regulation Skills Questionnaire (ERSQ; Berking & Znoj, 2008) to assess the skills included in the model. This self-report instrument utilizes a 5-point Likert scale to assess each skill of the ART model (plus an additional subscale assessing the ability to use body sensations to identify experienced affective states) with three items per scale. Additionally, a total score can be computed as the average of all items. In several studies, the ERSQ displayed adequate to good internal consistencies (Cronbach's alpha = .82–.94). Results from exploratory and confirmatory factor analyses provide support for the

assumed dimensionality of the measure. Moreover, sensitivity to change has been demonstrated in multiple samples of clients undergoing psychotherapy, and all scales have demonstrated positive associations with measures of well-being and mental health, and negative associations with measures of psychopathology and ER deficits (Berking, Ebert, Cuijpers, & Hofmann, 2013; Berking et al., 2011, 2012; Berking, Meier, & Wupperman, 2010; Berking, Orth, Wupperman, Meier, & Caspar, 2008; Berking, Wupperman, et al., 2008; Berking & Znoj, 2008; Ebert, Christ, & Berking, 2013).

More specifically, all skills in the ART model have been shown to be cross-sectionally associated with mental health in nonclinical samples (Berking et al., 2012; Berking, Wupperman, et al., 2008; Berking & Znoj, 2008), and clinical samples and nonclinical controls report less successful application of all skills than do patients treated for mental disorders (Berking et al., 2011; Berking, Wupperman, et al., 2008; Berking et al., 2013). Additionally, an increase in successful affect regulation during CBT was shown to be associated with greater symptom reduction (Berking, Wupperman, et al., 2008). The differences and correlations reported in these studies were particularly strong for modification and acceptance/tolerance. This finding provides preliminary support for assumption that these skills are particularly relevant for mental health. Moreover, multivariate regression analyses identified only these two skills areas as making a unique contribution when predicting treatment outcome from changes in affect regulation skills (e.g., Berking, Wupperman, et al., 2008). Further research indicated that affect regulation skills assessed through the ERSQ predicted subsequent indicators of psychopathology over a 2-week and a 5-year period, whereas these indicators did not predict subsequent success in applying affect regulation skills (Berking, Orth, et al., 2008; Berking, Wirtz, Svaldi, & Hofmann, under review). This finding suggests that deficits in affect regulation skills are not merely a symptom of mental health problems but a relevant antecedent factor.

In two other studies, affect regulation skills assessed through the ERSQ predicted relapse during and after inpatient treatment for alcohol dependency (Berking et al., 2011) and the reduction of depressive symptoms in patients treated for major depressive disorder (MDD; Radkovsky, McArdle, Bockting, & Berking, under review). Finally, in both a clinical and a nonclinical sample, successful modification of undesired affective states (partly) mediated the relationship between most ERSQ skills and psychopathology, but not for acceptance and psychopathology (Berking et al., 2012), which suggests that only acceptance and tolerance are relevant for mental health, regardless of whether these skills facilitate the modification of affective states. In summary, a growing body of evidence supports the hypotheses that the skills included in the ART model help maintain or restore mental health and are thus promising for treatments of mental health problems.

Brief Description of the ART Program

Support for the ART model led to the development of the ART program, which integrates principles and interventions from various psychotherapeutic approaches to foster general affect regulation skills in individuals likely to benefit from more adaptive ways of coping with unwanted affective states. The following sections provide a brief description of the ART program (for a more detailed description, see Berking & Whitley, 2013).

General Structure

ART first introduces participants to the nature of affective states and their evolutionary background, purposes, potential risks, and benefits, as well as the mechanisms involved in regulating affective processing. From our understanding of the mechanisms involved, seven skills have demonstrated effectiveness in facilitating adaptive coping with affective states that are particularly challenging to manage. These skills include muscle relaxation, breathing relaxation, nonjudgmental awareness, acceptance and tolerance, compassionate self-support, analysis of affective states, and modification of affective states. Each skill is practiced in a long version, then shortened to a version that can be applied in very short periods of

time (3 seconds to 3 minutes). The shortened *ART skills* are then chained together, creating the *ART sequence* (see Figure 31.2). This sequence can be used whenever participants suffer from any undesired affective state.

ART heavily relies on an intensive training regimen in which participants practice the whole ART sequence for 20–30 minutes daily and additionally engage in at least three short exercises (for 3 seconds to 3 minutes) over a period of at least 6 weeks. To support this practice, participants receive worksheets, handouts, and an information booklet during the training. Additionally, they receive specific audio files that guide them through the exercises of the ART sequence, and a set of 140 text messages is automatically sent to participants throughout the training. After participants have mastered the skills, the focus shifts to practicing in daily life the learned skills to cope successfully with challenging situations.

Muscle and Breathing Relaxation

Every skills building module starts with a short psychoeducation that explains how different areas of the brain may mutually activate each other, thus forming *neuroaffective vicious cycles* that can override processes responsible for down-regulating undesired affective states. For example, when the amygdala is activated by a situation that is interpreted as a potential threat, it initiates physiological changes such as an increase in muscle tension and an acceleration of breathing. Because such changes have occurred in the past when encountering threatening situations, the muscle tension and rapid breathing themselves may in turn have become danger signals to the amygdala (i.e., classical conditioning or the concept of *somatic markers;* Damasio, 2000). As a result, a vicious cycle may develop in which the activation of the amygdala causes increased muscle tension and rapid breathing, which in turn causes further activation of the amygdala and even more muscle tension and further rapid breathing (for details on how somatic processes are integrated into information processing relevant for emotion elicitation and maintenance, see Stemmler, 2004; Teasdale & Barnard, 1993). When participants realize that they are experiencing stress or negative affective states, they can break through these vicious cycles by consciously relaxing their muscles and purposefully calming their breathing. By engaging in these exercises, they can reduce activation of the sympathetic system, the limbic system, and the brainstem (Porges, 2007).

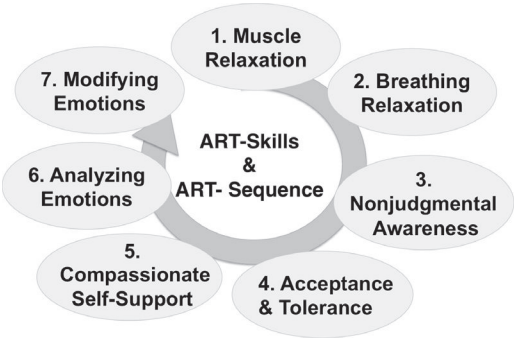

FIGURE 31.2. ART skills and ART sequence. This is the prescriptive model of affect regulation as it is presented to participants. It includes the same skills as the ART model of adaptive affect regulation (Figure 31.1); however, for didactic purposes, it presents them in a more concrete fashion, providing a step-by-step guideline for coping with undesired affective states.

After this vicious cycle has been introduced and discussed with participants, we introduce participants to progressive muscle relaxation techniques (Jacobson, 1964) combined with a simple exercise to calm breathing. Studies have consistently shown the effectiveness of muscle and breathing relaxation in the treatment of a variety of mental and physical disorders (e.g., Öst, 1987; Conrad & Roth, 2007). Additionally, with reduced limbic activity through breathing and muscle relaxation, a shift can occur that changes the focus from primarily amygdala-driven limbic- and brainstem-focused responses back to more prefrontal, cortical responses that are reflective rather than reactive (Arnsten, 2009). Thus, reducing psychophysiological arousal with the help of muscle and breathing relaxation facilitates the subsequent use of techniques requiring more cognitive resources.

Nonjudgmental Awareness

When the amygdala detects potential threats, it alerts the prefrontal cortex (PFC),

which then focuses attention and conscious processing on threat-relevant stimuli (LeDoux, 2012). Such effortful processing aims to more validly evaluate potential threats and determine behavioral responses most likely to be effective. However, if this processing does not lead to a solution of the problem, an ongoing focus on aversive experiences may lead to an ongoing activation of the amygdala. This activation may cause the PFC to focus even more attention on the problem (Amaral, Price, Pitkänen, & Carmichael, 1992; Gray & McNaughton, 2000; Vuilleumier, Richardson, Armony, Driver, & Dolan, 2004), thus potentially resulting in a cyclical reaction of even more intense processing of problematic stimuli and further activation in the amygdala. In attempt to prevent such a negative cycle, some individuals try to rigidly suppress "negative" thinking (Ottenbreit & Dobson, 2004). However, this strategy is likely to have a paradoxical effect of increasing the intensity of negative thinking instead of reducing it (Wenzlaff & Wegner, 2000). Thus, instead of fighting negative thinking, we encourage participants simply to observe the situation and their affective states without interpreting, judging, or reacting. This kind of mindfulness-based affect labeling has been shown to disrupt amygdala activity (Lieberman et al., 2007) and to be effective as a reappraisal or distraction in the regulation of negative affect (Lieberman, Inagaki, Tabibnia, & Crockett, 2011).

When applying this technique as ART Skill 3, participants start by switching from regulating their breathing to simply observing it. Then, they start focusing on their sensual experiences (sensations in the body, sounds, odors, and visual perceptions); their thoughts ("What is the next thought that comes to mind?"); and their current salient wishes, desires, and goals ("If everything was going exactly according to your wishes, where would you be now? What would you be doing now? Imagine this perfect scene. . . . Briefly name the wish or goal expressed in this image"). At the end of this sequence, participants are invited to be aware of their affective states without judging them. Instead, they are instructed to (1) find a matter-of-fact label for this emotion (e.g., anger), (2) rate its intensity on a scale from 1 to 10, and (3) identify where the emotion is felt in the body. Finally, participants are invited to check for additional feelings that might lie behind the most prominent one and repeat the same three-step process on them. This technique of becoming aware of one's affective states in nonjudgmental ways aims to recruit cognitive resources to facilitate effective affect regulation (Gyurak & Etkin, this volume; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003). Evidence for the efficacy of nonjudgmental awareness includes studies on the effectiveness of mindfulness-based interventions (Farb et al., this volume).

Acceptance and Tolerance of Affective States

Challenging affective states, such as fear or anger, constitute threats to the goals of feeling good and of being in control (Grawe, 2006). Reponses of the amygdala toward such threats likely include the activation of areas responsible for avoidance motivation (Grawe, 2006). Under the influence of avoidance goals (Grawe, 2002, 2006), people may rigidly focus on eliminating the "threatening" affective states. However, the goal of getting rid of an undesired emotion is often difficult to attain. The emotion-generating system often acts autonomously of the areas of the brain that initiate consciously controlled behavior (Amaral et al., 1992; see also the concept of core valuations outlined by Ochsner & Gross, this volume). Such a design can be hypothesized to have provided a significant evolutionary advantage to our ancestors. If negative affective states could simply be switched off through conscious control, a strong temptation to simple down-regulate unpleasant emotions without going through the trouble of engaging in the behavior the emotion is supposed to facilitate could interfere with the emotions cueing adaptive behavior. Additionally, emotional reactions often cause significant changes in the body that take some time to dissipate and, as long as they are present, may impede the ability to make emotional changes (Teasdale & Barnard, 1993). It can also been hypothesized that areas such as the ventromedial PFC and the anterior cingulate cortex, which are assumed to help in down-regulation of undesired emotions under the influence of dorsal or lateral areas of the PFC, may get "hijacked" by strong emotions

in a way that leads to ruminative processing of emotion-related information and impedes effective top-down emotion regulation (Johnstone & Walter, this volume).

In summary, for various reasons, it is often very difficult to control affective states consciously. Thus, rigid attempts to shut down an undesired affective state instantly through sheer willpower may fail. Such unsuccessful attempts at attaining the salient goal of eliminating an undesired affective state will likely cue amygdala activation and increase the salience of the goal (Martin, Tesser, & McIntosh, 1993; Rothermund, 2003). Because of the resulting increase in negative arousal, the likelihood of attaining an increasingly salient goal is significantly reduced. Thus, a vicious cycle may develop in which fighting against the emotion only makes it stronger. To prevent this vicious cycle, developing the ability to accept and tolerate negative affective states is important (at least as long as such states cannot be changed). To enhance acceptance and tolerance skills, ART first works to help participants find a helpful definition of acceptance/tolerance and to understand why these skills are important. Then, participants develop their personal, five-step Acceptance and Tolerance Plan, which includes the following steps: (1) Consciously decide to work on accepting the emotion; (2) strengthen this decision by finding at least one reason why acceptance is helpful; (3) work to see affective states as allies providing important information and supporting behavior that might be used for effective coping with challenging situations (e.g., for an adaptive conceptualization of affective states, see Cosmides & Tooby, 2000); (4) become aware of your ability to tolerate negative affective states for a certain period of time; and (5) realize that affective states are usually impermanent experiences that do not last forever and are likely to fade away (or at least decrease in intensity).

At the end of the Acceptance and Tolerance Plan, participants devise a Personal Acceptance and Tolerance Statement, such as the following, to use as self-talk, until they feel that they can allow affective states to exist:

> "I will work to accept this unpleasant emotion because this is the smartest thing to do as long as I cannot change it; I know that this emotion wants to provide me with very relevant information; it wants to point out that . . . ; it aims to help me by enhancing my ability to . . . ; I also know that I have experienced many unpleasant emotions in the past and have always been able to get through this in one way or another; finally, I know that this emotion will not last forever, I will only have to tolerate it for so long."

In the ART sequence, this kind of acceptance-enhancing self-talk is applied to affective states that have been perceived nonjudgmentally at the end of ART Skill 3. Preliminary evidence for the therapeutic potential for the skills of acceptance and tolerance comes from research on the relevance of avoidance for the development and maintenance of mental disorders (e.g., Berking, Neacsiu, Comtois, & Linehan, 2009), from studies demonstrating the effectiveness of mindfulness-based approaches (Farb et al., this volume), and from acceptance and commitment therapy, in which acceptance of negative affective states and willingness to experience them is one of the primary aims of therapy (Hayes, Luoma, Bond, Masuda, & Lillis, 2006).

Compassionate Self-Support in Emotionally Challenging Situations

The next vicious cycle starts when someone who feels stressed or upset begins to self-blame or self-criticize. That person might think, "I can't do anything right. I am a complete failure. Something is wrong with me." These statements are self-inflicted attacks that threaten the need to feel valued (Grawe, 2002), cueing additional negative affective states (e.g., anger, shame, guilt) associated with further activation of the amygdala (Longe et al., 2010). The activation of the amygdala through self-criticism and additional negative feelings increases the stress response, likely leading to more negative feelings, which in turn lead the person to be even more self-critical. Activating a compassionate attitude toward oneself and engaging in self-supportive behaviors is an effective strategy to preclude this vicious cycle (Gilbert, 2011). ART participants learn how to provide compassionate self-support

by imagining themselves in a burdensome situation accompanied by negative feelings. While reexperiencing these feelings, they learn to self-assess from the perspective of a kind and caring (albeit somewhat distant) inner observer. They are instructed to "let the feeling of compassion toward themselves arise from within," which creates a strong and warm feeling of self-empathy, accompanied by the desire to help and to end their suffering (Weissman & Weissman, 1996). The observing part should then approach the suffering part of the participant, supporting and comforting that part by normalizing the negative affective states, providing encouragement, and maybe adding a physical gesture of compassion (e.g., giving oneself an imaginary hug). Preliminary evidence for the effectiveness of interventions that enhance compassionate self-support has been found in several empirical studies (Hofman, Grossman, & Hinton, 2011).

Constructive Analysis of Affective States

Under stress, the amygdala activates processes that release stress hormones into the brain. An excessive release of such hormones can impair cognitive processing in other brain areas, including the PFC and the hippocampus (Huether, 1998; LeDoux, 2012), which play important roles in the analysis of affective states. The ability to identify the causes of one's current feelings correctly leads to a greater sense of self-mastery and control over one's affective states. If through weakened prefrontal and hippocampal functioning, the ability to self-analyze one's affective states is diminished, a vicious cycle develops in which the loss of orientation and control further activates the amygdala, which in turn perpetuates the cycle. Thus, maintaining or regaining a sense of mastery and control over one's affective states in stressful situations helps prevent the escalation of negative affect.

In ART Skill 6, participants are trained to utilize the *Analyzing Affective States Worksheet* (depicted in Figure 31.3) to enhance the ability to identify correctly the causes of one's current emotions. The worksheet is based on an integrative model of how affective states are cued and maintained. It focuses on emotions and feelings as crucial affective states, but we broadly define these terms in the training (to avoid burdening patients with the academic need for clear classifications). Thus, these terms include other affective states, such as stress responses or moods. The general model of how emotions (i.e., affective states) are elicited includes external or internal events that cue the emotion; attention deployment toward and appraisals of these events; needs, wishes, and goals that are relevant for these appraisals; secondary emotions (Greenberg, 2004) cued by appraising the primary emotion in a certain way; and body sensations, motivational impulses, and behavior associated with the emotion, all of which provide feedback into the emotion generating system and thus affect the subsequent course of the emotion. To clarify the course of subsequent action, the worksheet ends with a summary of the short- and long-term advantages and disadvantages of the emotion.

ART trainers explain the model and help participants to apply it to their own affective states. Eventually, the skill will be included in the ART sequence in the form of the caring inner observer from ART Skill 5, who guides the suffering part of the client through the analysis ("What situation prompted this feeling?"; "How did you interpret and evaluate the situation?"; "What was at stake in this situation?"). Partly based on the finding that insight-oriented psychological treatments have been shown to be effective (e.g., Gibbons, Crits-Christoph, Barber, & Schamberger, 2007) and our own clinical experience, we hypothesize that simply understanding challenging affective states with the help of a model normalizes and hence validates such states; that the impression of having understood the problem cues a general feeling of control and hope; and that offers of specific and promising ways to cope successfully with undesired states significantly contributes to the (short-term) reduction and/or acceptance of undesired affective states.

Effective Modification of Affective States

ART Skill 7 aims to empower participants to actively modify quality, intensity, or duration of an undesired emotion and restore their sense of control. This skill, which pro-

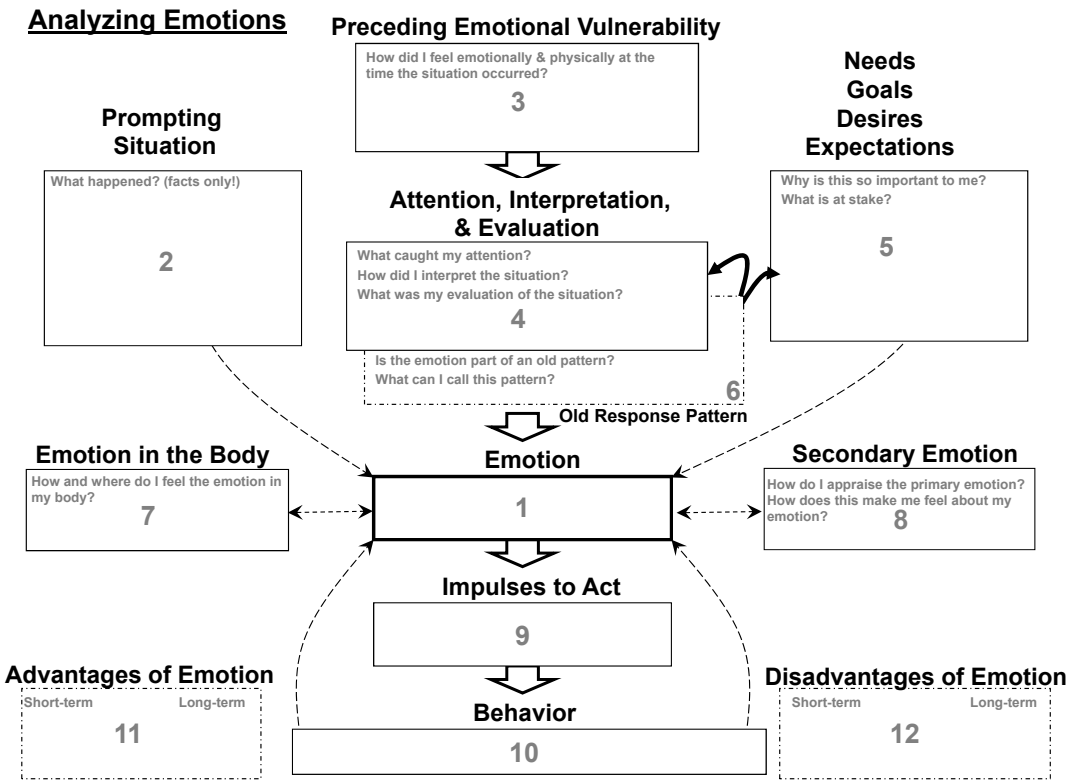

FIGURE 31.3. The Analyzing Affective States Worksheet. To reduce the complexity wherever possible, the distinction between affect and emotions is not addressed in the training. Instead, the terms *emotions* and *feelings* are used in a very broad sense (e.g., with regard to feeling stressed out and relative to various mood states). Although the worksheet is fairly complex, participants who have been trained intensively in its use have rated it as one of the most helpful components of ART. Typical explanations for its popularity include "It gets everything I somehow felt was relevant for how I feel on one sheet of paper."

vides a step-by-step process to modify affective states, is based on the general problem-solving model (D'Zurilla & Nezu, 2010) and utilizes the *Modification of Affective States Worksheet* depicted in Figure 31.4.

Based on the long- and short-term advantages and disadvantages of the emotion summary from the Analyzing Affective States Worksheet, the Modifying Affective States Worksheet first requires participants to set as a goal how they would rather feel. Then, for each relevant maintaining factor (i.e., each box of the worksheet), participants brainstorm how changes in these areas could help cue the target emotion. After participants have identified as many helpful ideas as possible for each box, they select the most promising ideas to plan specific behaviors that cue the target emotion. Then, they put the plan into practice and evaluate the outcome. With regard to the latter, patients are also taught a plan for how to deal effectively with unsuccessful modification attempts (see bottom right-hand corner of Figure 31.4), because such failures often lead to a complete disengagement from functional regulation endeavors and the use instead of impulsive strategies that might effectively improve one's mood in the short run but are associated with negative long-term outcomes (e.g., engaging in avoidance, substance use, worry, depressogenic thinking).

After the model is introduced and practiced with a couple of examples, Skill 7 is included in the ART sequence. At this point, the compassionate, helping part of

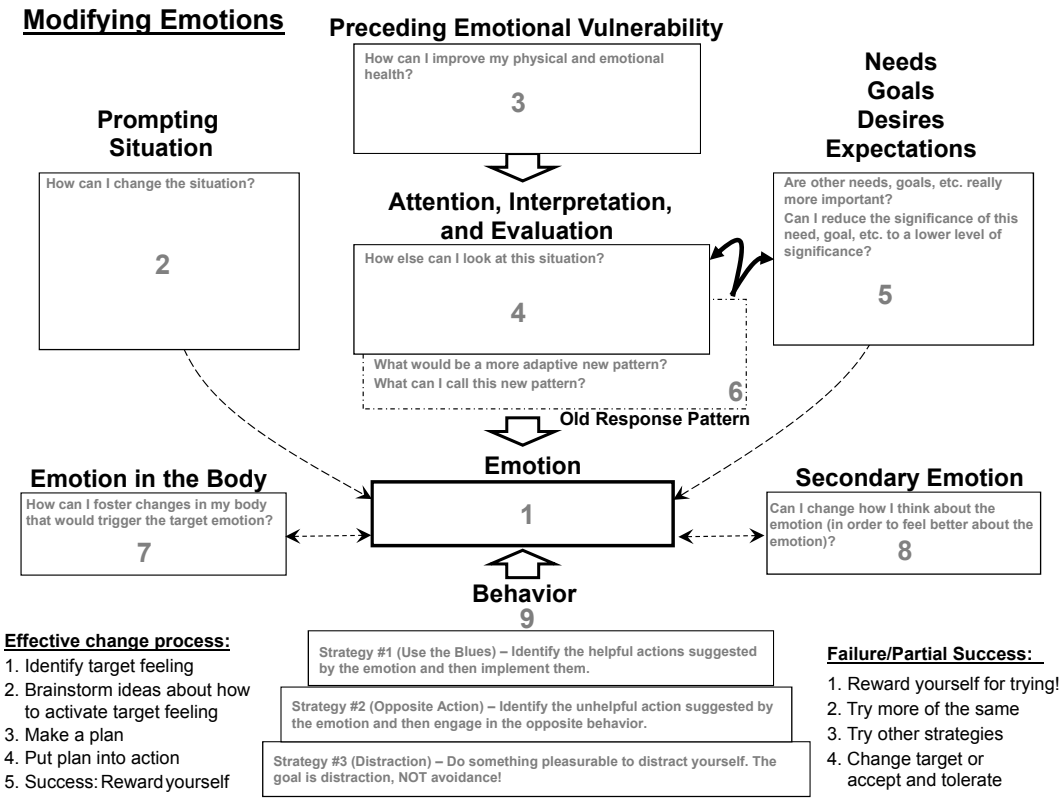

FIGURE 31.4. The Modifying Affective States Worksheet. When using distraction as a behavior-oriented modification strategy, it is explained to participants that the same behavior can be considered (dysfunctional) avoidance or distraction, which can be a very effective modification strategy, depending on one's willingness to experience the affective state to be regulated ("As long as we could also face the undesired state we are free to use distraction"); ART facilitates the adaptive use of distraction by focusing on experiencing and accepting unwanted affective states in ART Skills 3 and 4 prior to focusing on modification. "Opposite Action": See Linehan (1993).

the self (established in Skill 5) guides the vulnerable part of the self (also established in Skill 5) in modifying negative affective states ("How do you actually want to feel in this situation?"; "What is your target emotion?"; "How could you change the situation to achieve your target emotion?"). As such, ART Skill 7 builds on the other skills of the ART sequence and utilizes the general problem-solving framework to modify undesired affective states.

Efficacy of ART

Research on the effectiveness of ART is still in the early stages. However, several studies have been conducted in a variety of psychotherapeutic settings and early results are promising. One published study (Berking, Wupperman, et al., 2008) involved 289 patients from a mental health hospital. The patients all received 6 weeks of CBT supplemented with additional multidisciplinary treatment as necessary. A randomly selected subgroup of patients was offered the option to replace four treatment sessions of CBT per week with an abbreviated version of ART during a 3-week period. Results showed that at the end of the 6-week treatment, patients who had participated in ART displayed significantly greater improvement of affect regulation skills than patients who had received CBT only. Additionally, patients participat-

ing in ART showed a significantly greater decrease in symptoms of depression and negative affect, as well as greater treatment gains in positive affect.

Similar results were found during a recent, completely randomized controlled trial with a sample of over 400 inpatient participants who met diagnostic criteria for MDD. In this study, the effectiveness of CBT was significantly improved by supplementing traditional CBT with the ART program (Berking et al., 2013). Additional gains in the CBT + ART condition were particularly strong with regard to the abilities of acceptance, tolerance, compassionate self-support, and modification. All these strategies have been hypothesized to be particularly important for mental health (see the section on empirical evidence for the ART model). In another study (Berking et al., 2010), a group of police officers participated in ART. Prior to the training, their affect regulation skills were significantly lower than those of a control sample. After participation in the training, the police officers' affect regulation skills had increased to the point that they no longer differed from the controls. These effects did not occur in the control condition. Moreover, in a recent quasi-experimental study, a combination of ART and CBT was effective for treating patients with medically unexplained somatic symptoms (Gottschalk & Rief, 2012).

Finally, in experimental studies with clinical and nonclinical samples, we found evidence for the short-term effectiveness of specific ART skills. For these studies, we developed an experimental paradigm in which health-relevant affective states are repeatedly induced with the help of a combination of music and the Velten Mood Induction Procedure (1968). After each induction phase, participants are instructed to use affect regulation skills, such as the ones included in the ART model. Before and after each induction and regulation, participants rate the intensity and acceptability of the affective state. To control for order effects, the complete permutation of all possible combinations of strategies and control conditions is implemented over the total sample. Using this paradigm in a healthy sample of undergraduates and a clinically depressed sample, we found that in both samples the ART-based audio instructions on how to cope with an experimentally induced depressive mood were more effective in reducing the mood's intensity than the spontaneous regulation control condition (Fischer, Kirchner, Kandl, Hiller, & Berking, 2012; Kowalsky et al., 2013). Although all these studies provide significant support for the assumed efficacy of ART, additional studies are dearly needed to clarify further the populations that benefit from ART, and whether and to what extent these benefits exceed those of other bona fide treatments.

Future Directions for ART

Several limitations in our basic research on affect regulation, ART as a clinical intervention, and our studies evaluating the efficacy of ART need to be addressed in future research. For example, basic research initiated by our group has made extensive use of self-reports in an area that, at least in part, is difficult to assess through introspection (e.g., the ability to identify one's emotions correctly). Moreover, the ERSQ asks participants whether they can successfully apply certain skills to their feelings or emotions. Because a depressed patient might refer to different emotions than would a participant suffering from an anxiety disorder, the instrument refers to different affective states in different participants, and the extent that these particular difficulties are valid indicators of their *general* emotion regulation skills is unclear. Additionally, many assumptions in our model (e.g., that the application of promising affect regulation skills itself can contribute to short-term mood deterioration) are largely grounded in theory and/or clinical experience, and need to be investigated in more detail empirically.

With regard to ART as a clinical intervention, it is noteworthy that in spite of the obvious advantages of transdiagnostic interventions (e.g., easier to disseminate in a cost-effective group setting), the disadvantages of potentially neglecting relevant characteristics of particular disorders have to be carefully considered. Moreover, from our clinical work, we developed the impression that using theories and findings from the neurosciences to derive health-relevant affect regulation skills helps to engage participants in the systematic training routine.

However, we also learned that finding the right balance between a clinically helpful and a scientifically valid neuroaffective model of affect regulation is often difficult to attain. For example, anthropomorphizing relevant brain areas (e.g., by using the expression "The amygdala only wants to protect you") can be a very effective psychotherapeutic intervention, but the approach is inadequate in the context of pure scientific explanations. Additionally, we have to acknowledge that remaining up-to-date with the latest findings in the rapidly developing field of the neurosciences, while simultaneously conducting large randomized clinical trials, represents a remarkable challenge. During the time it takes to develop a treatment program, write a treatment manual, raise the funds for efficacy trials, and conduct and publish the results of those trials, neuroscientific findings and theories used for developing the treatment program are likely to be out of date.

With regard to efficacy research, it is of note that in clinical populations we have investigated ART exclusively as an adjunctive intervention, which is in line with our assumption that the characteristics of specific disorders must also be considered when offering transdiagnostic interventions enhancing general emotion regulation skills. However, the current lack of studies evaluating ART as a stand-alone intervention also prevents us from clarifying which disorders can be treated effectively with interventions focusing exclusively on enhancing such skills. We should also note that because of the lack of multiple assessments and untreated control conditions, we have not yet been able to conduct meaningful mediation analyses clarifying the extent to which ART exerts positive effects through improving affect regulation skills as hypothesized. Finally, it is noteworthy that the current ART program teaches a broad range of affect regulation skills, which takes about 18 hours to complete. From a theoretical and a treatment dissemination perspective, an essential subsequent step will be to clarify whether all parts of the program are necessary to obtain the desired effects.

To overcome these limitations, we have initiated several research projects. For example, to address the measurement problems, we have recently developed and validated a modified version of the ERSQ called the Affect Regulation Skills Questionnaire (ARSQ; Ebert et al., 2013), which assesses affect regulation skills separately for various affective states.[1] This measure helps to clarify whether individuals differ in their affect regulation skills across different affective states, and it helps to identify which skills for which affective states are most important for maintaining or restoring mental health.

Currently we are also limited in our attempts to use findings from the affective neurosciences to improve psychotherapeutic interventions. We acknowledge that we are unlikely to become cutting-edge experts in two research fields as vast as the neurosciences and treatment development and evaluation. Thus, we are currently looking for neuroscience experts who share our interest in translational research. By collaborating with experts in the neurosciences, we aim to enhance further the validity of the translational approach used in ART and thereby bridge the current gap between basic research and clinical application.

With regard to the need for more efficacy trials, in two, large, multicenter trials we are currently investigating whether ART can also be used as an effective stand-alone treatment for MDD and/or binge-eating disorder (BED). In these trials we use experimental, biological, observer-based, and ambulatory momentary assessments of affect regulation skills, in addition to retrospective self-report measures. Moreover, through multiple assessments and designs that include an untreated control condition, we will be able to conduct meaningful mediation analyses. To clarify whether all parts of the program are necessary to obtain the desired effects, we are currently conducting a dismantling study, in which we compare an ART version that focuses exclusively on acceptance and versions that focus exclusively on modification or on both acceptance and modification. Additionally, we acquired funding for a large, multicenter trial in which the efficacy of CBT + ART is compared with traditional CBT in a sample of patients suffering from medically unexplained somatic symptoms.

We are also exploring technology enhancements to ART. Text messages have been used since 2004 to support participants in their daily skills practice. Since then, numerous innovations have been presented in the

rapidly developing field of computerized psychological treatment. For example, we are currently finalizing an interactive online version of ART, as well as several serious games that can be used to practice ART skills in a leisurely fashion. We hope that these endeavors will help individuals in need of more effective affect regulation to acquire and continuously practice the most effective strategies and thereby overcome their mental health problems and thereby improve their well-being and satisfaction with life.

Note

1. An English version of the ARSQ can be obtained from Matthias Berking.

References

Amaral, D. G., Price J. L., Pitkänen, A., & Carmichael, S. T. (1992). Anatomical organization of the primate amygdaloid complex. In J. P. Aggleton (Ed.), *The amygdala: Neurobiological aspects of emotion, memory and mental dysfunction* (pp. 1–66). New York: Wiley-Liss.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Arnsten, A. F. T. (2009). Stress signaling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience, 10*(6), 410–422.

Beck, J. S. (1995). *Cognitive therapy: Basics and beyond*. New York: Guilford Press.

Berking, M. (2010). *Training emotionaler Kompetenzen* (2nd ed.). Berlin/Heidelberg: Springer-Verlag.

Berking, M., Ebert, D., Cuijpers, P., & Hofmann, S. (2013). Emotion-regulation skills training enhances the efficacy of cognitive behavioral therapy for major depressive disorder. *Psychotherapy and Psychosomatics, 82*, 234–245.

Berking, M., Margraf, M., Ebert, D., Wuppermann, P., Hofmann, S., & Junghanns, K. (2011). Emotion regulation skills as a predictor of relapse during and after treatment of alcohol dependence. *Journal of Consulting and Clinical Psychology, 79*(3), 307–318.

Berking, M., Meier, C., & Wupperman, P. (2010). Enhancing emotion-regulation skills in police officers: Results of a pilot controlled study. *Behavior Therapy, 41*(3), 329–339.

Berking, M., Neacsiu, A., Comtois, K. A., & Linehan, M. M. (2009). The impact of experiential avoidance on the reduction of depression in treatment for borderline personality disorder. *Behaviour Research and Therapy, 47*(8), 663–670.

Berking, M., Orth, U., Wupperman, P., Meier, L., & Caspar, F. (2008). Prospective effects of emotion regulation on emotional adjustment. *Journal of Counseling Psychology, 55*(4), 485–494.

Berking, M., Poppe, C., Luhmann, M., Wuppermann, P., Jaggi, V., & Seifritz, E. (2012). Emotion-regulation skills and psychopathology: Is the ability to modify one's negative emotions the ultimate pathway by which all other skills affect symptoms of mental disorders? *Journal of Behavior Therapy and Experimental Psychiatry, 43*, 931–937.

Berking, M., & Whitley, B. (2013). *Affect regulation training*. New York: Springer.

Berking, M., Wirtz, C., Svaldi, J., & Hofmann, S. (under review). Emotion regulation skills predict symptoms of depression over five years in individuals at risk for depression. *Behaviour Research and Therapy*.

Berking, M., & Wupperman, P. (2012). Emotion regulation and mental health: Recent findings, current challenges and future directions. *Current Opinion in Psychiatry, 25*(2), 128–134.

Berking, M., Wupperman, P., Reichardt, A., Pejic, T., Dippel, A., & Znoj, H. (2008). Emotion-regulation skills as a treatment target in psychotherapy. *Behaviour Research and Therapy, 46*(11), 1230–1237.

Berking, M., & Znoj, H. (2008). Entwicklung und Validierung eines Fragebogens zur standardisierten Selbsteinschätzung emotionaler Kompetenzen (SEK-27) [Development and validation of a standardized self-report measure assessing emotional competencies]. *Zeitschrift für Psychiatrie Psychologie und Psychotherapie, 56*(2), 141–153.

Bogdan, R., & Pizzagalli, D. A. (2006). Acute stress reduces reward responsiveness: Implications for depression. *Biological Psychiatry, 60*(10), 1147–1154.

Bonanno, G. A., Papa, A., Lalande, K., Westphal, M., & Coifman, K. (2004). The importance of being flexible: The ability to both enhance and suppress emotional expression predicts long-term adjustment. *Psychological Science, 15*(7), 482–487.

Catanzaro, S. J., & Greenwood, G. (1994). Expectancies for negative mood regulation, coping, and dysphoria among college students.

Journal of Counseling Psychology, 41(1), 34–44.

Conrad, A., & Roth, W. T. (2007). Muscle relaxation therapy for anxiety disorders: It works but how? *Journal of Anxiety Disorders, 21*, 243–264.

Cosmides, L., & Tooby, J. (2000). Evolutionary psychology and the emotions. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of emotions* (2nd ed., pp. 91–115). New York: Guilford Press.

Damasio, A. (2000). *The feeling of what happens: Body and emotion in the making of consciousness*. New York: Harcourt Brace.

Dingemans, A. E., Martijn, C., Jansen, A., & Furth, E. F. (2009). The effect of suppressing negative emotions on eating behavior in binge eating disorder. *Appetite, 52*, 51–57.

Duckworth, A. L., Steen, T. A., & Seligman, M. E. P. (2005). Positive psychology in clinical practice. *Annual Review of Clinical Psychology, 1*, 629–651.

D'Zurilla, T. J., & Nezu, A. M. (2010). Problem-solving therapy. In K. S. Dobson (Ed.), *Handbook of cognitive behavioral therapies* (Vol. 39, pp. 197–226). New York: Guilford Press.

Ebert, D., Christ, O., & Berking, M. (2013). Entwicklung und Validierung eines Fragebogens zur emotionsspezifischen Selbsteinschätzung emotionaler Kompetenzen (SEK-ES) [Development and validation of a self-report instrument for the assessment of affect-specific regulation skills]. *Diagnostica, 59*(1), 17–32.

Farb, N. A. S., Anderson, A. K., Irving, J. A., & Segal, Z. V. (this volume, 2014). Mindfuless interventions and emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 548–568). New York: Guilford Press.

Feldman-Barrett, L., Gross, J., Christensen, T. C., & Benvenuto, M. (2001). Knowing what you're feeling and knowing what to do about it: Mapping the relation between emotion differentiation and emotion regulation. *Cognition and Emotion, 15*(6), 713–724.

Feldner, M. T., Zvolensky, M. J., Stickle, T. R., Bonn-Miller, M. O., & Leen-Feldner, E. W. (2006). Anxiety sensitivity-physical concerns as a moderator of the emotional consequences of emotion suppression during biological challenge: An experimental test using individual growth curve analysis. *Behaviour Research and Therapy, 44*(2), 249–272.

Fischer, A., Kirchner, M., Kandl, M., Hiller, W., & Berking, M. (2012, May). *Effektivität von Emotionsregulationsstrategien zur Reduktion dysphorischer Stimmung bei Depression* [Effects of emotion regulation strategies on the reduction of depressive mood in depressed patients]. Poster presented at the 30th annual conference of the Section Clinical Psychology and Psychotherapy of the German Psychological Association, Luxembourg.

Foa, E. B., Riggs, D. S., Massie, E. D., & Yarczower, M. (1995). The impact of fear activation and anger on the efficacy of exposure treatment for posttraumatic stress disorder. *Behavior Therapy, 26*, 487–499.

Gibb, B. E., Beevers, C. G., Andover, M. S., & Holleran, K. (2006). The hopelessness theory of depression: A prospective multi-wave test of the vulnerability–stress hypothesis. *Cognitive Therapy and Research, 30*(6), 763–772.

Gibbons, M. B. C., Crits-Christoph, P., Barber, J. P., & Schamberger, M. E. (2007). Insight in psychotherapy: A review of empirical literature. In L. G. Castonguay & C. E. Hill (Eds.), *Insight in psychotherapy* (pp. 143–165). Washington, DC: American Psychological Association.

Gilbert, P. (2011). *Compassion focused therapy: The distinctive features*. London: Routledge.

Gottman, J., & DeClaire, J. (1997). *The heart of parenting: Raising an emotionally intelligent child*. New York: Simon & Schuster.

Gottschalk, J.-M., & Rief, W. (2012). Psychotherapeutische Ansätze für Patienten mit somatoformen Störungen [Psychotherapeutic treatments for patients with somatoform disorders]. *Der Nervenarzt, 83*, 1115–1127.

Grawe, K. (2002). *Psychological therapy*. Cambridge, MA: Hogrefe & Huber.

Grawe, K. (2006). *Neuropsychotherapy*. New York: Psychology Press.

Gray, J. A., & McNaughton, N. (2000). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system* (2nd ed.). Oxford, UK: Oxford University Press.

Greenberg, L. S. (2004). Emotion-focused therapy. *Clinical Psychology and Psychotherapy, 11*(1), 1–2.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*(3), 271–299.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gyurak, A., & Etkin, A. (this volume, 2014). A neurobiological model of implicit and explicit emotion regulation. In J. J. Gross (Ed.),

Handbook of emotion regulation (2nd ed., pp. 76–90). New York: Guilford Press.

Hariri, A. R., Mattay, V. S., Tessitore, A., Fera, F., & Weinberger, D. R. (2003). Neocortical modulation of the amygdala response to fearful stimuli. *Biological Psychiatry, 53*, 494–501.

Hayes, S. C., Luoma, J. B., Bond, F. W., Masuda, A., & Lillis, J. (2006). Acceptance and commitment therapy: Model, processes and outcomes. *Behaviour Research and Therapy, 44*(1), 1–25.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy.* New York: Guilford Press.

Hofmann, S. G., Grossman, P., & Hinton, D. E. (2011). Loving-kindness and compassion meditation: Potential for psychological interventions. *Clinical Psychology Review, 31*(7), 1126–1132.

Hofmann, S. G., Sawyer, A. T., Fang, A., & Asnaani, A. (2012). Emotion dysregulation model of mood and anxiety disorders. *Depression and Anxiety, 29*, 409–412.

Huether, G. (1998). Stress and the adaptive self-organization of neuronal connectivity during early childhood. *International Journal of Developmental Neuroscience, 16*, 297–306.

Jacobson, E. (1964). *Anxiety and tension control: A physiologic approach.* Philadelphia: Lippincott.

Johnstone, T., & Walter, H. (this volume, 2014). In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 58–75). New York: Guilford Press.

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*(6), 617–627.

Koole, S. L., & Rothermund, K. (2011). "I feel better but I don't know why": The psychology of implicit emotion regulation. *Cognition and Emotion, 25*(3), 389–399.

Kowalsky, J., Schwarz, J., Tauch, C., Ruppert, M., Wert, H., & Berking, M. (2013, May). *Effektivität von Emotionsregulationsstrategien—eine experimentelle Untersuchung* [Efficacy of emotion regulation strategies—an experimental investigation]. Poster presented at the 31th annual conference of the Section Clinical Psychology and Psychotherapy of the German Psychological Association in Trier.

Lane, R. D., Sechrest, L., Reidel, R., Weldon, V., Kaszniak, A. W., & Schwartz, G. E. (1996). Impaired verbal and nonverbal emotion recognition in alexithymia. *Psychosomatic Medicine, 58*, 203–210.

Larsen, R. J. (2000). Toward a science of mood regulation. *Psychological Inquiry, 11*(3) 129–141.

Lazarus, R. S. (1991). Emotion and adaptation. In L. A. Pervin (Ed.), *Handbook of personality theory and research* (Vol. 21, pp. 609–637). Oxford, UK: Oxford University Press.

Leahy, R. L. (2002). A model of emotional schemas. *Cognitive and Behavioral Practice, 9*(3), 177–190.

LeDoux, J. E. (2012). Evolution of human emotion: A view through fear. *Progress in Brain Research, 195*, 431–442.

Levitt, J. T., Brown, T. A., Orsillo, S. M., & Barlow, D. H. (2004). The effects of acceptance versus suppression of emotion on subjective and psychophysiological response to carbon dioxide challenge in patients with panic disorder. *Behavior Therapy, 35*(4), 747–766.

Lieberman, M. D., Eisenberger, N. I., Crockett, M. J., Tom, S. M., Pfeifer, J. H., & Way, B. M. (2007). Putting feelings into words: Affect labeling disrupts amygdala activity in response to affective stimuli. *Psychological Science, 18*(5), 421–428.

Lieberman, M. D., Inagaki, T. K., Tabibnia, G., & Crockett, M. J. (2011). Subjective responses to emotional stimuli during labeling, reappraisal, and distraction. *Emotion, 11*(3), 468–480.

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder.* New York: Guilford Press.

Longe, O., Maratos, F. A., Gilbert, P., Evans, G., Volker, F., Rockliff, H., et al. (2010). Having a word with yourself: Neural correlates of self-criticism and self-reassurance. *NeuroImage, 49*(2), 1849–1856.

Martin, L. L., Tesser, A., & McIntosh, W. D. (1993). Wanting but not having: The effects of unattained goals on thoughts and feelings. In E. Daniel, M. Wegner, E. James, & W. Pennebaker (Eds.), *Handbook of mental control* (pp. 552–572). New York: Prentice Hall.

Mauss, I. B., & Tamir, M. (this volume, 2014). Emotion goals: How their content, structure, and operation shape emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 361–375). New York: Guilford Press.

Mennin, D. S., & Fresco, D. M. (2009). Emotion regulation as an integrative framework for understanding and treating psychopathology.

In A. M. Kring & D. M. Sloan (Eds.), *Emotion regulation and psychopathology: A transdiagnostic approach to etiology and treatment* (pp. 356–379). New York: Guilford Press.

Mennin, D. S., & Fresco, D. M. (this volume, 2014). Emotion regulation therapy. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 469–490). New York: Guilford Press.

Morrow, J., & Nolen-Hoeksema, S. (1990). Effects of responses to depression on the remediation of depressive affect. *Journal of Personality and Social Psychology, 58*(3), 519–527.

Moses, E. B., & Barlow, D. H. (2006). Current directions in psychological science: Approach for emotional disorders based on emotion science. *Psychological Science, 15*(3), 146–150.

Neasciu, A. D., Bohus, M., & Linehan, M. (this volume, 2014). Dialectical behavir therapy: An intervention for emotion dysregulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 491–507). New York: Guilford Press.

Ochsner, K. N., & Gross, J. J. (this volume, 2014). The neural bases of emotion and emotion regulation: A valuation perspective. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 23–42). New York: Guilford Press.

Öst, L.-G. (1987). Applied relaxation: Description of a coping technique and review of controlled studies. *Behaviour Research and Therapy, 25*(5), 397–409.

Ottenbreit, N. D., & Dobson, K. S. (2004). Avoidance and depression: The construction of the cognitive behavioral avoidance scale. *Behaviour Research and Therapy, 42*, 293–313.

Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology, 74*, 116–143.

Radkovsky, A., McArdle, J., Bockting, C., & Berking, M. (under review). Emotion regulation skills predict subsequent reduction of symptom severity in patients suffering from major depressive disorder: A latent change analysis. *Journal of Consulting and Clinical Psychology*.

Rothermund, K. (2003). Automatic vigilance for task-related information: Perseverance after failure and inhibition after success. *Memory and Cognition, 31*, 343–352.

Saarni, C. (1999). *The development of emotional competence*. New York: Guilford Press.

Salovey, P., & Mayer, J. D. (1990). Emotional intelligence. *Imagination, Cognition, and Personality, 9*, 185–211.

Stemmler, G. (2004). Physiological processes during emotion. In P. Philippot & R. S. Feldman (Eds.), *The regulation of emotion* (pp. 33–70). Mahwah, NJ: Erlbaum.

Swart, M., Kortekaas, R., & Aleman, A. (2009). Dealing with feelings: Characterization of trait alexithymia on emotion regulation strategies and cognitive-emotional processing. *PLoS ONE, 4*(6), e5751.

Teasdale, J. D., & Barnard, P. J. (1993). *Affect, cognition and change: Re-modelling depressive thought*. Hove, UK: Erlbaum.

Van Rijsbergen, G. D., Bockting, C. L. H., Berking, M., Koeter, M. W. J., & Schene, A. H. (2012). Can a one-item mood scale do the trick?: Predicting relapse over 5.5-years in recurrent depression. *PloS ONE, 7*(10), e46796.

Velten, E. (1968). A laboratory task for the induction of mood states. *Behavioral Research and Therapy, 6*, 473–482.

Vuilleumier, P., Richardson, M. P., Armony, J. L., Driver, J., & Dolan, R. J. (2004). Distant influences of amygdala lesion on visual cortical activation during emotional face processing. *Nature Neuroscience, 7*(11), 1271–1278.

Weissman, S., & Weissman, R. (1996). *Meditation, compassion and lovingkindess. An approach to Vipassana practice*. York Beach, ME: Weiser.

Wenzlaff, R. M., & Wegner, D. M. (2000). Thought suppression. *Annual Review of Psychology, 51*, 59–91.

Werner, K., & Gross, J. J. (2010). Emotion regulation and psychopathology: A conceptual framework. In A. M. Kring & D. M. Sloan (Eds.), *Emotion regulation and psychopathology: A transdiagnostic approach to etiology and treatment* (pp. 13–37). New York: Guilford Press.

Wupperman, P., Marlatt, G. A., Cunningham, A., Bowen, S., Berking, M., Mulvihill-Rivera, N., et al. (2012). Mindfulness and modification therapy for behavioral dysregulation: Results from a pilot study targeting alcohol use and aggression in women. *Journal of Clinical Psychology, 67*, 1–17.

Wupperman, P., Neumann, C. S., & Axelrod, S. R. (2008). Do deficits in mindfulness underlie borderline personality features and core difficulties? *Journal of Personality Disorders, 22*, 466–482.

CHAPTER 32

Mindfulness Interventions and Emotion Regulation

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Mindfulness training (MT) represents the secular adaptation of Buddhist contemplative practices aimed to reduce suffering and foster well-being. Over the past three decades, MT interventions have been increasingly recognized for their ability to reduce psychological distress across a variety of clinical disorders (Baer, 2003; Bohlmeijer, Prenger, Taal, & Cuijpers, 2010; Grossman, Niemann, Schmidt, & Walach, 2004). They have been particularly effective in promoting adaptive emotion regulation in affective disorders such as anxiety and depression (Hofmann, Sawyer, Witt, & Oh, 2010; Piet & Hougaard, 2011), and appear to promote self-regulated behavior and positive emotional states (Brown, Ryan, & Creswell, 2007). Our understanding of mindful emotion regulation is imperfect, but it is supported by a recent groundswell of experimental and clinical research (for detailed reviews, see Holzel et al., 2011; Shapiro, Carlson, Astin, & Freedman, 2006).

This chapter outlines the theoretical foundations of mindfulness, the mechanisms by which MT appears to support adaptive emotion regulation, and its most prevalent forms of delivery. We begin by exploring definitions of mindful emotion regulation, integrating contributions from Western psychological research and Buddhist contemplative theory. From these perspectives, mindfulness can be viewed as a regulatory strategy that operates primarily at the level of attention deployment, subsequently promoting a unique blend of regulatory techniques. Next, we discuss how mindfulness is developed through attention training, reviewing emergent research on the cognitive and neural mechanisms associated with MT interventions. Psychologically, the primary mechanism in MT interventions is the promotion of present-moment awareness, which is realized through the cultivation of interoceptive attention and an attitude of acceptance toward experience. Neuroscientifically, three mechanisms of training are evident: (1) increased access to sensory representation; (2) decreased activation of a network for habitual self-elaboration; and, in longer term practitioners, (3) increased attentional stability and decreased reactivity to emotion provocation. Finally, we review some of the best-validated MT interventions, discussing their clinical benefits and limitations, and exploring future directions for research in this field.

What Is Mindful Emotion Regulation?

Mindfulness has been described as "the awareness that arises from paying attention on purpose, in the present moment and nonjudgmentally to things as they are" (Williams, 2007, p. 47). This definition distinguishes mindfulness from regulatory efforts to improve the hedonic tone of experience, such as using cognitive reappraisal to make an image of suffering appear less aversive (Gross, 2002). Instead, mindfulness promotes exploratory attention to momentary experience regardless of its unpleasantness. Such exploration creates opportunities for insight into one's mental habits, reducing automatic efforts to control or manipulate feeling states. Indeed, the Pali term *vipassana*, which is the origin of the English term *mindfulness*, literally means "insight" (Bischoff, 1996). To describe how such insight occurs, we present a mindfulness theory that is consistent with both Buddhist and secular models of emotion regulation.

Mindfulness from a Buddhist/ Contemplative Model of Mind

Much of Buddhist theory on mindfulness is contained in a discourse known as the *Maha Sattpatthana Sutta*, which translates to "the Great Discourse on the Foundations on Mindfulness" (Silananda, 2002). Within this history of the Buddha's teachings, mindfulness rests upon four contemplative foundations that represent necessary targets for attentional focus. These foundations are (1) the body, (2) feelings, (3) the mind or consciousness, and (4) the *Dhammas*, or mental qualities. It is telling that these first three foci explicitly exclude conceptual elaboration or judgment but instead entail the allocation of attention to concretely perceived experiences, such as physical sensations, emotional responses, and thoughts. The fourth category, the *Dhammas*, describes the optimal attitudes and common pitfalls surrounding mindful attention to objects in these first three categories. In effect, this fourth category serves as a set of top-down regulatory goals for effective mindfulness practice. It is asserted that it is only through the correct practice of attention deployment that the pinnacle of emotion regulation may be achieved: the complete extinction of suffering.

Mechanistically, the primary tenet of the Buddhist psychological framework is that the mind may only hold one object at a time. Therefore, attention deployment to the firt three foundations limits conceptual elaboration that triggers or extends negative emotion (Silananda, 1998). Thus, in the face of an emotional challenge, mindfulness includes the somewhat paradoxical regulatory instruction to focus directly upon one's negative feelings; by focusing on a feeling rather than its inciting attentional object, a person diminishes the elaborative appraisal that iteratively incites negative feeling. Over time, it is argued, the habitual tendency to engage in negative appraisals will itself diminish, as one internalizes the practice of nonjudgmental attention. Of course, letting go of past events, particularly aversive ones, is often easier said than done. Thus, Buddhist psychology has a rationale for using meditation formally to train attention. Mindfulness from a Buddhist perspective is therefore a practical rather than mystical regulatory strategy, complete with its own psychological account of emotion regulation. The phenomenological description of how attention deployment leads to the attenuation of negative emotions provides a rich set of hypotheses compatible with Western psychological models of emotion regulation.

Mindfulness from a Psychological Science Perspective

The idea of attention deployment as emotion regulation is not foreign to scientific accounts of emotion regulation. For example, the process model distinguishes between multiple strategies for regulation that can occur before and after emotional awareness, distinguishing broadly between antecedent-focused and response-focused regulation (Gross, this volume). Generally speaking, the earlier one regulates emotion following stress perception the better: For example, the antecedent-focused strategy of cognitive reappraisal appears to be a healthier regulatory strategy than the response-focused strategy of expressive suppression, leading to lower stress-related symptoms (Gross & John, 2003; Moore, Zoellner, & Mollenholt, 2008). However, most research

on antecedent-focused emotion regulation focuses on appraisal processes, a relatively late regulation strategy for cognitive change. Few studies have examined earlier forms of regulation such as attention deployment, and those that do tend to focus on distraction (Gross, this volume), a withdrawal-oriented strategy often employed in the face of high-intensity emotional challenge (Sheppes, this volume).

Mindfulness substantively contributes to psychological models of emotion regulation by advancing a regulatory model of approach-oriented attention deployment (Figure 32.1A). Unlike distraction, mindfulness is characterized by acceptance rather than withdrawal from aversive emotional experience (Kabat-Zinn, 1982). Through MT, participants learn nonjudgmental attention to present-moment sensation, returning attention their situation rather than progressing to appraisal processes and responses (Figure 32.1B). By engaging cognitive resources in effortful attention deployment, mindfulness limits the recruitment of habitual secondary appraisals and reactions. The disruption of automatic reactions and return to sensory attention allows new aspects of a situation to be perceived, effectively altering situation construal. This altered situation perception then permits the generation of novel responses rather than mapping experiences into a preexisting conceptual field. The iteration between "top-down" attentional control and "bottom-up" sensation forms the basis for how mindfulness promotes insight and limits appraisals of suffering (Teasdale & Chaskalson, 2011). Since responses following such iteration are more finely tuned to the idiosyncratic demands of the situation, they can be more adaptive and appropriate than habitual reactions (Figure 32.1C). Thus, despite its primary focus on the deployment of attention, mindfulness appears to impact additional stages of the regulatory process: It introduces novel regulatory intentions, facilitates novel appraisals of emotional experience, and promotes expression rather than suppression of emotional responses.

What Makes Mindful Emotion Regulation Unique?

Framing mindfulness within the process model does not undermine the unique con-

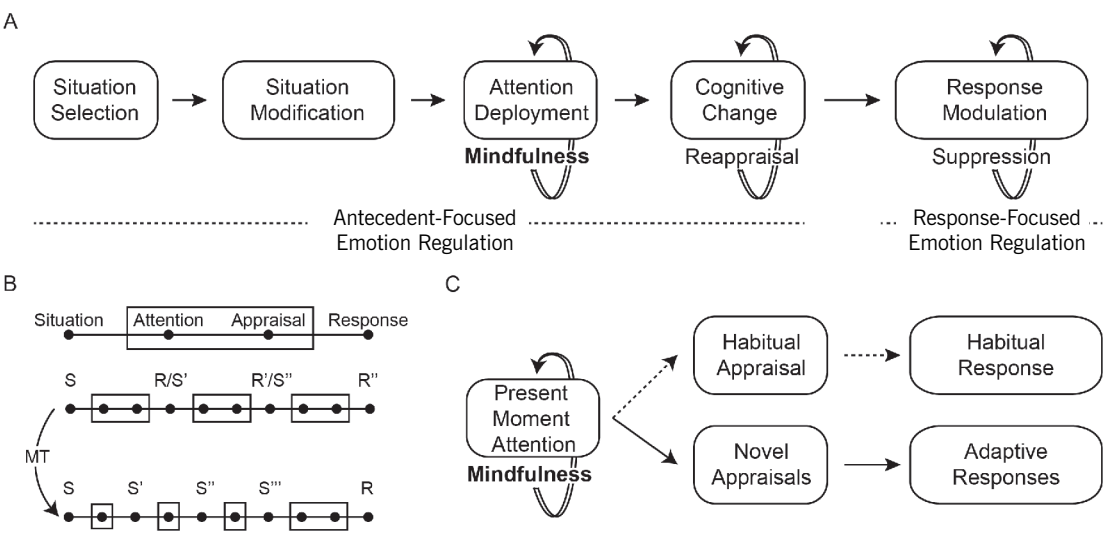

FIGURE 32.1. A process model account of mindful emotion regulation. Mindfulness can be situated in Gross's (2002) process model of emotion regulation, acting primarily on attention deployment (Panel A). Attention redeployment allows new situation perception, without requiring an immediate emotional response, thereby promoting the flexible generation of novel appraisals and responses (Panel B). The repeated redeployment of attention to sensation serves to interfere with and eventually extinguish habitual appraisals (Panel C).

tribution of mindfulness to existing models of emotion regulation. Mindfulness is unique in promoting meta-awareness of emotion regulation strategies, detecting and disrupting rumination, and in creating a trajectory for self-change. We describe each of these capacities in detail below.

First, mindfulness serves as a meta-strategy: While MT practices begin with an emphasis on attention deployment, this attentional focus is designed to cultivate awareness of regulatory habits. In acknowledging these habits, a person is then better able to choose flexibly between regulatory responses rather than automatically defaulting to particular strategies. Perhaps because of its adaptive nature, mindfulness often leads to superior regulatory effects when compared to existing forms of regulation mentioned in the process model, such as expressive suppression (Liverant, Brown, Barlow, & Roemer, 2008), rumination (Arch & Craske, 2006), or distraction (Goldin & Gross, 2010). This is not to disparage the value of any of the aforementioned techniques, but rather to suggest that mindfulness may enhance regulation by increasing flexibility in regulatory strategy selection. By cultivating an attitude of curiosity in response to an emotional challenge, mindfulness provides an opportunity to tailor regulatory responses to address both personal coping capacity and fluctuating situational demands.

A second unique contribution of mindfulness to emotion regulation is its ability to disrupt rumination or rehearsal of negative emotional triggers. By widening attention to focus on the evolving nature of emotional experience, mindful attention disrupts cognitive elaboration on negative events. Many of us have had the experience of ruminating at length over a perceived slight. By mindfully attending to momentary experience rather than rehearsing an upsetting memory, negative emotions are permitted to subside. Consistent with this theory, research suggests that mindfulness is particularly effective for reducing dysphoric rumination, outperforming relaxation training control groups in several cases (Feldman, Greeson, & Senville, 2010; Jain et al., 2007). By attending to negative emotions rather than treating them as a problem to be solved, mindfulness reduces suffering by limiting cognitive elaboration on aversive events.

A third unique contribution of mindful emotion regulation is its ability to promote a trajectory of self-change. Mindfulness requires an intensive investment: As discussed below, participants in MT interventions are asked to engage in meditative practices for about an hour a day for 8 weeks or more, with the explicit goal of increasing mindfulness throughout daily life. Participants are asked to take on attitudinal qualities in a trait-like manner. While salutary MT effects have been observed after training periods as brief as 20 minutes a day for 3 days (Zeidan, Gordon, Merchant, & Goolkasian, 2010), longitudinal benefits of mindfulness are more frequently reported following training periods of weeks or more. It is unknown whether formal training regimens involving reappraisal or suppression generate similar levels of self-reported change, although there is reason to believe that that MT employs unique mechanisms for effecting change, as discussed more fully below.

How Does MT Work?

Attending nonjudgmentally to negative emotion is often easier said than done. For this reason, mindfulness is viewed as a skill that requires training and practice. Like other traditions in the growing "third wave" of cognitive psychotherapy techniques (Hayes, 2004), MT is designed to promote self-awareness of the interpretive context that drives a person's maladaptive reactions. Yet, unlike talk-therapy interventions, MT employs formal meditation practice to achieve these aims. In mainstream interventions such as cognitive-behavioral therapy (CBT), insight is a stepping-stone to the main therapeutic focus: The recognition of maladaptive patterns of appraisal allows their replacement with adaptive alternatives. In MT, however, increased awareness into automatic reaction patterns is itself the goal of the intervention. It is fair to wonder then: How does awareness alone promote adaptive behavior? In this section, we outline how MT impacts attention, and through such effects improves the well-being of its practitioners.

To strengthen attention, MT begins with concentrative attention practices that often focus on physical sensations, such as fine-

grained "body scans," in which individuals attend to physical sensation from specific body parts, or feelings of respiration (Kabat-Zinn, 1990). Such concentrative practices may already hold some regulatory benefits, because intense focal attention on a particular object may help to dissolve self–object boundaries (Lutz, Slagter, Dunne, & Davidson, 2008), and thereby help to suspend patterns of self-absorbed rumination. However, mindfulness practices then transition from focal attention to *open monitoring*, widespread attention to all sensations, thoughts, and emotions. Open-monitoring practices aim to increase awareness of the transitory nature of experience, yielding insight into one's own habitual patterns of distraction and reaction. Encouragingly, short-term mindfulness interventions appear to increase the ability to notice changes in the sensory field and refocus attention appropriately (Jha, Krompinger, & Baime, 2007; Schmertz, Anderson, & Robins, 2009), and may preserve working memory capacity that would ordinarily degrade in times of stress (Jha, Stanley, Kiyonaga, Wong, & Gelfand, 2010).

Of course, the ability to engage in open, mindful attention is not purely an acquired skill; we are all mindful of different things during the day. Nonjudgmental, open awareness has been shown to arise naturalistically, particularly in conjunction with positive moods (Fredrickson & Branigan, 2005). The tendency to be mindful may vary widely from person to person, as attested to by recently developed mindfulness scales, which also demonstrate that these trait indices of mindfulness appear to increase with meditative practice (Baer et al., 2008; Lau et al., 2006; Walach, Buchheld, Buttenmuller, Kleinknecht, & Schmidt, 2006). Conversely, trait mindfulness may also predict responsiveness to MT and the specific attention tasks therein (Shapiro, Brown, Thoresen, & Plante, 2011), suggesting a reinforcing cycle among central components of mindful intentions, attention practice, and attitudes.

Central Components of MT

Mindfulness-based interventions appear to operate at multiple stages of emotion regulation. At a higher-order, conceptual level, MT represents an interplay between participants' intentions for self-improvement, the attentional practices themselves, and the use of these practices to foster an attitude of acceptance and nonreactivity to negative emotion (Shapiro, Carlson, Astin, & Freedman, 2006). At a mechanistic level, mindfulness can be explained by the effects of the attentional practices themselves, detailing how mindful attention alters the preexisting relationship between events and subsequent self-attribution, limiting automatic self-evaluative processing (Frewen, Evans, Maraj, Dozois, & Partridge, 2008). To provide a complete account of how mindfulness impacts each aspect of emotion regulation, we present intention, attention, perception, implication, and attitude in a single conceptual model (Figure 32.2).

While empirical research has focused on how mindfulness alters cognition through attention training, it is important to recognize that the success of such practices depends on a participant's motivation and expectations for such training. The generation of a proper *intention* is critical to the success of contemporary MT practices (Kabat-Zinn, 1990; Shapiro & Schwartz, 2000); participants must expect to have some benefit from the practice to engage consistently in a novel and intensive practice of restructuring cognitive habits. Within the meditative practices themselves, intentions serve as a foundation for the direction of attention; by formally setting an intention to attend to one's body or breath, a wandering mind becomes indicative of a correctable loss of attention. Central to holding "right intention" in mindfulness is the promotion of *participant autonomy*, that participants are ultimately accountable to themselves for their self-improvement efforts. For example, in therapeutic contexts, MT emphasizes daily, self-directed practice rather than relying on the clinician for directing inquiry and generating new appraisals. Mindfulness teachers provide the occasional reminder to check for mind wandering during these practices, but with decreasing frequency over the weeks of a mindfulness course. Instead, participants are entrusted with an increasing responsibility for monitoring and reallocating attention during meditation. Indeed, it has been argued that one yardstick of mindful intentions may be participants' willingness to ascribe personal responsibility for their reactions to stressful events (Lakey, Kernis, Heppner, & Lance, 2008).

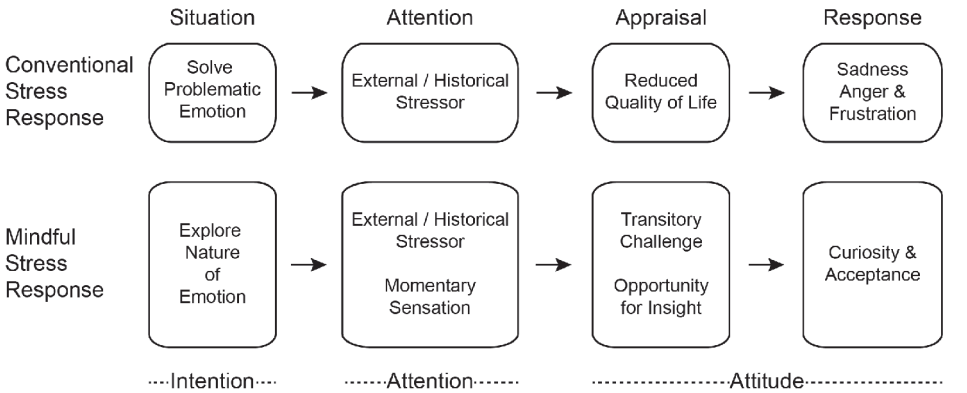

FIGURE 32.2. Broader regulatory consequences of mindfulness. Mindfulness effects extend beyond attention deployment in modulating stress responses. Different stages of the process model for emotion regulation are displayed for conventional (top panel) and mindful (bottom panel) stress responses. Conventionally, stressful situations are construed as demanding a regulatory response, prompting attention to the stressor, dysphoric appraisals, and generation of negative emotional responses. Mindfulness acts on all four of these phases: Mindful intentions cast stressful situations as opportunities to practice; mindful attention broadens the field of awareness to include changes in sensation; and mindful attitudes promote appraisals of engagement and responses of acceptance rather than an obligatory dysphoric stance.

The second major factor in MT interventions is *attention*. The distinguishing characteristic of MT is a set of meditative practices focusing on sensory experiences of the body. These meditative practices serve at least three major functions in promoting adaptive emotion regulation. First, attention to momentary changes in body sensation creates a broad awareness of experience than extends beyond a vigilant or ruminative focus on stressors. By attending to a broader set of experiences from moment to moment, participants learn to *reperceive* the world (Shapiro et al., 2006). Reperceiving refers to the developed capacity to disengage from "the drama of our personal experience" and attend to novel sensations and interpretations (p. 377). Second, this broader attentional focus creates a psychological distance between dysphoric feelings and broader self-attributions about efficacy or value. This psychological distancing is known as *decentering*, in which self- and situational appraisals are experienced as momentary events rather than immutable facts (Fresco, Segal, Buis, & Kennedy, 2007). Finally, the novel perspective afforded by decentering may serve to disrupt habitual interpretations, creating a *broadened context for appraisal* (Garland, Gaylord, & Fredrickson, 2011). An increased ability to disengage from ruminative self-elaboration in favor of sensory exploration constitutes a broader context from which to view the world. From this broadened context, participants are able to explore novel interpretations for events and more skillful means for responding to emotional challenges.

The final major factor in MT is *attitude*. Negative emotions naturally arise during both meditative practice and in everyday life. However, it is one's attitude toward these emotions that determines whether they are appraised as self-diagnostic. For example, attention and reflection on negative emotions can be beneficial when approached with an attitude of curiosity and openness, leading to constructive cognitive reappraisal; however, this same reflective process can be maladaptive when accompanied by an attitude that negative emotions are unacceptable and problematic, breeding rumination and dysphoric self-evaluation (Gotlib & Joormann, 2010). To counter this maladaptive tendency, *nonjudgment* is espoused as a core attitudinal goal in MT, a goal supported by a constitutive attentional practice. To cultivate nonjudgment, participants strive to direct attention away from judging the validity of experience and toward attending to the sense of the experience itself. Over time, the habitual reorienting of attention to sensation

disengages automatic evaluative responses to sensations, feelings, and thoughts. In this fashion, a negative emotion or disparaging thought about oneself is treated as a momentary experience with no more self-diagnostic value than an itch upon the skin.

While the relationship between intention, attentional practices, and attitude can be seen as a linear progression in the cultivation of mindful emotion regulation skills, realistically these components are mutually reinforcing (Shapiro et al., 2006). Below we discuss how the interplay among these components creates a mechanism for promoting stress tolerance and well-being.

Psychological Mechanisms of Mindfulness Training

A recent review of the research literature links MT to improved attention, body awareness, emotion regulation, and altered self-perception (Holzel et al., 2011). How MT practices generate these positive changes may, however, benefit from further clarification. It may seem strange, for instance, that repeated attention to body sensations should provide widespread therapeutic benefits. How can attention to the breath and other physical sensations promote a broadening of attention and reduction of automatic patterns of reactivity? Furthermore, one may question the importance of attending to emotional appraisals in the first place. Emotions hold a natural capacity to capture attention (Ohman, Flykt, & Esteves, 2001); why then would practice in attending to emotions help in regulating them? To address these questions, we expand upon a theoretical model (Bishop et al., 2004) of how mindful attention directly supports adaptive emotion regulation based on two major principles: (1) *attention to the present*: broad attention to present-moment sensation, with an emphasis on body sensation rather than cognitive deliberation; and (2) *nonjudgment*: the suspension of judging experience to be intrinsically good or bad in favor of a more general attitude of acceptance. Together, these principles mutually support the gradual process of reconfiguring attention and cognition, extinguishing maladaptive patterns of reactivity, and introducing cognitive flexibility in the response to stress (Figure 32.3).

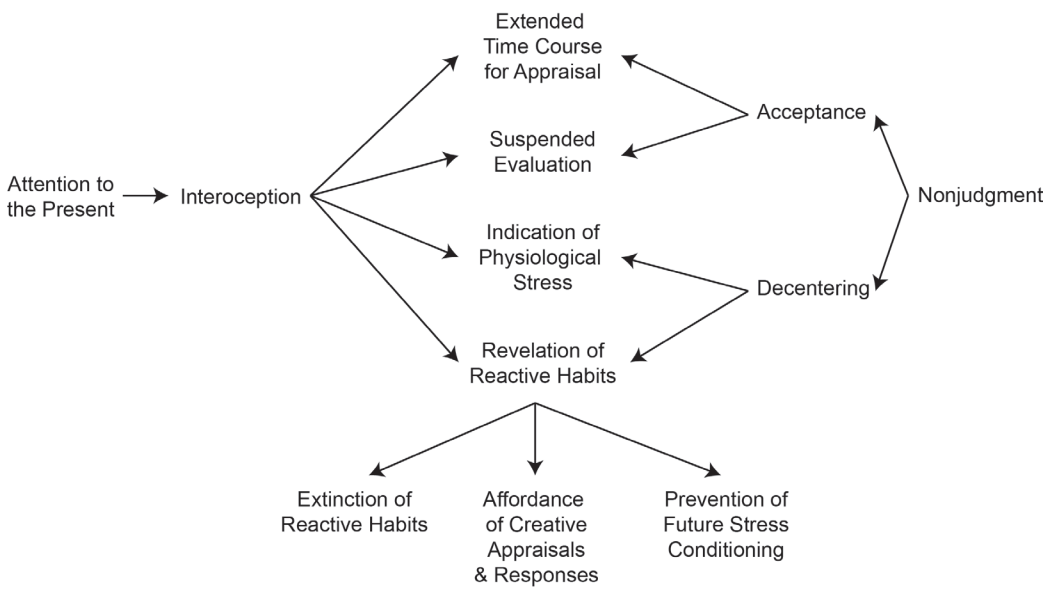

FIGURE 32.3. The two foundations of mindful attention: Attention to the present and nonjudgment. Together they support a suspension of self-critical evaluation and are revelatory of reactivity habits. Over time, such information can yield insights that afford the creation of novel and more adaptive stress responses, help to dismantle existing patterns of reactivity, and can prevent the formation of novel conditioned or "knee-jerk" reactions to stress.

Attention to the Present

The cultivation of momentary awareness is central to mindfulness practices. Through repeated exercises in attending to the sensations of the breath or different regions of the body, participants are encouraged to develop *interoception*, an awareness of momentary sensations from inside the body. These sensations then act as an access point for understanding one's reactions to the world as they unfold; rather than conceptually interpreting or predicting responses to events, participants are encouraged to explore their actual reactions "in real time," using interoceptive attention to monitor changes in body sensation from moment to moment. While myriad factors determine the efficacy of a mindfulness intervention, attention to interoceptive sensation is important for several reasons. First, focusing on body sensation helps to *suspend evaluative processes*, disrupting the habitual progression from sensation to appraisal by redirecting attentional resources from such processes. Second, in the absence of habitual evaluation, a person extends the *time course of appraisal*, reducing the need to quickly map events on a preexisting conceptual field, thereby disempowering deeply entrenched dysphoric interpretations. Third, interoception serves as an *indication of physiological stress*: By noticing one's breathing, heartbeat, muscle tone, or digestion change, one can gain insight into one's own emotional appraisals and notice the kinds of situations that trigger automatic reactions. Finally, this sensitivity to environmental triggers allows for the *revelation of reactive habits*, because intrusive cognitive failures of interoceptive attention provide an opportunity to notice where the mind goes when it wanders. Perhaps because interoceptive perceptual details are so distinct from thoughts and evaluations, these details act as attentional "anchors" for perception that make mind wandering more apparent.

Nonjudgment

The cultivation of present-moment attention is complemented by an intention to refrain from judgment and cognitive reactivity. Nonjudgment supports formal meditation practice, and also works with attention to the present moment to regulate emotional challenge adaptively. The formal practice of mindfulness meditation offers ample opportunity to cultivate nonjudgment. Mind wandering is inevitable during the attempted maintenance of interoceptive attention, prompting from participants a range of negative reactions, such as self-criticism, anxiety, frustration, or dejection. Rather than deny, suppress, or distract attention from these lapses and subsequent feelings, participants are encouraged to notice and accept all experience, including attentional lapses and feelings of frustration. This attitude of acceptance then allows a more rapid return to present-moment sensation than would be expected from digression into self-recrimination.

The practice of nonjudgment also has important implications for emotion regulation beyond formal meditation practices. By focusing on noticing changes in experience rather than formulating reactions, an attitude of nonjudgment limits the association between initial appraisals of failure or challenge and subsequent dysphoric or avoidant secondary appraisals. This curtailing feature of nonjudgment is critical to recovery from affective and substance use disorders, in which inciting mental appraisals habitually lead to self-destructive mental and behavioral habits (Scher, Ingram, & Segal, 2005). Indeed, both dispositional and treatment-related increases in mindfulness predict greater facility in letting go of negative thoughts (Frewen et al., 2008), and lower levels of avoidance and rumination (Kumar, Feldman, & Hayes, 2008). This development of nonjudgment can be thought of as two complementary and related capacities: *acceptance* of experience and *decentering* from attributing self-relevance to experience, which together support the outcomes of interoceptive attention described earlier. First, *acceptance* indicates a willingness to tolerate outcomes even when they diverge from intentions, limiting the automatic expansion of evaluative reactions. For example, one study of a mindfulness intervention reported improved quality-of-life, depression, and fatigue scores in a multiple sclerosis population, but unaltered ratings of physical symptoms, such as lower and upper limb mobility (Grossman et al., 2010), suggesting that the symptom acceptance training reduced broader secondary appraisals to

produce a training benefit. Second, *decentering* involves reducing reliance on conceptual self-appraisal as the primary determinant of well-being. Instead, such self-appraisal is viewed on par with physical sensation, a momentary experience that does not imply the existence of a temporally extended, enduring self. Recent research suggests that the ability to view one's emotional reactions from a more objective viewpoint is a critical determinant of whether reflection on emotion can be constructive or degenerate into maladaptive rumination (Kross, Gard, Deldin, Clifton, & Ayduk, 2012).

Together, attending to the present and cultivating a nonjudgmental attitude mutually reinforce one another: Present-moment attention provides a context in which to practice nonjudgment, and nonjudgment enhances the stability of attention. Beyond supporting each other, these two principles can account for many of the benefits attributed to MT. First, mindfulness enables the *extinction of reactive habits* by removing conditioned appraisal responses from their triggering stimuli and replacing them with interoceptive attention. For example, MT has helped chronic pain sufferers to reduce subjective reports of distress, although the intensity of pain sensation ratings themselves did not change (Morone, Greco, & Weiner, 2008; Rosenzweig et al., 2010). Second, by reducing reactive habits, mindfulness *affords new, creative appraisals*, allowing a person to approach experiences with a sense of curiosity and exploration. Novel appraisals then may extend to include previously unattended positive elements, including interpersonal relationships, enjoyment of daily activities, or compassion toward oneself and others (Allen, Bromley, Kuyken, & Sonnenberg, 2009; Kuyken et al., 2010). Third, continued mindfulness practice *prevents future stress conditioning* and provides competing knowledge of constantly changing interoceptive sensations that weakens the need to turn to conceptual evaluations (Kabat-Zinn, 1990). The suspension of engagement in automatic conceptual evaluation during stress can thereby prevent further conditioning of dysphoric cognition. For example, participants completing MT reported significant reduction in their experience of daily hassles and distress compared to a control group (Williams, Kolar, Reger, & Pearson, 2001). Together, these psychological mechanisms promote and sustain flexibility in the regulatory response to emotional challenge.

Neural Mechanisms of Mindfulness Training

The notion that MT cultivates a qualitatively distinct approach to emotion regulation is supported by emerging neuroscience research. Traditionally, emotion regulation such as reappraisal has been associated with the prefrontal cortex (PFC), which tends to be active during the manipulation and evaluation of information (Ochsner, Bunge, Gross, & Gabrieli, 2002). The PFC in turn appears to be component of a broader frontoparietal attention network associated with directing attention to external objects, filtering the endless stream of sensory information based on its motivational relevance (Corbetta & Shulman, 2002). In a sense, this well-defined attention system seeks to fix sensory information as a set of objective facts that can be judged as being good or bad depending upon their relationship to a person's goals. In the case of an undesired outcome, cognitive reappraisal directs attention toward changing one's interpretive context to mitigate the obligatory emotional impact of contextually derived failure appraisal on the self.

Mindfulness, however, argues for a qualitative shift away from attachment to external outcomes. To do so, there is an attempt to shift attention away from attentional habits that link external sensations to conceptual manipulation and elaboration. Instead, MT teaches individuals to direct attention internally, to representation of visceral components of emotion, as supported by a distinct neural pathway for interoception, the sensation of the body's internal state, including signals from the breath (Farb, Segal, & Anderson, 2013; Farb et al., 2007), and heartbeat (Lutz, Greischar, Perlman, & Davidson, 2009). Through reliance on the transitory and unelaborated representations of internal body states, MT may appeal to a relatively unconditioned system in the sense that such physical sensations do not have the same level of association with conceptual elaboration or regulatory reactivity. Indeed, one reason that many mindfulness practices involve interoceptive attention is that such

practices improve the stability and frequency with which one perceives the transitory and reinterpretable nature of human experience (Baer, Smith, Hopkins, Krietemeyer, & Toney, 2006; Brown et al., 2007; Ivanovski & Malhi, 2007; Kabat-Zinn, 1982).

Recent research evidence supports the idea that MT improves interoceptive access by decoupling sensory cortices from brain networks involved in the habitual evaluation and elaboration of experience. Activation of cortical midline regions such as the medial prefrontal cortex (mPFC) appear are associated with self-referential evaluation (Farb et al., 2007; Kelley et al., 2002), depressive rumination (Farb, Anderson, Bloch, & Segal, 2011), and negative mood (Farb et al., 2010; Goldin & Gross, 2010). MT appears to reduce both rumination (Ramel, Goldin, & Carmona, 2004) and cortical midline activation (Farb et al., 2010; Goldin, Ramel, & Gross, 2009); furthermore, mindful attention can begin to reduce mPFC activation even in untrained individuals (Farb et al., 2007). Meditation has been linked to extensive cortical deactivations (Baerensten et al., 2010; Ives-Deliperi, Solms, & Meintjes, 2011; Lazar et al., 2000), suggesting its importance for disengaging conceptual processing. Following training, participants demonstrate a reduced connection between sensory and evaluative cortices, but greater connectivity within sensory cortices and between the brain's executive control regions and sensory cortices (Farb et al., 2007, 2013; Kilpatrick et al., 2011).

A third important neural mechanism by which MT promotes effective emotion regulation is by enhancing attention control in more experienced meditators. In one study, 3 months of intensive meditation training was associated with reduced variability in low frequency brain signal electroencephalographic (EEG) activity (Lutz, Slagter, et al., 2009). These brain changes were correlated with reduced variability in reaction time in a focused attention task, suggesting that this stabilization of neural activity represents a more stable cognitive workspace in which to perceive and manipulate sensory objects. Furthermore, long-term meditators demonstrated reduced network strength in the cortical midline default network associated with conceptual elaboration and mind wandering but enhanced connectivity within attention-controlling brain regions (Brewer et al., 2011), again denoting an improved ability to resist distraction and to effectively deploy attention.

In advanced stages, there is evidence to suggest that mindfulness training may itself become automatic and promote trait-like effects. An intriguing neuroimaging study examined the meditative state across a range of practice experience (Brefczynski-Lewis, Lutz, Schaefer, Levinson, & Davidson, 2007). Relatively novice meditators demonstrated pervasive cortical and limbic activation relative to controls when entering a meditative state, typical of engagement in an effortful and resource intensive cognitive process. However, advanced meditators (with more than 10,000 hours of practice) showed little neural change during meditation, suggesting that their default state may have become something akin to mindful attention. Thus, the long-term effects of MT may involve a reconfiguration of the brain's information processes to dwell primarily in unelaborated sensation, in contrast to the highly automated pattern of cognitive elaboration found in most studies of Westerners to date.

Therapeutic Applications of Mindfulness

The first documented clinical effect of MT was reducing the suffering of patients with chronic pain (Kabat-Zinn, 1982). Subsequent research has confirmed that the affective appraisal of pain is altered when experienced through an open, mindful state (Perlman, Salomons, Davidson, & Lutz, 2010). Freed from pain-reactive, negative appraisals about one's self-worth and quality of life, cognition can be engaged constructively toward the cultivation of empathy and compassion (Leary, Tate, Adams, Allen, & Hancock, 2007).

The reframing of painful or otherwise upsetting experiences is especially important in mood disorders. The effortful down-regulation of negative emotion is perceived as more difficult by patients with a mood disorder (Keightley et al., 2003), for whom self-compassion seems like a worthwhile but unachievable strategy (Pauley & McPherson, 2010). An important clinical implication of

these findings is that for patients with mood disorders, instructions to reappraise cognitively or otherwise regulate negative emotion may be ineffective. In depression, powerful and habitual dysphoric interpretations may be activated during such regulatory efforts, leading to rumination, in which elaborating on negative affect ironically perpetuates dysphoric mood (Nolen-Hoeksema, 2000; Watkins, Moberly, & Moulds, 2008).

If the engagement of a patient's cognitive faculties only serves to exacerbate negative mood states, one might question how any behavioral intervention can effect positive change. Since cognitive elaboration efforts may automatically trigger negative self-judgments that cannot be voluntarily overridden, MT provides an alternative to such elaborative habits. Through the cultivation of attention to unelaborated, present-moment sensation, mindfulness presents patients with a task that does not require the deployment of cognitive evaluation and elaboration. Furthermore, by explicitly practicing nonjudgment, patients can begin to recognize and disengage from conceptual judgment in response to their sensory experiences.

Critically, mindful emotion regulation does not aim to inhibit elaborative processing through thought avoidance, a regulatory strategy that ironically predicts dysphoric affect (Ottenbreit & Dobson, 2004). In MT, attention is instead positively directed toward present-moment sensation, providing a nonconceptual and nonthreatening attentional focus that does not rely on cortical midline activity. Depression can be thought of as a combination of both approach deficits and heightened avoidance motivation (Trew, 2011); mindfulness targets both types of motivation, replacing the tendency to avoid negative experiences with an exploration of these experiences' constituent sensations.

As one might expect from such a fundamental mechanism of action, mindful emotion regulation strategies can be applied to a variety of contexts extending beyond pain and depression. One recent review catalogued beneficial psychological effects of mindfulness-based interventions in the treatment of anxiety disorders, substance abuse, cancer, heart disease, arthritis, fibromyalgia, multiple sclerosis, psoriasis, and HIV (Chiesa & Serretti, 2010). MT has also been of benefit in older populations, particularly in improving the coping skills of familial caregivers (Oken et al., 2010).

The recent proliferation of MT in clinical contexts has been supported by the creation of manualized therapeutic interventions. Customized interventions now exist that employ MT techniques to address the needs of specific clinical populations, such as those suffering from issues of pain, depression, and addiction. The standardization of these interventions has been critical for clinical research, allowing a consistent application MT to be evaluated in specific clinical contexts. Below, we discuss some of the most widely researched therapeutic interventions of mindfulness, detailing their background, format, and target populations.

Mindfulness-Based Stress Reduction

Background

Mindfulness-based stress reduction (MBSR) was developed as an outpatient program at the University of Massachusetts Medical School in 1979, based on creator Jon Kabat-Zinn's personal experience with *vipassana* (insight) meditation, a practice that originated in the millennia-old Theravada Buddhist tradition. The first published report on MBSR described reduced pain and symptoms of negative mood in a group of 51 patients with chronic pain (Kabat-Zinn, 1982). Since that time, MBSR has been standardized with broad efficacy in reducing chronic pain and stress.

Format

MBSR is delivered as an 8-week program. Participants attend weekly group sessions with between 10 and 30 participants, where they are introduced to formal mediation practices, gentle yoga, and psychosocial education. The program also includes a day of silent meditation retreat that falls in the latter half of the course. Group members are asked to practice yoga and/or meditation for approximately 40 minutes a day for homework, along with reflection and journaling exercises. Formal meditation practices

include breath monitoring; body scans (the progressive direction of attention to different parts of the body); and diffuse direction of attention to sounds, thoughts, feelings, and bodily sensations. Group sessions generally last for 2.5 hours and focus on group meditation practice and discussion of these practices. However, other commonly discussed themes include stress and the body, habitual patterns of reactivity, and creative ways to respond to stress. Group dialogue on difficulties in performing practices and insights gained are also valuable components of an MBSR course. During these dialogues, instructors model key components of mindful awareness, such as noticing the body sensations that accompany different thoughts and impulses.

As part of the weekly homework, courses often include diary exercises, which are later discussed in the group setting. Commonly, participants spend a week taking notice of pleasant events, then a week on unpleasant events, and another week on monitoring stressful communications. However, common even to these externalized practices, there is an emphasis on how these events occurred from an interoceptive perspective (i.e., how one felt in one's body during these events). There are no hard-and-fast rules for course compliance, although participants who are absent are typically contacted by course instructors to probe commitment to course participation. Participant retention in MBSR is generally high, with 80% of enrolled participants generally completing the course (Farb et al., 2007; Kabat-Zinn, Lipworth, & Burney, 1985).

Target Population

In healthy individuals (i.e., those without a specific medical or psychiatric diagnosis), MBSR appears to yield substantial but nonspecific reductions in reported levels of stress, in addition to specific reductions in levels of rumination and anxiety (Chiesa & Serretti, 2009). Research suggests that MBSR promotes well-being across a variety of clinical conditions, including chronic pain, anxiety, fibromyalgia, cancer, psoriasis, and coronary artery disease (Baer, 2003; Grossman et al., 2004). A third stream of research suggests that MBSR benefits extend to health care professionals, improving their ability to cope with stress (Irving, Dobkin, & Park, 2009). Finally, adapted forms of the MBSR program have improved mental function and reduced stress in primary caregivers of people with terminal diseases such as dementia (Oken et al., 2010) and advanced-stage cancer (Lengacher et al., 2012).

Mindfulness-Based Cognitive Therapy

Background

Mindfulness-based cognitive therapy (MBCT) was developed to target the specific challenges inherent in chronic mood disorders. Major depressive disorder, a leading cause of disability worldwide (Gelenberg, 2010), has a chronic, cyclical nature due to high risk of relapse. Presently, maintenance pharmacotherapy is the most widely implemented approach to reducing relapse risk in depression (Kupfer et al., 1992). However, long-term antidepressant use involves problems with drug tolerance and side effects, and does not target the dysphoric cognition that contributes to relapse risk in response to future stressors. Individuals with a history of depressive illness, including those in remission, are prone to increased cognitive reactivity, a pattern of increased negative self-judgment in response to emotion challenge. Dysphoric cognitive reactivity appears to be an important determinant of depressive relapse, along with genetics, environment, and social support (Segal et al., 2006). However, such reactivity may be modifiable; addressing these patterns of dysphoric thinking may provide posttreatment protection against relapse (Paykel et al., 1999).

While Beck's (1979) formulation of cognitive therapy focused on modifying underlying dysfunctional attitudes and core beliefs, Ingram and Hollon (1986) argued that prophylactic effects following remission are dependent on enhancing an individual's ability to take a distanced, disidentified perspective from depressive thoughts (i.e., decentering). To create a therapy that maximized long-term prophylactic effects, Segal, Williams, and Teasdale (2002) expanded on the existing CBT framework to create MBCT, which uses mindfulness practices to develop

participant ability to engage in decentered attention.

Format

The MBCT program involves medium-size groups (between eight and 13 participants). The length of sessions ranges between 2 and 2.5 hours in order to accommodate session content flexibly and any clinical issues that may arise. Participants are also offered a full-day "silent retreat" between the sixth and seventh weeks of the program. Every session is initiated with a meditation to ground participants in "being" mode and help them to transition out of the business and "doing" mode of their everyday lives. Meditation practices include the body scan, gentle yoga, and mindful walking, as well as sitting meditation practices with foci such as the breath, sounds, body sensations, and thoughts. Each session includes a review of the home practice, in addition to group discussion of insession mindfulness practices.

MCBT also features the 3-minute breathing space, a condensed version of formal mindfulness meditation in which participants broadly inventory mood and quality of thought, shift attention to the breath, then return to broad attention on sensation and cognition. The inclusion of the 3-minute breathing space offers an opportunity to integrate practice mindfulness in situations where entrenched patterns of dysphoric cognition make extended meditation difficult or impossible. It also affords participants a chance to "check in" with themselves during the day, limiting patterns of rumination. While initially incorporated into MBCT, the 3-minute breathing space is increasingly making its way into MBSR and related MT interventions.

Target Population

MBCT was designed as prophylactic support in prevention of depressive relapse. In two large, randomized control trials, MBCT has been shown to be efficacious for patients with three or more past episodes of depression by halving relapse rates better than did a treatment-as-usual control condition (Ma & Teasdale, 2004; Teasdale et al., 2000). In a more recent, large-scale trial, MBCT was been shown to be as effective as maintenance antidepressant medication in preventing depressive relapse, with both surpassing the performance of a placebo control condition (Segal et al., 2010).

While originally designed to prevent relapse during remission, MBCT has also shown efficacy in the treatment of acutely depressed patients, including those with treatment-resistant depression (Barnhofer et al., 2009; Eisendrath, Chartier, & McLane, 2011). MBCT has also been adapted for children ages 9–13 (Semple, Lee, Rosa, & Miller, 2010), and for adults over age 65 (Smith, Graham, & Senthinathan, 2007). MBCT may be appropriate for patients with other mood disorders, but special care must be taken to ensure that patients have adequate capacity to cope with negative emotions during the meditation process (Germer, Siegel, & Fulton, 2005).

Mindfulness-Based Relapse Prevention

Background

Mindfulness-based relapse prevention (MBRP), an intervention developed to target relapse vulnerability in substance use disorders (Marlatt & Gordon, 1985), is modeled after MBCT to help people recognize internal triggers to addictive behavior without responding to them. The course features a central theme of distinguishing between primary emotional experience and secondary appraisals, with an emphasis on the idea that if the primary experiences can be accepted, secondary appraisals of coping inadequacy will lose power to trigger relapse behaviors.

Format

The intervention takes place over eight weekly sessions and introduces many of the same formal meditation techniques described in MBSR and MBCT. The focus of the group sessions centers on three major themes: Sessions 1–3 employ MT to provide awareness of how automatic patterns of reactivity lead to relapse behavior; Sessions 4–6 focus on implementing mindfulness techniques in high-risk or trigger situations to prevent thoughts from triggering relapse behaviors; Sessions 7 and 8 focus on integrating mindfulness practice into daily life

to provide continued development of self-care skills to protect against relapse in the future.

Similar to the 3-minute breathing space that is central to MBCT, MBRP introduces a "SOBER" breathing space (Stop, Observe, Breathe, Expand, Respond) designed to allow participants to observe the arousal of urges and respond skillfully rather than habitually. By maintaining awareness of the urge, the participant somewhat ironically prevents his or her response to the urge, allowing it to follow a homeostatic time course and eventually subside. Similar to the positive and negative events diary in MBSR, MBRP employs an "awareness of triggers" diary. By fostering awareness of thoughts, feelings, sensations, and behaviors during the arousal of drug cravings, the diary helps to create recognition of triggering situations.

Target Population

MBRP targets people with a history of subject use disorders, generally subsequent to completion of an intensive detoxification and rehabilitation program. In a recent study comparing the effects of MBRP to standard relapse-prevention treatment, MBRP group participants showed lower rates of relapse and greater levels of acceptance and mindful awareness than those in the standard treatment group (Bowen et al., 2009). Importantly, subsequent research suggests that reductions in craving following MBRP were mediated by increases in acceptance and mindful awareness (Witkiewitz, Bowen, Douglas, & Hsu, 2013).

Other Interventions Incorporating Mindfulness

Many other contemporary therapeutic interventions incorporate aspects of MT in a broader clinical protocol. Dialectical behavior therapy (DBT; see Neacsiu, Bohus, & Linehan, this volume) is modeled on the central struggle between acceptance of the present moment and desire for change. MT is an important facet of this therapy, helping participants develop radical acceptance of a problematic situation in order to direct efforts skillfully toward adaptive change. Unlike the interventions discussed earlier, DBT does not require extensive formal meditation practice but instead includes a variety of meditative exercises that may be customized to a client's needs and abilities. Acceptance and commitment therapy (ACT) also eschews formal meditation techniques but places a strong emphasis on viewing experiences with a sense of acceptance or nonjudgment, while focusing efforts on changing behaviors rather than thoughts, feelings, and sensations. In both DBT and ACT, there is much greater emphasis on the adjustment of behavior than in mindfulness-based interventions, providing more therapist-led structure around plans of action.

Contraindications

Despite the promise of MT interventions in a variety of clinical domains, it is important to remember that these techniques have only been validated with specific ailments. In fact, original MT studies excluded individuals with active substance abuse, psychotic disorders, eating disorders, obsessive–compulsive disorder (OCD), and borderline personality disorder (BPD). The reasons for such exclusion criteria were manifold; individuals with acute depression often experience compromised attention and concentration, which may pose significant challenges when learning an already challenging practice, while individuals with OCD, BPD, and active substance abuse might find long formal meditation practices difficult to tolerate due to intensively distressing intrusive thoughts, impulses, cravings, or a general tendency toward states of emotional dysregulation (Hayes, 2004; Linehan, 1993). Moreover, in these populations, reliance on emotion regulation strategies such as avoidance may be deeply ingrained, and the approach-oriented nature of mindfulness practices may initially be difficult to tolerate. Effective treatments such as cognitive therapy for acute depression or DBT for BPD exist and have a strong evidence base (Beck, 1979; Linehan, 1993). Moreover, they are tailored to fit the needs and tolerances of the individuals enrolled in such programs. For example, the development of mindfulness skills is a significant component of training in DBT skills. However, skills practices are shorter and more varied, building up over the course of several months rather than in 8 weeks as in the formats of MBSR and MBCT.

Clinicians and expert meditation teachers alike have suggested various precautions when treating individuals who have a significant history of trauma with MBSR and MBCT. Crane and Williams (2010) have discussed the importance of preexisting "grounding skills" for those individuals who may be at-risk of experiencing intrusive thoughts or being particularly cognitively reactive. Additionally, they suggest preparing such individuals for the fact that distress may arise, in light of evidence that a tendency toward brooding and cognitive reactivity is predictive of dropout in MBCT. Patients planning to engage in this type of intensive MT require the skills necessary to redirect their attention should it become drawn into destabilizing memories or overwhelming affective states prior to commencing the MBCT program. A recent review of the literature examining reports of adverse events and contraindications for mindfulness-based approaches found that the topic had been relatively neglected (Dobkin, Irving, & Amar, 2012) and warrants further investigation.

Concluding Remarks

Interest in mindful emotion regulation has increased rapidly in recent years, especially as contemplative models of mind have been increasingly supported by modern psychological science. This has enhanced our understanding of emotion regulation in two major ways: (1) It informs research on attention deployment as a form of emotion regulation, without resorting to distraction from emotion; and (2) it has begun substantively to connect scientific inquiry to broader issues in emotion regulation, such as how flexibility in regulation techniques serves as a "meta-regulatory" skill, or how intention and commitment to a regulatory goal increase realization of longitudinal improvements in regulatory capacity. Psychologically, the study of MT provides a window into the mechanisms of personal growth, combining elements of attention, intention, and attitude to form an upward spiral of regulatory efficacy. Neurobiologically, the study of mindfulness helps to validate the idea that sensory attention recruits a distinct interoceptive attention pathway. This pathway appears to be amenable to voluntary access, and such access can be facilitated through attention training. Critically, engagement of sensory attention seems to disengage brain networks involved in habitual elaborative processing, allowing for the extinction of automatic stress reactions while strengthening networks for the control of attention.

With elements of mindfulness now being identified in a variety of other "new wave," health-promoting practices such as Tai Chi, yoga, and, more generally, in psychotherapeutic practice, the challenge before us is to justify the spread of these techniques. As such, it is increasingly necessary to provide an empirically replicable model of the core components of mindful emotion regulation. Such a model will distinguish necessary regulatory elements from intervention elements that are simply customary, and help to assuage criticisms that mindfulness involves ushering mysticism into Western, evidence-based medicine. For example, the relative contributions of attitude, intention, and attention are unknown. It may be the case that daily exercises in setting beneficial self-intentions account for many of the salutary effects of mindfulness, allowing one to dispense with much of the meditative practice and instead engage in habits of self-affirmation. Alternatively, it may be that attentional improvements are necessary precursors to the experience of insight and improvements in well-being. Finally, it may be the commitment to regulating experience with an attitude of acceptance that really pays benefits, with meditative exercises simply helping to reinforce this commitment. While there is suggestive evidence that the three core elements of mindfulness—attitude, intention, and attention—are critical ingredients, it will be important to find better ways to measure their relative contributions to advance the science of mindfulness and determine its potential for integration into secular Western life.

References

Allen, M., Bromley, A., Kuyken, W., & Sonnenberg, S. J. (2009). Participants' experiences of mindfulness-based cognitive therapy: "It changed me in just about every way possible." *Behavioral and Cognitive Psychotherapy, 37*(4), 413–430.

Arch, J. J., & Craske, M. G. (2006). Mechanisms of mindfulness: Emotion regulation following a focused breathing induction. *Behaviour Research and Therapy, 44*(12), 1849–1858.

Baer, R. A. (2003). Mindfulness training as a clinical intervention: A conceptual and empirical review. *Clinical Psychology Science and Practice, 10*(2), 125–143.

Baer, R. A., Smith, G. T., Hopkins, J., Krietemeyer, J., & Toney, L. (2006). Using self-report assessment methods to explore facets of mindfulness. *Assessment, 13*(1), 27–45.

Baer, R. A., Smith, G. T., Lykins, E., Button, D., Krietemeyer, J., Sauer, S., et al. (2008). Construct validity of the five facet mindfulness questionnaire in meditating and nonmeditating samples. *Assessment, 15*(3), 329–342.

Baerensten, K. B., Stodkilde-Jorgensen, H., Summerlund, B., Hartmann, T., Damsgaard-Madsen, J., Fosnaes, M., et al. (2010). An investigation of brain processes supporting meditation. *Congitive Processing, 11*(1), 57–84.

Barnhofer, T., Crane, C., Hargus, E., Amarasinghe, M., Winder, R., & Williams, J. M. (2009). Mindfulness-based cognitive therapy as a treatment for chronic depression: A preliminary study. *Behavioural Research and Therapy, 47*(5), 366–373.

Beck, A. T. (1979). *Cognitive therapy of depression*. New York: Guilford Press.

Bischoff, R. (1996). Some notes concerning the entry "Vipassana" in the Oxford English Dictionary Additions Series, Vol. 2 (Clarendon Press, Oxford, UK: 1993). *International Journal of Lexicography, 9*(1), 35–37.

Bishop, S. R., Lau, M., Shapiro, S., Carlson, L., Anderson, N. D., Carmody, J., et al. (2004). Mindfulness: A proposed operational defintion. *Clinical Psychology: Science and Practice, 11*(3), 230–241.

Bohlmeijer, E., Prenger, R., Taal, E., & Cuijpers, P. (2010). The effects of mindfulness-based stress reduction therapy on mental health of adults with a chronic medical disease: A meta-analysis. *Journal of Psychosomatic Research, 68*(6), 539–544.

Bowen, S., Chawla, N., Collins, S. E., Witkiewitz, K., Hsu, S., Grow, J., et al. (2009). Mindfulness-based relapse prevention for substance use disorders: A pilot efficacy trial. *Substance Abuse, 30*(4), 295–305.

Brefczynski-Lewis, J. A., Lutz, A., Schaefer, H. S., Levinson, D. B., & Davidson, R. J. (2007). Neural correlates of attentional expertise in long-term meditation practitioners. *Proceedings of the National Academy of Sciences USA, 104*(27), 11483–11488.

Brewer, J. A., Worhunsky, P. D., Gray, J. R., Tang, Y. Y., Weber, J., & Kober, H. (2011). Meditation experience is associated with differences in default mode network activity and connectivity. *Proceedings of the National Academy of Sciences USA, 108*(50), 20254–20259.

Brown, K. W., Ryan, R. M., & Creswell, J. D. (2007). Mindfulness: Theoretical foundations and evidence for salutary effects. *Psychological Inquiry, 18*(4), 211–237.

Chiesa, A., & Serretti, A. (2009). Mindfulness-based stress reduction for stress management in healthy people: A review and meta-analysis. *Journal of Alternative and Complementary Medicine, 15*(5), 593–600.

Chiesa, A., & Serretti, A. (2010). A systematic review of neurobiological and clinical features of mindfulness meditations. *Psychological Medicine, 40*(8), 1239–1252.

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience, 3*(3), 201–215.

Crane, C., & Williams, J. M. (2010). Factors associated with attrition from mindfulness-based cognitive therapy in patients with a history of suicidal depression. *Mindfulness, 1*(1), 10–20.

Dobkin, P. L., Irving, J. A., & Amar, S. (2012). For whom may participation in a mindfulness-based stress reduction program be contraindicated? *Mindfulness, 3*(1), 44–50.

Eisendrath, S., Chartier, M., & McLane, M. (2011). Adapting mindfulness-based cognitive therapy for treatment-resistant depression: A clinical case study. *Cognitive and Behavioral Practice, 18*(3), 362–370.

Farb, N. A., Anderson, A. K., Bloch, R. T., & Segal, Z. V. (2011). Mood-linked responses in medial prefrontal cortex predict relapse in patients with recurrent unipolar depression. *Biological Psychiatry, 70*(4), 366–372.

Farb, N. A., Anderson, A. K., Mayberg, H., Bean, J., McKeon, D., & Segal, Z. V. (2010). Minding one's emotions: Mindfulness training alters the neural expression of sadness. *Emotion, 10*(1), 25–33.

Farb, N. A., Segal, Z. V., & Anderson, A. K. (2013). Mindfulness meditation training alters cortical representations of interoceptive attention. *Social Cognitive and Affective Neuroscience, 8*(1), 15–26.

Farb, N. A., Segal, Z. V., Mayberg, H., Bean, J., McKeon, D., Fatima, Z., et al. (2007). Attending to the present: Mindfulness meditation reveals distinct neural modes of self-reference. *Social Cognitive and Affective Neuroscience, 2*(4), 313–322.

Feldman, G., Greeson, J., & Senville, J. (2010). Differential effects of mindful breathing, progressive muscle relaxation, and loving-kindness meditation on decentering and negative reactions to repetitive thoughts. *Behaviour Research and Therapy, 48*(10), 1002–1011.

Fredrickson, B. L., & Branigan, C. (2005). Positive emotions broaden the scope of attention and thought–action repertoires. *Cognition and Emotion, 19*(3), 313–332.

Fresco, D. M., Segal, Z. V., Buis, T., & Kennedy, S. (2007). Relationship of posttreatment decentering and cognitive reactivity to relapse in major depression. *Journal of Consulting and Clinical Psychology, 75*(3), 447–455.

Frewen, P. A., Evans, E. M., Maraj, N., Dozois, D. J., & Partridge, K. P. (2008). Lettting go: Mindfulness and negative auotmatic thinking. *Cognitive Therapy and Research, 32*(6), 758–774.

Garland, E. L., Gaylord, S. A., & Fredrickson, B. L. (2011). Positive reappraisal mediates the stress-reductive effects of mindfulness: An upward spiral process. *Mindfulness, 2*(1), 59–67.

Gelenberg, A. J. (2010). The prevalence and impact of depression. *Journal of Clinical Psychiatry, 71*(3), e06.

Germer, C. K., Siegel, R. D., & Fulton, P. R. (2005). *Mindfulness and psychotherapy*. New York: Guilford Press.

Goldin, P., Ramel, W., & Gross, J. (2009). Mindfulness meditation training and self-referential processing in social anxiety disorder: Behavioral and neural effects. *Journal of Cognitive Psychotherapy, 23*(3), 242–257.

Goldin, P. R., & Gross, J. J. (2010). Effects of mindfulness-based stress reduction (MBSR) on emotion regulation in social anxiety disorder. *Emotion,10*(1), 83–91.

Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: Current status and future directions. *Annual Review of Clinical Psychology, 6*, 285–312.

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*(3), 281–291.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*(2), 348–362.

Grossman, P., Kappos, L., Gensicke, H., D'Souza, M., Mohr, D. C., Penner, I. K., et al. (2010). MS quality of life, depression, and fatigue improve after mindfulness training: A randomized trial. *Neurology, 75*(13), 1141–1149.

Grossman, P., Niemann, L., Schmidt, S., & Walach, H. (2004). Mindfulness-based stress reduction and health benefits. A meta-analysis. *Journal of Psychosomatic Research, 57*(1), 35–43.

Hayes, S. C. (2004). Acceptance and commitment therapy, relational frame theory, and the third wave of behavioral and cognitive therapies. *Behavior Therapy, 35*, 639–665.

Hofmann, S. G., Sawyer, A. T., Witt, A. A., & Oh, D. (2010). The effect of mindfulness-based therapy on anxiety and depression: A meta-analytic review. *Journal of Consulting and Clinical Psychology, 78*(2), 169–183.

Holzel, B. K., Lazar, S. W., Gard, T., Schuman-Olivier, Z., Vago, D. R., et al. (2011). How does mindfulness meditation work?: Proposing mechanisms of action from a conceptual and neutral perspective. *Perspectives on Psychological Science, 6*(6), 537–559.

Ingram, R. E., & Hollon, S. D. (1986). Information processing approaches to clinical psychology. In R. E. Ingram (Ed.), *Personality, psychopathology, and psychotherapy series* (Vol. xi, pp. 259–281). San Diego, CA: Academic Press.

Irving, J. A., Dobkin, P. L., & Park, J. (2009). Cultivating mindfulness in health care professionals: A review of empirical studies of mindfulness-based stress reduction (MBSR). *Complementary Therapies in Clinical Practice, 15*(2), 61–66.

Ivanovski, B., & Malhi, G. S. (2007). The psychological and neurophysiological concomitants of mindfulness forms of meditation. *Acta Neuropsychiatrica, 19*, 76–91.

Ives-Deliperi, V. L., Solms, M., & Meintjes, E. M. (2011). The neural substrates of mindfulness: An fMRI investigation. *Journal of Neuroscience, 6*(3), 231–242.

Jain, S., Shapiro, S. L., Swanick, S., Roesch, S. C., Mills, P. J., Bell, I., et al. (2007). A ran-

domized controlled trial of mindfulness meditation versus relaxation training: Effects on distress, positive states of mind, rumination, and distraction. *Annals of Behavioral Medicine, 33*(1), 11–21.

Jha, A. P., Krompinger, J., & Baime, M. J. (2007). Mindfulness training modifies subsystems of attention. *Cognitive, Affective, and Behavioral Neuroscience, 7*(2), 109–119.

Jha, A. P., Stanley, E. A., Kiyonaga, A., Wong, L., & Gelfand, L. (2010). Examining the protective effects of mindfulness training on working memory capacity and affective experience. *Emotion, 10*(1), 54–64.

Kabat-Zinn, J. (1982). An outpatient program in behavioral medicine for chronic pain patients based on the practice of mindfulness meditation: Theoretical considerations and preliminary results. *General Hospital Psychiatry, 4*(1), 33–47.

Kabat-Zinn, J. (1990). *Full catastrophe living: Using the wisdom of your body and mind to face stress, pain and illness*. New York: Delacorte.

Kabat-Zinn, J., Lipworth, L., & Burney, R. (1985). The clinical use of mindfulness meditation for the self-regulation of chronic pain. *Journal of Behavioral Medicine, 8*(2), 163–190.

Keightley, M. L., Seminowicz, D. A., Bagby, R. M., Costa, P. T., Fossati, P., & Mayberg, H. S. (2003). Personality influences limbic–cortical interactions during sad mood induction. *NeuroImage, 20*(4), 2031–2039.

Kelley, W. M., Macrae, C. N., Wyland, C. L., Caglar, S., Inati, S., & Heatherton, T. F. (2002). Finding the self?: An event-related fMRI study. *Journal of Cognitive Neuroscience, 14*(5), 785–794.

Kilpatrick, L. A., Suyenobu, B. Y., Smith, S. R., Bueller, J. A., Goodman, T., Creswell, J. D., et al. (2011). Impact of mindfulness-based stress reduction training on intrinsic brain connectivity. *NeuroImage, 56*(1), 290–298.

Kross, E., Gard, D., Deldin, P., Clifton, J., & Ayduk, O. (2012). "Asking why" from a distance: Its cognitive and emotional consequences for people with major depressive disorder. *Journal of Abnormal Psychology, 121*(3), 559–569.

Kumar, S., Feldman, G., & Hayes, A. (2008). Changes in mindfulness and emotion regulation in an exposure-based cognitive therapy for depression. *Cognitive Therapy and Research, 32*(6), 734–744.

Kupfer, D. J., Frank, E., Perel, J. M., Cornes, C., Mallinger, A. G., Thase, M. E., et al. (1992). Five-year outcomes for maintenance therapies in recurrent depression. *Archives of General Psychiatry, 49*, 769–773.

Kuyken, W., Watkins, E., Holden, E., White, K., Taylor, R. S., Byford, S., et al. (2010). How does mindfulness-based cognitive therapy work? *Behavioural Research and Therapy, 48*(11), 1105–1112.

Lakey, C. E., Kernis, M. H., Heppner, W. L., & Lance, C. E. (2008). Individual differences in authenticity and mindfulness as predictors of verbal defensiveness. *Journal of Research in Personality, 42*(1), 230–238.

Lau, M. A., Bishop, S. R., Segal, Z. V., Buis, T., Anderson, N. D., Carlson, L., et al. (2006). The Toronto Mindfulness Scale: Development and validation. *Journal of Clinical Psychology, 62*(12), 1445–1467.

Lazar, S. W., Bush, G., Gollub, R. L., Fricchione, G. L., Khalsa, G., & Benson, H. (2000). Functional brain mapping of the relaxation response and meditation. *NeuroReport, 11*(7), 1581–1585.

Leary, M. R., Tate, E. B., Adams, C. E., Allen, A. B., & Hancock, J. (2007). Self-compassion and reactions to unpleasant self-relevant events: The implications of treating oneself kindly. *Journal of Personality and Social Psychology, 92*(5), 887–904.

Lengacher, C. A., Kip, K. E., Barta, M. K., Post-White, J., Jacobsen, P., Groer, M., et al. (2012). A pilot study evaluating the effect of mindfulness-based stress reduction on psychological status, physical status, salivary cortisol, and interleukin-6 among advanced-stage cancer patients and their caregivers. *Journal of Holistic Nursing, 30*(3), 170–185.

Linehan, M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York: Guilford Press.

Liverant, G. I., Brown, T. A., Barlow, D. H., & Roemer, L. (2008). Emotion regulation in unipolar depression: The effects of acceptance and suppression of subjective emotional experience on the intensity and duration of sadness and negative affect. *Behavioural Research and Therapy, 46*(11), 1201–1209.

Lutz, A., Greischar, L. L., Perlman, D. M., & Davidson, R. J. (2009). BOLD signal in insula is differentially related to cardiac function during compassion meditation in experts vs. novices. *NeuroImage, 47*(3), 1038–1046.

Lutz, A., Slagter, H. A., Dunne, J. D., & David-

son, R. J. (2008). Attention regulation and monitoring in meditation. *Trends in Cognitive Sciences, 12*(4), 163–169.

Lutz, A., Slagter, H. A., Rawlings, N. B., Francis, A. D., Greischar, L. L., & Davidson, R. J. (2009). Mental training enhances attentional stability: Neural and behavioral evidence. *Journal of Neuroscience, 29*(42), 13418–13427.

Ma, S. H., & Teasdale, J. D. (2004). Mindfulness-based cognitive therapy for depression: Replication and exploration of differential relapse prevention effects. *Journal of Consulting and Clinical Psychology, 72*(1), 31–40.

Marlatt, G. A., & Gordon, J. R. (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors.* New York: Guilford Press.

Moore, S. A., Zoellner, L. A., & Mollenholt, N. (2008). Are expressive suppression and cognitive reappraisal associated with stress-related symptoms? *Behavioural Research and Therapy, 46*(9), 993–1000.

Morone, N. E., Greco, C. M., & Weiner, D. K. (2008). Mindfulness meditation for the treatment of chronic low back pain in older adults: A randomized controlled pilot study. *Pain, 134*(3), 310–319.

Neacsiu, A. D., Bohus, M., & Linehan, M. M. (this volume, 2014). Dialectical behavior therapy: An intervention for emotion dysregulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 491–507). New York: Guilford Press.

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology, 109*(3), 504–511.

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14*(8), 1215–1229.

Ohman, A., Flykt, A., & Esteves, F. (2001). Emotion drives attention: Detecting the snake in the grass. *Journal of Experimental Psychology: General, 130*(3), 466–478.

Oken, B. S., Fonareva, I., Haas, M., Wahbeh, H., Lane, J. B., Zajdel, D., et al. (2010). Pilot controlled trial of mindfulness meditation and education for dementia caregivers. *Journal of Alternative and Complementary Medicine, 16*(10), 1031–1038.

Ottenbreit, N. D., & Dobson, K. S. (2004). Avoidance and depression: The construction of the Cognitive-Behavioral Avoidance Scale. *Behavioural Research and Therapy, 42*(3), 293–313.

Pauley, G., & McPherson, S. (2010). The experience and meaning of compassion and self-compassion for individuals with depression or anxiety. *Psychology and Psychotherapy: Theory, Research and Practice, 83*(Pt. 2), 129–143.

Paykel, E. S., Scott, J., Teasdale, J. D., Johnson, A. L., Garland, A., Moore, R., et al. (1999). Prevention of relapse in residual depression by cognitive therapy: A controlled trial. *Archives of General Psychiatry, 56*(9), 829–835.

Perlman, D. M., Salomons, T. V., Davidson, R. J., & Lutz, A. (2010). Differential effects on pain intensity and unpleasantness of two meditation practices. *Emotion, 10*(1), 65–71.

Piet, J., & Hougaard, E. (2011). The effect of mindfulness-based cognitive therapy for prevention of relapse in recurrent major depressive disorder: A systematic review and meta-analysis. *Clinical Psychology Review, 31*(6), 1032–1040.

Ramel, W., Goldin, P. R., & Carmona, P. E. (2004). The effects of mindfulness meditation on cognitive processes and affect in patients with past depression. *Cognitive Therapy and Research, 28*, 433–455.

Rosenzweig, S., Greeson, J. M., Reibel, D. K., Green, J. S., Jasser, S. A., & Beasley, D. (2010). Mindfulness-based stress reduction for chronic pain conditions: Variation in treatment outcomes and role of home meditation practice. *Journal of Psychosomatic Research, 68*(1), 29–36.

Scher, C. D., Ingram, R. E., & Segal, Z. V. (2005). Cognitive reactivity and vulnerability: Empirical evaluation of construct activation and cognitive diatheses in unipolar depression. *Clinical Psychology Review, 25*(4), 487–510.

Schmertz, S. K., Anderson, P. L., & Robins, D. L. (2009). The relation between self-report mindfulness and performance on tasks of sustained attention. *Behavioral Science, 31*(1), 60–66.

Segal, Z. V., Bieling, P., Young, T., MacQueen, G., Cooke, R., Martin, L., et al. (2010). Antidepressant monotherapy vs sequential pharmacotherapy and mindfulness-based cognitive therapy, or placebo, for relapse prophylaxis in recurrent depression. *Archives of General Psychiatry, 67*(12), 1256–1264.

Segal, Z. V., Kennedy, S., Gemar, M., Hood, K., Pedersen, R., & Buis, T. (2006). Cognitive

reactivity to sad mood provocation and the prediction of depressive relapse. *Archives of General Psychiatry, 63*(7), 749–755.

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression: A new approach to preventing relapse*. New York: Guilford Press.

Semple, R. J., Lee, J., Rosa, D., & Miller, L. F. (2010). A randomized trial of mindfulness-based cognitive therapy for children: Promoting mindful attention to enhance social–emotional resiliency in children. *Journal of Child and Family Studies, 19*, 218–229.

Shapiro, S. L., Brown, K. W., Thoresen, C., & Plante, T. G. (2011). The moderation of mindfulness-based stress reduction effects by trait mindfulness: Results from a randomized controlled trial. *Journal of Clinical Psychology, 67*(3), 267–277.

Shapiro, S. L., Carlson, L. E., Astin, J. A., & Freedman, B. (2006). Mechanisms of mindfulness. *Journal of Clinical Psychology, 62*(3), 373–386.

Shapiro, S. L., & Schwartz, G. E. (2000). Intentional systemic mindfulness: An integrative model for self-regulation and health. *Advances in Mind–Body Medicine, 16*(2), 128–134.

Sheppes, G. (this volume, 2014). Emotion regulation choice: Theory and findings. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 126–139). New York: Guilford Press.

Silananda, S. U. (1998). *The four foundations of mindfulness (A summary)*. Retrieved November 18, 2012, from *www.buddhanet.net/budsas/ebud/ebmed063.htm*.

Silananda, S. U. (2002). *The four foundations of mindfulness* (2nd ed.). Boston: Wisdom.

Smith, A., Graham, L., & Senthinathan, S. (2007). Mindfulness-based cognitive therapy for recurring depression in older people: A qualitative study. *Aging and Mental Health, 11*(3), 346–357.

Teasdale, J. D., & Chaskalson, M. (2011). How does mindfulness transform suffering?: II. The transformation of Dukka. *Contemporary Buddhism: An Interdisciplinary Journal, 12*(1), 103–124.

Teasdale, J. D., Segal, Z. V., Williams, J. M., Ridgeway, V. A., Soulsby, J. M., & Lau, M. A. (2000). Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *Journal of Consulting and Clinical Psychology, 68*(4), 615–623.

Trew, J. L. (2011). Exploring the roles of approach and avoidance in depression: An integrative model. *Clinical Psychology Review, 31*(7), 1156–1168.

Walach, H., Buchheld, N., Buttenmuller, V., Kleinknecht, N., & Schmidt, S. (2006). Measuring mindfulness—The Freiburg Mindfulness Inventory (FMI). *Personality and Individual Differences, 40*, 1543–1555.

Watkins, E., Moberly, N. J., & Moulds, M. L. (2008). Processing mode causally influences emotional reactivity: Distinct effects of abstract versus concrete construal on emotional response. *Emotion, 8*(3), 364–378.

Williams, J. M. G. (2007). *The mindful way through depression: Freeing yourself from chronic unhappiness*. New York: Guilford Press.

Williams, K. A., Kolar, M. M., Reger, B. E., & Pearson, J. C. (2001). Evaluation of a wellness-based mindfulness stress reduction intervention: A controlled trial. *American Journal of Health Promotion, 15*(6), 422–432.

Witkiewitz, K., Bowen, S., Douglas, H., & Hsu, S. H. (2013). Mindfulness-based relapse prevention for substance craving. *Addictive Behaviors, 38*(2), 1563–1571.

Zeidan, F., Gordon, N. S., Merchant, J., & Goolkasian, P. (2010). The effects of brief mindfulness meditation training on experimentally induced pain. *Journal of Pain, 11*(3), 199–209.

PART IX

HEALTH IMPLICATIONS

CHAPTER 33

Emotion Regulation and Gene Expression

Steven W. Cole

Inhibiting the expression of emotions has long been believed to undermine physical health, and recent developments in our ability to map neural–immune interactions at the genomic level have begun to identify potential biological bases for such effects. This chapter reviews basic research linking *emotional inhibition* (or *suppression*; I use the terms interchangeably) to physical illness and outlines an emerging theoretical paradigm in "social genomics" that sheds light on both the biological mechanisms of such effects and their potential evolutionary origin. This perspective construes emotion regulation as one component of a broader set of biobehavioral adaptations that have emerged in response to the evolution of a hypersocial "life history strategy" for humans (Darwin, 1872; McDade, 2003; Richerson, Boyd, & Henrich, 2010; Wilson, 2012). The social–neural–genomic programs that evolved to mediate this adaptation likely held little health cost under primordial conditions in which trauma and infection were our primary causes of death (Finch, 2010). However, in the very different social, physical, and cultural environments that we now inhabit, these biological programs may functionally connect everyday "civilized" social behavior to low-level anticipatory threat reactions that can aggravate many drivers of "modern mortality," such as cardiovascular, neurodegenerative, metabolic, and neoplastic diseases (Finch, 2007; Sterling, 2004).

Emotion Regulation and Human Health

As far back as the second century A.D., the Greek physician Galen noted that people who were emotionally inexpressive seemed to be at increased risk of cancer (Siegel, 1968). Subsequent epidemiological studies underscored this observation (Gross, 1989; Mund & Mitte, 2012) and expanded the range of health risks associated with emotional inhibition to include cardiovascular and immune-related diseases (Bell, Jasnoski, Kagan, & King, 1990; Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997; Cohen, Doyle, Turner, Alper, & Skoner, 2003; Cole, Kemeny, & Taylor, 1997; Cole, Kemeny, Taylor, & Visscher, 1996; Denollet, Gidron, Vrints, & Conraads, 2010; Denollet, Pedersen, Vrints, & Conraads, 2006; Denollet et al., 1996; Ironson, O'Cleirigh, Weiss, Schneiderman, & Costa, 2008; Kagan, 1994; Kagan, Snidman, Julia-Sellers, & Johnson, 1991; Mund & Mitte, 2012). Recent experimental studies also suggest that the active expression of emotions can mirror the effects of suppression and reduce

the risk of minor illnesses (Booth, 2012; Frattaroli, 2006; Pennebaker, 1988). The disease risks associated with emotional inhibition range widely in their specific biology and in their overall health significance, but they share in common one basic teleological puzzle: Why has the human body evolved to lose vitality when we inhibit the expression of our emotions?

Surprisingly, mechanistic answers to this question have emerged more rapidly than true functional explanations. It is now easer to understand how emotional inhibition might affect human health than why that should be so. Much of our growing mechanistic insight stems from dramatic technical advances in molecular genetics, and our growing ability to understand physical disease in terms of changing patterns of human gene expression (Cole, 2009; Finch, 2007; Miller, Chen, & Cole, 2009). The equation of disease with gene expression dynamics represents an oversimplification to be sure. However, construing health and disease through the lens of molecular genetics has the significant heuristic advantage of connecting these dynamics to theoretical and teleological perspectives derived from evolutionary biology. Harnessing the tremendous technical infrastructure of molecular genomics offers opportunities to understand not only how social inhibition affects health but also why that connection should have emerged in the first place. The keys to realizing this opportunity lie in understanding how genes change their behavior in response to our psychological experiences.

Genetics versus Genomics

As genes have come to be understood as concrete DNA sequences rather than abstractions inferred from inheritance, it has become increasingly apparent that social and psychological factors can regulate the activity or *expression* of human genes (Cole, 2009). DNA encodes the potential for cellular behavior, but that potential is only realized if the gene is expressed—if the DNA is transcribed into RNA (Figure 33.1). It is RNA and its subsequent translation into protein that shapes a cell's structure and identity, and endows its functional capacities such as movement, metabolism, and biochemical response to external stimuli (e.g., neurotransmission or immune response). Absent their transcription, DNA genes have no effect on health or behavioral phenotypes. With the advent of a sequenced human genome and the emergence of DNA microarray and sequencing technologies, scientists can now survey the expression of all human genes simultaneously and map the specific subset of genes that is actively expressed in RNA within a given cell at a given point in time. One surprising finding from such studies of *functional genomics* is that the expression of a given gene is often more an exception than the rule. Cells are highly selective about which genes they express, and our DNA encodes a great deal more genetic potential than is realized in RNA and protein (Djebali et al., 2012). As such, the subset of genes that is expressed in RNA—the cell's *transcriptome*—can be interpreted as the product of a cellular decision-making process regarding which particular sets of genes would be most adaptive to express under the prevailing circumstances. Mechanistically, these decisions are mediated by protein *transcription factors* that bind to specific DNA sequences in the *regulatory region* (or promoter) of a gene. Most transcription factors target a large number of genes that contribute in different and often partially overlapping ways to a common biological process (e.g., multiple genes encoding receptors, signaling molecules, and transcription factors that collectively allow a particular cell to recognize and respond to a neurotransmitter or microbe). As such, transcription factors are best construed as activating general gene transcriptional programs or *gene modules* rather than acting on specific individual genes in isolation.

What evolutionary theory adds to this mechanistic picture is a set of principles for determining which genes should be coactivated by a given transcription factor (i.e., the inclusion–exclusion structure or intension of a particular gene module) and which transcription factors should be activated in response to the stimuli present in the cell's environment. In other words, transcription factors and their "target gene modules" constitute a biochemical stimulus–response program, and the development of the whole system of such programs encoded in a genome represents the product of evolution-

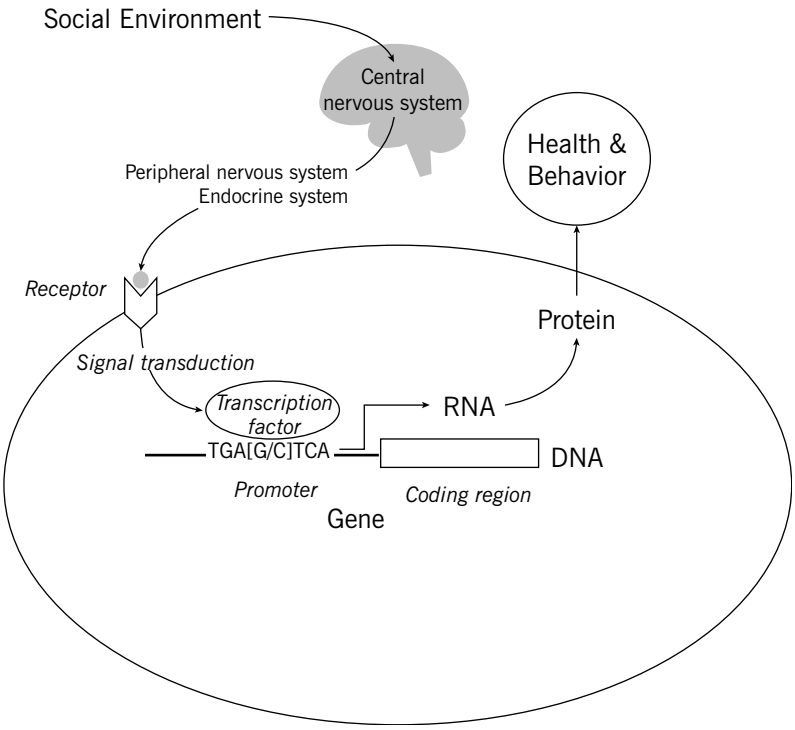

FIGURE 33.1. Social signal transduction. Socioenvironmental processes regulate human gene expression by activating CNS processes that subsequently influence hormone and neurotransmitter activity in the periphery of the body. Peripheral signaling molecules interact with cellular receptors to activate transcription factors, which bind to characteristic DNA motifs in gene promoters to initiate (or repress) gene expression. Only genes that are transcribed into RNA actually impact health and behavioral phenotypes. Individual differences in promoter DNA sequences (e.g., the [G/C] polymorphism shown here) can affect the binding of transcription factors and thereby influence genomic sensitivity to socioenvironmental conditions. Adapted from Cole (2009).

ary trial and error in adapting to changing environmental conditions. Some "constitutive" transcription factors show steady levels of activity and act mainly to continue the expression of genes that confer a cell's particular biological identity (e.g., maintaining the expression of neuron-defining genes in a particular neuron, so it continues to be a neuron). Other transcription factors are "inducible" and can dramatically up-regulate their activity from almost total quiescence to levels 10–100 times above baseline. These inducible transcription factors act mainly to alter gene expression in response to changing environmental conditions as detected by cellular receptor systems (e.g., increasing the expression of immune response genes when a microbe is detected, or changing gene expression profiles in response to cellular growth factors, hormones, or neurotransmitters).

For both types of transcription factor, the mapping of an individual factor onto its distinctive subset of "target genes" confers a sort of biological "meaning" to the transcription factor—an intension set of transcriptional responses that presumably have helped the organism thrive under basal conditions and adapt to emerging challenges. This connection between the biological characteristics of coregulated gene modules and the nature of the upstream transcription factors and receptor systems that activate those gene modules provides a conceptual strategy for extracting organismic-level meaning and teleological design principles from genomewide transcriptional profiles of RNA (Cole, 2010). These mappings reflect

a sort of evolutionarily acquired programming, or a "wisdom of the genome" regarding which genes are most adaptive to express in a given type of environment.

Accumulating data on the specific receptor systems that activate particular transcription factors also allow a set of more specific inferences regarding the extracellular physiological signaling pathways that may trigger observed changes in the cellular transcriptome. These mappings allow us, for example, to translate genomewide transcriptional profiles into theories regarding the specific hormones and neurotransmitters that may distribute central nervous system (CNS)-mediated experiences of social or emotional stimuli into changes in the peripheral gene expression dynamics that underlie health and disease (see Ochsner & Gross; Proudfit, Dunning, Foti, & Weinberg; Johnstone & Walter; and Gyurak & Etkin, this volume, for more on the CNS mechanisms of emotion regulation). These bioinformatics projections essentially reverse the normal flow of biological information from outside the body to inside (Figure 33.1), thus providing a "bottom-up" strategy for detecting the physiological signaling pathways that may mediate the effects of emotion regulation on health. Such approaches can complement a more "top-down" strategy that surveys known health-relevant peripheral neural and endocrine signals, such as the sympathetic nervous system (SNS) or hypothalamic–pituitary–adrenal (HPA) axis (Sapolsky, 1994), to determine their potential contributions to the health effects of emotional suppression. So, where might the top-down effects of emotional suppression meet up with the bottom-up programming of the human genome?

Emotion Regulation and Peripheral Neural Function

Some of the earliest research in human psychophysiology found that people who were relatively inexpressive of their emotions nevertheless showed elevated autonomic responses to stressful or threatening stimuli (Buck, 1979; Jones, 1935, 1950, 1960; Notarius & Levenson, 1979; Weinberger, Schwartz, & Davidson, 1979). More recent studies have also noted inhibition-related differences in basal levels of SNS activity (Cole, Kemeny, Fahey, Zack, & Naliboff, 2003; Kagan, 1994; Kagan, Reznick, & Snidman, 1988), suggesting that dispositional differences in peripheral autonomic activity might potentially act on a tonic, daily basis to alter basal physiological homeostasis. A growing body of experimental research largely parallels findings from the individual-differences literature in linking emotional suppression to increased SNS activity. Asking people to inhibit the expression or experience of an emotion in response to emotionally arousing stimuli can increase SNS activity (Dan-Glauser & Gross, 2011; Gross, 1998, 2002; Gross & Levenson, 1993, 1997; Harris, 2001; Jackson, Malmstadt, Larson, & Davidson, 2000; Lissek et al., 2007; Pennebaker & Chew, 1985; Robinson & Demaree, 2009), and actively inducing emotional expressions in written or spoken form can reduce inhibition-related SNS responses (Hughes, Uhlmann, & Pennebaker, 1994; Pennebaker, Barger, & Tiebout, 1989; Pennebaker, Hughes, & O'Heeron, 1987) and transiently increase indicators of physical health (e.g., self-reported illnesses or objectively reported health care utilization, vaccine-induced antibody responses, wound healing, and immune system biological parameters; Booth, 2012).

Although some inconsistent results exist, the majority of published research suggests that emotional suppression is associated with SNS activation. Moreover, in the one study of individual differences that has simultaneously assessed psychological inhibition, SNS activity, and biological indicators of disease progression, individual differences in SNS activity emerged as a statistically plausible mediator of relationships between psychological inhibition and HIV-1 viral load (Cole, 2008; Cole et al., 2001, 2003). A key role for the SNS is also supported by a growing body of data showing that other manipulations of SNS activity can aggravate a wide variety of disease processes, including the growth and metastasis of cancer (Cole & Sood, 2012; Sloan et al., 2010; Thaker et al., 2006). In fact, it was in studies of disease pathogenesis that the human genome was first found to be modulated in a broad and systematic way by psychological processes and their effects on the autonomic nervous system.

Neural Regulation of Human Gene Expression

The potential for psychosocial regulation of human gene expression first emerged in the context of studies analyzing the effect of social stress and SNS neurotransmitters on viral genomes such as herpes simplex viruses (Glaser, Kiecolt-Glaser, Speicher, & Holliday, 1985; Jenkins & Baum, 1995; Kupfer & Summers, 1990; Leib, Nadeau, Rundle, & Schaffer, 1991; Padgett et al., 1998; Rasmussen, Marsh, & Brill, 1957; Schuster, Chasserot-Golaz, Urier, Beck, & Sergeant, 1991), human immunodeficiency virus (HIV-1; Capitanio, Mendoza, Lerche, & Mason, 1998; Cole et al., 1997; Cole, Kemeny, Taylor, Visscher, & Fahey, 1996; Sloan et al., 2007), Epstein–Barr virus (Glaser et al., 1985; Yang et al., 2010), cytomegalovirus (Glaser et al., 1985; Prosch et al., 2000), and the Kaposi's sarcoma-associated human herpesvirus 8 (Chang et al., 2005). As obligate parasites of human host cells, viruses have evolved within a microenvironment structured by our own genome. If social processes and SNS neurotransmitters can regulate the expression of viral genes, it stands to reason that our own complement of roughly 21,000 genes might be modulated as well.

Over the past 5 years, a series of genome-wide transcriptional profiling studies has found that extended periods of psychological or social stress are often associated with a specific pattern of change in gene expression within immune cells. Across several distinct types of adversity such as social isolation (Cole, Hawkley, Arevalo, & Cacioppo, 2011; Cole et al., 2007; Creswell et al., 2012), imminent bereavement (Miller et al., 2008), low socioeconomic status (SES; Chen, Miller, Kobor, & Cole, 2011; Chen et al., 2009), early life social deprivation (Miller, Chen, Fok, et al., 2009), late life social adversity (Cole et al., 2010), traumatic stress (O'Donovan et al., 2011), diagnosis with a life-threatening illness (Antoni et al., 2012; Cohen et al., 2012), and experimentally imposed social threat (Cole et al., 2010; Cole, Arevalo, Ruggerio, Heckman, & Suomi, 2012; Sloan et al., 2007, 2010), circulating immune cells (leukocytes) show a *conserved transcriptional response to adversity* (CTRA) involving increased expression of genes involved in inflammation (e.g., *IL1B, IL6, IL8, TNF*) and decreased expression of genes involved in innate antiviral responses (*IFNB* and *IFI, MX*, and *OAS* gene families), and the production of a specific type of antibody (immunoglobulin G1 [IgG1]) (Cole, 2009, 2010; Irwin & Cole, 2011; Miller, Chen, & Cole, 2009). Each type of adversity is also associated with other transcriptional alterations that are relatively unique to that condition. However, this core CTRA pattern of proinflammatory and anti-antiviral transcriptome shift has emerged much more consistently than would be expected by chance. Similar patterns also emerge in response to experimentally imposed adversity in animal models of social instability, low social rank, and social threat or defeat (Cole et al., 2010, 2012; Irwin & Cole, 2011; Sloan et al., 2007; Tung et al., 2012).

Given the statistical challenges of multiple hypothesis testing across roughly 21,000 genes, different "social genomics" studies rarely find identical sets of differentially expressed genes. Where consistent patterns do become salient is in subsequent bioinformatic analyses extracting common functional themes from the lists of tens to hundreds of differentially expressed genes in each study (e.g., Gene Ontology annotations regarding shared biological functions and analyses of transcription control pathways regulating expression of multiple genes) (Cole, 2010). The recurrence of these core proinflammatory–anti-antiviral biological themes across both different adverse environments and different mammalian species suggests that the immune system of social mammals is programmed to generate a CTRA response whenever individuals experience extended periods of stress, threat, or uncertainty (Antoni et al., 2012; Cole et al., 2012; Irwin & Cole, 2011). This general transcriptional program may be expressed somewhat variably at the level of individual gene transcripts depending upon specifics of individual history, genetic background, and particulars of the current environment (Cole, 2010). However, the broader biological principle of *increase inflammation and suppress interferons and IgG1* seems to be programmed into the basic stimulus–response logic of the human genome (at least as expressed in the receptor/transcription factor logic of leukocytes).

A key role for psychological experience in triggering the CTRA dynamic is suggested by results from several small randomized controlled experiments showing that stress-reducing interventions can reverse CTRA transcriptional dynamics in human immune cells (Antoni et al., 2012; Black et al., 2013; Creswell et al., 2012). A key role for the SNS in mediating these effects is suggested by cellular studies showing that CTRA-related transcriptional dynamics can be mimicked by pharmacological agents that activate the beta-adrenergic receptor systems targeted by SNS neurotransmitters and can be blocked by pharmacological antagonists of those receptors (Cole et al., 2010; Collado-Hidalgo, Sung, & Cole, 2006; Hanke, Powell, Stiner, Bailey, & Sheridan, 2012; Wohleb et al., 2011). Moreover, the specific pattern of up-regulated inflammatory gene expression and down-regulated antiviral- and antibody-related gene expression observed in studies of human social adversity maps very closely to the effects of SNS pharmacology in laboratory experimental studies, whereas other stress-responsive systems that might potentially mediate such effects, such as glucocorticoid hormone release from the HPA axis, activate very different gene expression programs (Irwin & Cole, 2011). In fact, several studies have implicated a selective reduction in glucocorticoid signaling in contributing to the proinflammatory component of the CTRA profile (Cole et al., 2007; Hanke et al., 2012; Miller et al., 2008; Wohleb et al., 2011). These observations do not rule out a potential effect of glucocorticoids in other types of stress-mediated gene expression dynamics (e.g., responses to more severe, overwhelming stressors that elicit defeat/withdrawal responses; Henry, 1992; Lundberg & Frankenhaeuser, 1980; Sapolsky, 1994). However, the general patterns of transcriptional alteration associated with the chronic and highly prevalent socioenvironmental risk factors for human disease (social isolation, low SES, etc.) appear to parallel most closely the profiles of gene expression induced by beta-adrenergic receptor signaling in experimental cellular and animal models (Irwin & Cole, 2011).

Why should CTRA-related gene modules have evolved to become sensitive to beta-adrenergic signaling from the SNS? CTRA transcriptional dynamics appear to represent an evolutionarily adaptive "defensive program" that redeploys the immune system's transcriptional resources to "allostatically anticipate" (Sterling, 2004) the changing patterns of microbial exposure historically associated with changing life circumstances (e.g., increased risk of wound-related bacterial infection during periods of acute threat vs. increased risk of viral contagion during extended periods of close social contact; Cole et al., 2011; Irwin & Cole, 2011). Because antiviral and proinflammatory gene modules are, to some extent, mutually exclusive (Amit et al., 2009; Negishi et al., 2012; O'Connell et al., 2004; Shahangian et al., 2009; Tian et al., 2012), the immune system must "choose" which gene module to favor at any given time. The CTRA dynamic suggests that that choice is informed in part by the broader physiological and environmental conditions surrounding the individual (i.e., organism-level adaptive fitness), as perceived by the CNS and distributed into peripheral tissues via the SNS (Cole et al., 2011; Irwin & Cole, 2011). However, when the CTRA defensive program is chronically stimulated, the resulting proinflammatory–anti-antiviral shift in leukocyte transcriptional equilibrium may promote the complex pattern of "modern mortality" diseases involving elements of both up-regulated immune function (e.g., inflammation-related diseases such as heart disease, neurodegenerative diseases, and some types of cancer) and down-regulated immune function (e.g., impaired response to vaccines and viral infections) (Finch, 2007). The complex pattern of up- and down-regulated gene modules associated with the CTRA underscores the fact that stress is not broadly immunosuppressive, but instead selectively suppresses some groups of immune response genes (e.g., Type I interferons and some immunoglobulin genes) while simultaneously activating others (e.g., proinflammatory cytokines) (Irwin & Cole, 2011).

Beyond the CTRA dynamic in leukocytes, stress biology can also regulate gene expression in a wide variety of other tissues, including the CNS (Karelina et al., 2009; Karssen et al., 2007; Weaver, Meaney, & Szyf, 2006), peripheral immune system organs such as the lymph nodes and spleen (Cole et al., 2010; Sloan et al., 2007), and diseased tissues such as ovarian cancers and

stroke-wounded brain tissues (Karelina et al., 2009; Lutgendorf et al., 2009). Given the much smaller number of social genomics analyses targeting such solid tissues, and the relative difficulty in ascertaining the functional significance of specific transcriptional alterations outside the well-charted territories of the immune response, it has been much more difficult to map the specific gene programs modulated in these tissues and to deduce their associated teleological rationale (e.g., are these tissue "defensive programs" analogous to the leukocyte CTRA, or do they represent some other type of functional adaptation specific to the organ system involved?). However, it is clear that the social environment surrounding an individual can modulate broad swaths of gene activity in a diverse array of bodily tissues via CNS-mediated interpretations of the environment as safe/accommodating or threatening/hostile/uncertain.

Emotion Regulation and Gene Regulation

At the time of this writing, no published studies have directly examined the effects of emotional inhibition on human gene expression. However, coupling the two domains of research reviewed here suggests a natural hypothesis regarding how chronic emotional suppression might influence physical health via systematic changes in gene expression. To the extent that (1) emotional suppression activates the SNS and (2) the SNS activates gene expression programs such as the CTRA, chronic emotional suppression may induce a repetitive or temporally extended bias in basal gene expression profiles that simultaneously increases vulnerability to inflammation-related diseases (e.g., cardiovascular and neoplastic diseases) and decreases immune response to vaccines and viral infections. Assuming this plausible conjunction holds true empirically, emotion regulation can be construed as an indirect form of gene regulation. Moreover, the specific pattern of gene expression modulations that would be predicted based on this hypothesis corresponds quite closely to the profile of specific disease risks that has been linked to inhibition in empirical epidemiological studies. Beyond providing a plausible biological mechanism for effects of emotion regulation on human health (Miller, Chen, & Cole, 2009), however, this equation of emotion regulation with gene regulation also suggests a broader set of questions regarding the origins and consequences of emotion regulation in general physiological function (i.e., independent of its impacts on somatic disease).

One distinctive feature of gene transcription as an "output" from psychological processes is its simultaneous role as an "input" into the future biological characteristics of the individual (Cole, 2009). Genes can act recursively in the sense that they alter the nature of the molecular machinery that controls their own expression. Figure 33.2 outlines some of the developmental implications of such recursion in allowing environmental conditions (including psychological perceptions and emotional expression) to become embedded in the molecular biology of the individual in ways that affect subsequent perceptual, emotional, and neurobiological responses to future environmental conditions. For example, if one period of emotional suppression results in altered patterns of gene expression in the brain, and some of those regulated genes include molecules that mediate the perception of social threat and its transduction into gene expression (receptors, signal transduction molecules, transcription factors, etc.), then the individual's subsequent psychological response to a fixed social or emotional stimulus may well differ from what it would have been otherwise, purely as a function of that individual's history of exposures earlier in life. This recursive feedback dynamic provides one biological pathway by which early life social and psychological conditions can become embedded in an individual's basic neurobiological characteristics and subsequently influence later patterns of psychological experience and biological development (Cole, 2009). Recursive feedback dynamics also help clarify how particularly acute transient events can induce persisting alterations in neurobiological homeostasis (e.g., as in posttraumatic stress disorder [PTSD]).

Because genes can recursively regulate their own molecular triggers and production machinery, there exists great potential for nonlinear regulatory dynamics in which initially small stimuli have quantitatively

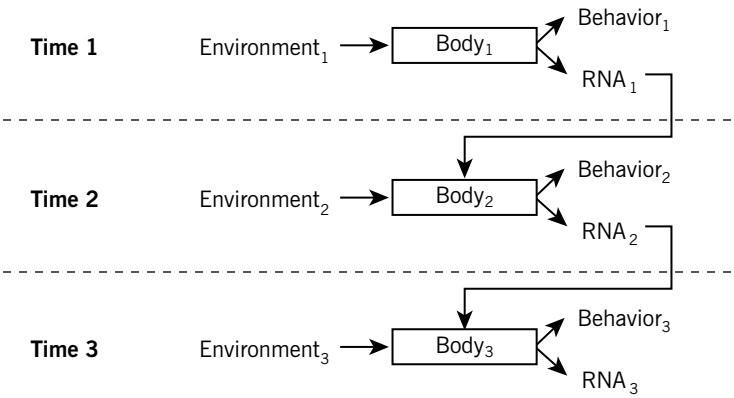

FIGURE 33.2. RNA as a molecular medium of recursive development. Social conditions at one point in time ($Environment_1$) are transduced into changes in behavior ($Behavior_1$) and gene expression (RNA_1) via CNS perceptual processes that trigger systemic neural and endocrine responses (mediated by $Body_1$). Those RNA transcriptional dynamics can alter the molecular characteristics of cells involved in environmental perception or response, resulting in a functionally altered $Body_2$. $Body_2$ may respond differently to a given environmental challenge than would the previous $Body_1$, resulting in different behavioral ($Behavior_2$) and transcriptional responses (RNA_2). The effect of RNA transcriptional dynamics on persisting cellular protein and functional characteristics provides a molecular framework for understanding how socioenvironmental conditions in the past may continue to affect current behavior and health, and how those historical conditions interact with current environments to shape our future trajectories (e.g., $Body_3$, $Behavior_3$, RNA_3). Because gene transcription serves as both a cause of social behavior (by shaping Body) and a consequence of social behavior (a product of Environment × Body), RNA serves as the physical medium for a recursive developmental trajectory that integrates genetic characteristics and historical–environmental regulators to shape individual biological and behavioral responses to current environmental conditions. Adapted from Cole (2009).

or temporally disproportionate effects. Whether this actually happens or not in any given situation depends largely on the feedback structure of the specific gene networks involved. To the extent that receptors, signal transduction molecules, and transcription factors number among those genes empirically modulated by a given stimulus, some form of feedback recursion is likely to occur. However, recursion alone does not guarantee nonlinear amplification. Whether that feedback is positive (amplifying), negative (damping), or nonexistent depends largely on the empirical pattern of connections among gene products, or the "wiring diagram" of the human genome as expressed in a given cell type (i.e., the specific set of receptors, signaling molecules, and transcription factors expressed in a given cell, or potentially induced in that cell in response to environmental stimuli). Outside of the immune system, the wiring diagram of the human genome remains almost completely unknown. However, as studies of CNS gene expression grow in number, sophistication, and neuroanatomical precision, new insights will rapidly emerge regarding the recursive architecture of the genome as expressed in the brain structures mediating emotional experience and behavior. Even in the absence of ideal longitudinal studies that directly map system dynamics, much may be learned by simply observing the pattern of changes induced by a single stimulus event. To the extent that stimulus changes the expression of genes with known recursive capacity (e.g., genes encoding receptors, signal transduction molecules, transcription factors, or epigenetic modulators) the potential for feedback and nonlinear dynamics becomes substantially greater, and the specific pattern of up- and down-regulated recursive molecules can provide some indications regarding the positive versus negative feedback elements of the system. As those wiring diagrams become increasingly

well mapped, the network structure of such systems may identify particularly influential recursive regulators that would provide highly leveraged targets for interventions to block the temporally propagating effects of early life adversity and acute trauma in adult life. Beyond such clinical implications, there also emerges a much broader set of questions regarding how the gene regulation inherent in emotion regulation might developmentally modify the "behavior factory" of the CNS, and the role that occasional tactical suppression may play in laying a molecular foundation for chronic suppression or dampened emotional experience.

Given that emotion regulation is an intrinsically social adaptation (Darwin, 1872), how might suppression affect an individual's social relationships? A growing body of evidence suggests that chronic emotional suppression may significantly undermine the number and quality of one's social connections to others (Butler et al., 2003; English, John, Srivastava, & Gross, 2012; Gross, 2002; Gross & John, 2003; Mauss et al., 2011; Srivastava, Tamir, McGonigal, John, & Gross, 2009). Experiences of social connection vs. disconnection can also affect gene expression (Cole et al., 2007, 2011), and were in fact the context in which the CTRA gene expression dynamic was first observed. As such, the health effects of emotional suppression may stem at least in part from their effects in reducing an individual's sense of social connection to others. Indeed, microacts of emotional suppression may kindle a broader change in other people's regard for the individual that comes to assume a temporal scope and situational breadth much greater than original biological insult created by a transient act of suppression. In this sense, the most significant biological implications of emotion regulation may well stem from its impact on an individual's social identity and degree of healthy attachment to others. Moreover, individual acts of emotional suppression can also activate the SNS in surrounding social partners (Butler et al., 2003), implying a contagious dimension extending beyond the suppressor's own body. A wide variety of theoretical perspectives note the fundamental role of secure social bonds in maintaining human health and well-being (Berkman & Kawachi, 2000; Bowlby, 1983; Cacioppo & Hawkley, 2009; Hofer, 1984). To the extent that emotional suppression undermines one's sense of true connection to others and more general faith in humanity, the social costs of suppression may well represent its most caustic health effect.

The bulk of the research on emotion regulation and health has focused on the long-observed relationship between emotional suppression and illness. However, it is worth considering how health and biology might be affected by the other broad class of emotion regulation strategies involving reframing or reappraisal (Gross, 2002). Much of the psychophysiological research suggests that reappraisal has relatively little impact on SNS activity (Gross, 2002; Gross & Levenson, 1993), and the equation of gene regulation with SNS activation might thus be taken to imply that reappraisal-based emotion regulation has relatively little effect on gene expression and health. A separate literature on the psychology of stress and the role of reappraisal-based coping strategies would also support that hypothesis (Lazarus & Folkman, 1984; Sapolsky, 1994). However, at the level of the CNS, where gene expression dynamics may also play a role in establishing the basic machinery of perception, interpretation, and behavioral response, it does seem likely that the habitual use of reappraisal strategies may potentially affect the molecular biology of neural development and plasticity. This might be particularly true within the brain structures involved in appraisal itself, the structures involved in metacognition or executive function processes that would control the deployment of reappraisal processes into the ongoing stream of conscious experience, and potentially also in the neurobiological structures that mediate stress responses (e.g., involving functional down-regulation or desensitization of these structures). To the extent that habitual and adaptive reappraisal processes build a different brain at the neuromolecular level, those biological developments themselves will likely be structured at least in part by differential activation of specific gene modules. Moreover, the general modularity of brain function implies that these neurogenomic impacts of habitual reappraisal (e.g., in down-regulating threat circuits in the limbic system, up-regulating analytic or appraisal systems, and possibly

reducing the need for intense conscious controlled inhibitory processes) may well spill over into other domains of experience, thus altering the activity of any other response systems that make use of the same functional brain modules. Again, the specific empirical structure of the gene networks involved remains to be mapped. However, we now have available a growing analytic and technical infrastructure for discovering the genomic mechanisms of appraisal-based emotion regulation once empirical transcriptome data become available. These considerations underscore the importance of developing experimental animal models that can potentially recapitulate at least some basic features of the perception/appraisal and suppression/reappraisal systems. More broadly, to the extent that reappraisal produces a more fundamentally human and behaviorally adaptive CNS, it may well have salutary effects on peripheral gene expression and health that extend far beyond a neutral basal state of physiological homeostasis.

Much remains to be learned about the molecular mechanisms and biological implications of human emotion regulation. However, what we can say from the data already at hand is that, in a highly social species such as humans, issues of expressing and concealing emotional reactions cut deeply and fundamentally to the core of what it means to be a participant in the human race. In that sense, it is perhaps not so surprising that our conscious and unconscious efforts to manage the experience and expression of our emotions may have deep implications for our health. If the genome is a lens through which health can be understood, it is even more surely a molecular blueprint for helping humans navigate the complexities of our physical and social worlds (Fox Keller, 2012). In that regard, it comes as no surprise if the deepest regions of our internal biology, and the very function of our own individual human genomes, are inextricably bound up in our experience of the external social world around us, the emotional experiences it evokes in us, and our emotional expressions back to that world. Genomes exist fundamentally to help us succeed as humans, and to the extent that requires the effective regulation of our emotional experience, the human genome must carry some molecular blueprints that help us do that. And to the extent that we fail to adaptively manage the experience and expression of our emotions, the genome also appears to carry molecular blueprints that may help defend us against the near-term social–biological costs of that failure, but only by mortgaging the molecular underpinnings of our long-term vitality.

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References

Amit, I., Garber, M., Chevrier, N., Leite, A. P., Donner, Y., Eisenhaure, T., et al. (2009). Unbiased reconstruction of a mammalian transcriptional network mediating pathogen responses. *Science, 326*(5950), 257–263.

Antoni, M. H., Lutgendorf, S. K., Blomberg, B., Stagl, J., Carver, C. S., Lechner, S., et al. (2012). Transcriptional modulation of human leukocytes by cognitive-behavioral stress management in women undergoing treatment for breast cancer. *Biological Psychiatry, 71*(4), 366–372.

Appleton, A. A., & Kubzansky, L. D. (this volume, 2014). Emotion regulation and cardiovascular disease risk. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 596–612). New York: Guilford Press.

Bell, J. R., Jasnoski, M. L., Kagan, J., & King, D. S. (1990). Is allergic rhinitis more frequent in adults with extreme shyness?: A preliminary study. *Psychosomatic Medicine, 52*, 517–525.

Berkman, L. F., & Kawachi, I. (2000). *Social epidemiology*. New York: Oxford University Press.

Black, D. S., Cole, S. W., Irwin, M. R., Breen, E., St. Cyr, N. M., Nazarian, N., et al. (2013). Yogic meditation reverses NF-kappaB and IRF-related transcriptome dynamics in leuko-

cytes of family dementia caregivers in a randomized controlled trial. *Psychoneuroendocrinology, 38*(3), 348–355.

Booth, R. J. (2012). Emotional expression and disclosure. In S. Segerstrom (Ed.), *The Oxford handbook of psychoneuroimmunology* (pp. 105–125). New York: Oxford University Press.

Bowlby, J. (1983). *Attachment*. New York: Basic Books.

Buck, R. W. (1979). Individual differences in nonverbal sending accuracy and electrodermal responding: The externalizing–internalizing dimension. In R. Rosenthal (Ed.), *Skill in nonverbal communication* (pp. 140–170). Cambridge, MA: Oelgeschlager, Gunn & Hain.

Butler, E. A., Egloff, B., Wilhelm, F. H., Smith, N. C., Erickson, E. A., & Gross, J. J. (2003). The social consequences of expressive suppression. *Emotion, 3*(1), 48–67.

Cacioppo, J. T., & Hawkley, L. C. (2009). Perceived social isolation and cognition. *Trends in Cognitive Sciences, 13*(10), 447–454.

Capitanio, J. P., Mendoza, S. P., Lerche, N. W., & Mason, W. A. (1998). Social stress results in altered glucocorticoid regulation and shorter survival in simian acquired immune deficiency syndrome. *Proceedings of the National Academy of Sciences USA, 95*(8), 4714–4719.

Chang, M., Brown, H., Collado-Hidalgo, A., Arevalo, J., Galic, Z., Symensma, T., et al. (2005). Beta-adrenoreceptors reactivate Kaposi's sarcoma-related herpesvirus lytic replication via PKA-dependent control of viral RTA. *Journal of Virology, 72*(21), 13538–13547.

Chen, E., & Miller, G. E. (this volume, 2014). Early-life socioeconomic status, emotion regulation, and the biological mechanisms of disease across the lifespan. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 586–595). New York: Guilford Press.

Chen, E., Miller, G. E., Kobor, M. S., & Cole, S. W. (2011). Maternal warmth buffers the effects of low early-life socioeconomic status on pro-inflammatory signaling in adulthood. *Molecular Psychiatry, 16*, 729–737.

Chen, E., Miller, G. E., Walker, H. A., Arevalo, J. M., Sung, C. Y., & Cole, S. W. (2009). Genome-wide transcriptional profiling linked to social class in asthma. *Thorax, 64*(1), 38–43.

Cohen, L., Cole, S. W., Sood, A. K., Prinsloo, S., Kirschbaum, C., Arevalo, J. M., et al. (2012). Depressive symptoms and cortisol rhythmicity predict survival in patients with renal cell carcinoma: Role of inflammatory signaling. *PLoS ONE, 7*(8), e42324.

Cohen, S., Doyle, W. J., Skoner, D. P., Rabin, B. S., & Gwaltney, J. M. (1997). Social ties and susceptibility to the common cold. *Journal of the American Medical Association, 227*, 1940–1944.

Cohen, S., Doyle, W. J., Turner, R., Alper, C. M., & Skoner, D. P. (2003). Sociability and susceptibility to the common cold. *Psychological Science, 14*(5), 389–395.

Cole, S. W. (2008). Psychosocial influences on HIV-1 disease progression: Neural, endocrine, and virologic mechanisms. *Psychosomatic Medicine, 70*(5), 562–568.

Cole, S. W. (2009). Social regulation of human gene expression. *Current Directions in Psychological Science, 18*(3), 132–137.

Cole, S. W. (2010). Elevating the perspective on human stress genomics. *Psychoneuroendocrinology, 35*, 955–962.

Cole, S. W., Arevalo, J. M., Ruggerio, A. M., Heckman, J. J., & Suomi, S. (2012). Transcriptional modulation of the developing immune system by early life social adversity. *Proceedings of the National Academy of Sciences USA, 109*(50), 20578–20583.

Cole, S. W., Arevalo, J., Takahashi, R., Sloan, E. K., Lutgendorf, S., Sood, A. K., et al. (2010). Computational identification of gene–social environment interaction at the human IL6 locus. *Proceedings of the National Academy of Sciences USA, 107*(12), 5681–5686.

Cole, S. W., Hawkley, L. C., Arevalo, J. M., & Cacioppo, J. T. (2011). Transcript origin analysis identifies antigen-presenting cells as primary targets of socially regulated gene expression in leukocytes. *Proceedings of the National Academy of Sciences USA, 108*(7), 3080–3085.

Cole, S. W., Hawkley, L. C., Arevalo, J. M., Sung, C. Y., Rose, R. M., & Cacioppo, J. T. (2007). Social regulation of gene expression in human leukocytes. *Genome Biology, 8*(R189), 1–13.

Cole, S. W., Kemeny, M. E., Fahey, J. L., Zack, J. A., & Naliboff, B. D. (2003). Psychological risk factors for HIV pathogenesis: Mediation by the autonomic nervous system. *Biological Psychiatry, 54*, 1444–1456.

Cole, S. W., Kemeny, M. E., & Taylor, S. E. (1997). Social identity and physical health: Accelerated HIV progression in rejection-sensitive gay men. *Journal of Personality and Social Psychology, 72*, 320–336.

Cole, S. W., Kemeny, M. E., Taylor, S. E., & Visscher, B. R. (1996). Elevated physical health risk among gay men who conceal their homosexual identity. *Health Psychology, 15*, 243–251.

Cole, S. W., Kemeny, M. E., Taylor, S. E., Visscher, B. R., & Fahey, J. L. (1996). Accelerated course of human immunodeficiency virus infection in gay men who conceal their homosexual identity. *Psychosomatic Medicine, 58*, 219–231.

Cole, S. W., Naliboff, B. D., Kemeny, M. E., Griswold, M. P., Fahey, J. L., & Zack, J. A. (2001). Impaired response to HAART in HIV-infected individuals with high autonomic nervous system activity. *Proceedings of the National Academy of Sciences USA, 98*, 12695–12700.

Cole, S. W., & Sood, A. K. (2012). Molecular pathways: Beta-adrenergic signaling in cancer. *Clinical Cancer Research, 18*(5), 1201–1206.

Collado-Hidalgo, A., Sung, C., & Cole, S. (2006). Adrenergic inhibition of innate antiviral response: PKA blockade of Type I interferon gene transcription mediates catecholamine support for HIV-1 replication. *Brain, Behavior, and Immunity, 20*(6), 552–563.

Creswell, J. D., Irwin, M. R., Burklund, L. J., Lieberman, M. D., Arevalo, J. M., Ma, J., et al. (2012). Mindfulness-based stress reduction training reduces loneliness and pro-inflammatory gene expression in older adults: A small randomized controlled trial. *Brain, Behavior, and Immunity, 26*(7), 1095–1101.

Dan-Glauser, E. S., & Gross, J. J. (2011). The temporal dynamics of two response-focused forms of emotion regulation: Experiential, expressive, and autonomic consequences. *Psychophysiology, 48*(9), 1309–1322.

Darwin, C. (1872). *The expression of the emotions in man and animals*. London: John Murray.

Denollet, J., Gidron, Y., Vrints, C. J., & Conraads, V. M. (2010). Anger, suppressed anger, and risk of adverse events in patients with coronary artery disease. *American Journal of Cardiology, 105*(11), 1555–1560.

Denollet, J., Pedersen, S. S., Vrints, C. J., & Conraads, V. M. (2006). Usefulness of type D personality in predicting five-year cardiac events above and beyond concurrent symptoms of stress in patients with coronary heart disease. *American Journal of Cardiology, 97*(7), 970–973.

Denollet, J., Sys, S. U., Stroobant, N., Rombouts, H., Gillebert, T. C., & Brutsaert, D. L. (1996). Personality as an independent predictor of long-term mortality in patients with coronary heart disease. *Lancet, 347*, 417–421.

Djebali, S., Davis, C. A., Merkel, A., Dobin, A., Lassmann, T., Mortazavi, A., et al. (2012). Landscape of transcription in human cells. *Nature, 489*(7414), 101–108.

English, T., John, O. P., Srivastava, S., & Gross, J. J. (2012). Emotion regulation and peer-rated social functioning: A 4-year longitudinal study. *Journal of Research in Personality, 46*(6), 780–784.

Finch, C. E. (2007). *The biology of human longevity: Inflammation, nutrition, and aging in the evolution of lifespans*. Burlington, MA: Academic Press.

Finch, C. E. (2010). Evolution in health and medicine Sackler colloquium: Evolution of the human lifespan and diseases of aging: Roles of infection, inflammation, and nutrition. *Proceedings of the National Academy of Sciences USA, 1*, 1718–1724.

Fox Keller, E. (2012). Genes, genomes, and genomics. *Biological Theory, 6*(2), 132–140.

Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin, 132*(6), 823–865.

Glaser, R., Kiecolt-Glaser, J. K., Speicher, C. E., & Holliday, J. E. (1985). Stress, loneliness, and changes in herpesvirus latency. *Journal of Behavioral Medicine, 8*(3), 249–260.

Gross, J. (1989). Emotional expression in cancer onset and progression. *Social Science and Medicine, 28*, 1239–1248.

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*(1), 224–237.

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*(3), 281–291.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*(2), 348–362.

Gross, J. J., & Levenson, R. W. (1993). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64*, 970–986.

Gross, J. J., & Levenson, R. W. (1997). Hiding

feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology, 106*(1), 95–103.

Gyurak, A., & Etkin, A. (this volume, 2014). A neurobiological model of implicit and explicit emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 76–90). New York: Guilford Press.

Hanke, M. L., Powell, N. D., Stiner, L. M., Bailey, M. T., & Sheridan, J. F. (2012). Beta adrenergic blockade decreases the immunomodulatory effects of social disruption stress. *Brain, Behavior, and Immunity, 26*(7), 1150–1159.

Harris, C. R. (2001). Cardiovascular responses of embarrassment and effects of emotional suppression in a social setting. *Journal of Personality and Social Psychology, 81*(5), 886–897.

Henry, J. P. (1992). Biological basis of the stress response. *Integrative Physiological and Behavioral Science, 27*(1), 66–83.

Hofer, M. A. (1984). Relationships as regulators: A psychobiologic perspective on bereavement. *Psychosomatic Medicine, 46*(3), 183–197.

Hughes, C. F., Uhlmann, C., & Pennebaker, J. W. (1994). The body's response to processing emotional trauma: Linking verbal text with autonomic activity. *Journal of Personality, 62*(4), 565–585.

Ironson, G. H., O'Cleirigh, C., Weiss, A., Schneiderman, N., & Costa, P. T., Jr. (2008). Personality and HIV disease progression: Role of NEO-PI-R openness, extraversion, and profiles of engagement. *Psychosomatic Medicine, 70*(2), 245–253.

Irwin, M. R., & Cole, S. W. (2011). Reciprocal regulation of the neural and innate immune systems. *Nature Reviews Immunology, 11*(9), 625–632.

Jackson, D. C., Malmstadt, J. R., Larson, C. L., & Davidson, R. J. (2000). Suppression and enhancement of emotional responses to unpleasant pictures. *Psychophysiology, 37*(4), 515–522.

Jenkins, F. J., & Baum, A. (1995). Stress and reactivation of latent herpes simplex virus: A fusion of behavioral medicine and molecular biology. *Annals of Behavioral Medicine, 17*(2), 116–123.

Johnstone, T., & Walter, H. (this volume, 2014). The neural basis of emotion dysregulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 58–75). New York: Guilford Press.

Jones, H. E. (1935). The galvanic skin response as related to overt emotional expression. *American Journal of Psychology, 47*, 241–251.

Jones, H. E. (1950). The study of patterns of emotional expression. In M. L. Reymert (Ed.), *Feelings and emotions: The Mooseheart Symposium*. New York: McGraw-Hill.

Jones, H. E. (1960). The longitudinal method in the study of personality. In I. Iscoe & H. W. Stevenson (Eds.), *Personality development in children* (pp. 3–27). Austin: University of Texas Press.

Kagan, J. (1994). *Galen's prophecy: Temperament in human nature*. New York: Basic Books.

Kagan, J., Reznick, J. S., & Snidman, N. (1988). Biological bases of childhood shyness. *Science, 240*, 167–171.

Kagan, J., Snidman, N., Julia-Sellers, M., & Johnson, O. (1991). Temperament and allergic symptoms. *Psychosomatic Medicine, 53*, 332–340.

Karelina, K., Norman, G. J., Zhang, N., Morris, J. S., Peng, H., & DeVries, A. C. (2009). Social isolation alters neuroinflammatory response to stroke. *Proceedings of the National Academy of Sciences USA, 106*(14), 5895–5900.

Karssen, A. M., Her, S., Li, J. Z., Patel, P. D., Meng, F., Bunney, W. E., Jr., et al. (2007). Stress-induced changes in primate prefrontal profiles of gene expression. *Molecular Psychiatry, 12*(12), 1089–1102.

Kupfer, S. R., & Summers, W. C. (1990). Identification of a glucocorticoid-responsive element in Epstein–Barr virus. *Journal of Virology, 64*(5), 1984–1990.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer.

Leib, D. A., Nadeau, K. C., Rundle, S. A., & Schaffer, P. A. (1991). The promoter of the latency-associated transcripts of herpes simplex virus type 1 contains a functional cAMP-response element: Role of the latency-associated transcripts and cAMP in reactivation of viral latency. *Proceedings of the National Academy of Sciences USA, 88*(1), 48–52.

Lissek, S., Orme, K., McDowell, D. J., Johnson, L. L., Luckenbaugh, D. A., Baas, J. M., et al. (2007). Emotion regulation and potentiated startle across affective picture and threat-of-shock paradigms. *Biological Psychology, 76*(1–2), 124–133.

Lundberg, U., & Frankenhaeuser, M. (1980). Pituitary–adrenal and sympathetic–adrenal

correlates of distress and effort. *Journal of Psychosomatic Research, 24*(3–4), 125–130.

Lutgendorf, S. K., Degeest, K., Sung, C. Y., Arevalo, J. M., Penedo, F., Lucci, J., III, et al. (2009). Depression, social support, and beta-adrenergic transcription control in human ovarian cancer. *Brain, Behavior, and Immunity, 23*(2), 176–183.

Mauss, I. B., Shallcross, A. J., Troy, A. S., John, O. P., Ferrer, E., Wilhelm, F. H., et al. (2011). Don't hide your happiness!: Positive emotion dissociation, social connectedness, and psychological functioning. *Journal of Personality and Social Psychology, 100*(4), 738–748.

McDade, T. W. (2003). Life history theory and the immune system: Steps toward a human ecological immunology. *American Journal of Physical Anthropology, 37*(Suppl.), 100–125.

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60*, 501–524.

Miller, G. E., Chen, E., Fok, A. K., Walker, H., Lim, A., Nicholls, E. F., et al. (2009). Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proceedings of the National Academy of Sciences USA, 106*(34), 14716–14721.

Miller, G. E., Chen, E., Sze, J., Marin, T., Arevalo, J. M., Doll, R., et al. (2008). A functional genomic fingerprint of chronic stress in humans: Blunted glucocorticoid and increased NF-kappaB signaling. *Biolical Psychiatry, 64*(4), 266–272.

Mund, M., & Mitte, K. (2012). The costs of repression: A meta-analysis on the relation between repressive coping and somatic diseases. *Health Psychology, 31*(5), 640–649.

Negishi, H., Yanai, H., Nakajima, A., Koshiba, R., Atarashi, K., Matsuda, A., et al. (2012). Cross-interference of RLR and TLR signaling pathways modulates antibacterial T cell responses. *Nature Immunology, 13*(7), 659–666.

Notarius, C. I., & Levenson, R. W. (1979). Expressive tendencies and physiological response to stress. *Journal of Personality and Social Psychology, 37*(7), 1204–1210.

Ochsner, K. N., & Gross, J. J. (this volume, 2014). The neural bases of emotion and emotion regulation: A valuation perspective. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 23–42). New York: Guilford Press.

O'Connell, R. M., Saha, S. K., Vaidya, S. A., Bruhn, K. W., Miranda, G. A., Zarnegar, B., et al. (2004). Type I interferon production enhances susceptibility to Listeria monocytogenes infection. *Journal of Experimental Medicine, 200*(4), 437–445.

O'Donovan, A., Sun, B., Cole, S., Rempel, H., Lenoci, M., Pulliam, L., et al. (2011). Transcriptional control of monocyte gene expression in post-traumatic stress disorder. *Disease Markers, 30*(2–3), 123–132.

Padgett, D. A., Sheridan, J. F., Dorne, J., Berntson, G. G., Candelora, J., & Glaser, R. (1998). Social stress and the reactivation of latent herpes simplex virus type 1. *Proceedings of the National Academy of Sciences USA, 95*(12), 7231–7235.

Pennebaker, J. W. (1988). Confession, inhibition, and disease. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 22, pp. 211–242). Orlando, FL: Academic Press.

Pennebaker, J. W., Barger, S. D., & Tiebout, J. (1989). Disclosure of traumas and health among Holocaust survivors. *Psychosomatic Medicine, 51*(5), 577–589.

Pennebaker, J. W., & Chew, C. H. (1985). Behavioral inhibition and electrodermal activity during deception. *Journal of Personality and Social Psychology, 49*, 1427–1433.

Pennebaker, J. W., Hughes, C. F., & O'Heeron, R. C. (1987). The psychophysiology of confession: Linking inhibitory and psychosomatic processes. *Journal of Personality and Social Psychology, 52*(4), 781–793.

Prosch, S., Wendt, C. E. C., Reinke, P., Priemer, C., Pooert, M., Kruger, D. H., et al. (2000). A novel link between stress and human cytomegalovirus (HCMV) infection: Sympathetic hyperactivity stimulates HCMV activation. *Virology, 272*, 357–365.

Proudfit, G., Dunning J. P., Foti, D., & Weinberg, A. (this volume, 2014). Temporal dynamics of emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 43–57). New York: Guilford Press.

Rasmussen, A. F., Marsh, J. T., & Brill, N. Q. (1957). Increases susceptibility to hepres simplex in mice subjected to avoidance-learning stress or restraint. *Proceedings of the Society for Experimental Biology and Medicine, 96*, 183–189.

Richerson, P. J., Boyd, R., & Henrich, J. (2010).

Colloquium paper: Gene–culture coevolution in the age of genomics. *Proceedings of the National Academy of Sciences USA, 107*(Suppl. 2), 8985–8992.

Robinson, J. L., & Demaree, H. A. (2009). Experiencing and regulating sadness: Physiological and cognitive effects. *Brain and Cognition, 70*(1), 13–20.

Sapolsky, R. M. (1994). *Why zebras don't get ulcers: A guide to stress, stress-related diseases, and coping.* New York: Freeman.

Schuster, C., Chasserot-Golaz, S., Urier, G., Beck, G., & Sergeant, A. (1991). Evidence for a functional glucocorticoid responsive element in the Epstein–Barr virus genome. *Molecular Endocrinology, 5*(2), 267–272.

Shahangian, A., Chow, E. K., Tian, X., Kang, J. R., Ghaffari, A., Liu, S. Y., et al. (2009). Type I IFNs mediate development of postinfluenza bacterial pneumonia in mice. *Journal of Clinical Investigation, 119*(7), 1910–1920.

Siegel, R. E. (1968). *Galen's system of physiology and medicine.* New York: Karger.

Sloan, E. K., Capitanio, J. P., Tarara, R. P., Mendoza, S. P., Mason, W. A., & Cole, S. W. (2007). Social stress enhances sympathetic innervation of primate lymph nodes: Mechanisms and implications for viral pathogenesis. *Journal of Neuroscience, 27*(33), 8857–8865.

Sloan, E. K., Priceman, S. J., Cox, B. F., Yu, S., Pimentel, M. A., Tangkanangnukul, V., et al. (2010). The sympathetic nervous system induces a metastatic switch in primary breast cancer. *Cancer Research, 70*(18), 7042–7052.

Srivastava, S., Tamir, M., McGonigal, K. M., John, O. P., & Gross, J. J. (2009). The social costs of emotional suppression: A prospective study of the transition to college. *Journal of Personality and Social Psychology, 96*(4), 883–897.

Sterling, P. (2004). Principles of allostasis: Optimal design, predictive regulation, pathophysiology and rational therapeutics. In J. Schulkin (Ed.), *Allostasis, homeostasis, and the costs of physiological adaptation* (pp. 17–64). Cambridge, UK: Cambridge University Press.

Thaker, P. H., Han, L. Y., Kamat, A. A., Arevalo, J. M., Takahashi, R., Lu, C., et al. (2006). Chronic stress promotes tumor growth and angiogenesis in a mouse model of ovarian carcinoma. *Nature Medicine, 12*(8), 939–944.

Tian, X., Xu, F., Lung, W. Y., Meyerson, C., Ghaffari, A. A., Cheng, G., et al. (2012). Poly I:C enhances susceptibility to secondary pulmonary infections by Gram-positive bacteria. *PLoS ONE, 7*(9), e41879.

Tung, J., Barreiro, L. B., Johnson, Z. P., Hansen, K. D., Michopoulos, V., Toufexis, D., et al. (2012). Social environment is associated with gene regulatory variation in the rhesus macaque immune system. *Proceedings of the National Academy of Sciences USA, 109*(17), 6490–6495.

Wagner, D. D., & Heatherton, T. F. (this volume, 2014). Emotion and self-regulation failure. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 613–628). New York: Guilford Press.

Weaver, I. C., Meaney, M. J., & Szyf, M. (2006). Maternal care effects on the hippocampal transcriptome and anxiety-mediated behaviors in the offspring that are reversible in adulthood. *Proceedings of the National Academy of Sciences USA, 103*(9), 3480–3485.

Weinberger, D. A., Schwartz, G. E., & Davidson, R. E. (1979). Low anxious, high anxious, and repressive coping styles: Psychometric patterns and physiological responses to stress. *Journal of Abnormal Psychology, 88*, 369–380.

Wilson, E. O. (2012). *The social conquest of Earth.* New York: Liveright/Norton.

Wohleb, E. S., Hanke, M. L., Corona, A. W., Powell, N. D., Stiner, L. M., Bailey, M. T., et al. (2011). Beta-adrenergic receptor antagonism prevents anxiety-like behavior and microglial reactivity induced by repeated social defeat. *Journal of Neuroscience, 31*(17), 6277–6288.

Yang, E. V., Webster Marketon, J. I., Chen, M., Lo, K. W., Kim, S. J., & Glaser, R. (2010). Glucocorticoids activate Epstein–Barr virus lytic replication through the upregulation of immediate early BZLF1 gene expression. *Brain, Behavior, and Immunity, 24*(7), 1089–1096.

CHAPTER 34

Early-Life Socioeconomic Status, Emotion Regulation, and the Biological Mechanisms of Disease across the Lifespan

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Health disparities—that is, differences in disease outcomes by socioeconomic status (SES)—remain one of the most pressing public health issues in our society. For example, low-SES individuals—meaning those who are low in education, income, or occupational status—are 2.7 times more likely to have repeated hospitalizations during a 1-year period than high-SES individuals (National Center for Health Statistics, 2010), and 3.5 times more likely to suffer activity limitations due to disease than high-SES individuals (Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010). And by age 25, those from the lowest SES group are expected to live 6 fewer years compared to those in the highest SES group (Braveman et al., 2010).

This issue has become such a widespread concern that *Healthy People 2010*, the national health objectives from the U.S. Department of Health and Human Services (2000), lists eliminating health disparities as one of two overarching goals. In addition, the National Institutes of Health (NIH) ranked the issue of health disparities third among its top five priorities (Thomson, Mitchell, & Williams, 2006).

Explanations for why health disparities are so pervasive have been difficult to unearth, because commonly suggested factors, such as access to health care, have not sufficiently explained existing disparities (Adler, Boyce, Chesney, Folkman, & Syme, 1993). In addition, there is growing consensus that social, and not just biomedical, determinants of disease are important to identify (Dankwa-Mullan et al., 2010). In this chapter, we explore the role that emotion regulation may play in explaining health disparities that emerge early in life. We do this by first providing an overview of links between childhood SES and disease outcomes into adulthood. Second, we examine whether emotion regulation may serve as one explanation for these associations by discussing links between emotion regulation and physiological processes implicated in disease, as well as meditational evidence for emotion regulation strategies in relationships between low-SES and these physiological processes. Third, we discuss the question of moderation—that is, whether certain types of emotion regulation strategies could also serve as protective buffers for a subgroup of those who are low in SES. Throughout this chapter, our premise is that low early life SES fosters certain emotion regulation strategies that emerge during childhood and have implications for physiological processes during childhood and into adulthood; hence, we discuss findings that provide potential

explanations for links between childhood SES and both childhood and adult diseases.

Early Life Environments and Risk for Disease

We begin by discussing epidemiological evidence that low SES increases risk for disease. Low SES in childhood confers greater risk for disease, both throughout childhood and into adulthood. A number of reviews have documented that the effects of low SES start early, and that low SES during childhood is associated with a number of different adverse health outcomes, including greater asthma morbidity, obesity, and injury rates, and poorer self- and parent-reports of health (Chen, Matthews, & Boyce, 2002; Goodman, 1999; Starfield, Riley, Witt, & Robertson, 2002; Starfield, Robertson, & Wiley, 2002).

In addition, the effects of low SES persist into adulthood (Miller, Chen, & Parker, 2011). For example, two reviews of the literature reported that the vast majority of studies found an increased risk of all-cause mortality in individuals who grew up in low- versus high-SES households (Galobardes, Lynch, & Smith, 2004, 2008). Moreover, controlling for adult SES did not eliminate these associations, indicating that something specific to low SES in childhood confers risk for early mortality. Another review documented a heightened risk of cardiovascular disease morbidity associated with low SES in childhood (Galobardes, Shaw, Lawlor, Lynch, & Smith, 2006). Again, associations held up after researchers controlled for adult SES.

These studies are corroborated by quasi-experimental evidence, such as the viral challenge paradigm in humans (Cohen, Doyle, Turner, Alper, & Skoner, 2004), in which a sample of adults was quarantined and exposed to rhinoviruses that cause colds. Participants who came from low-SES households in childhood were significantly more likely to become infected with the rhinovirus and to develop cold symptoms compared to those who came from high childhood SES households. These associations held even after researchers controlled for adult SES, suggesting again that experiencing low SES specifically during the childhood years increases risk for adverse health outcomes later in life.

In summary, a large body of epidemiological evidence demonstrates that low SES during childhood is associated with a variety of poor health outcomes both during childhood and into adulthood. In the next section, we explore the idea that one psychological factor contributing to this association may be difficulties with emotion regulation.

The Role of Emotion Regulation as a Pathway Linking SES and Disease

In this section, we discuss the idea that emotion regulation may serve as one psychological pathway linking SES and disease outcomes. To make this argument, we (1) provide a brief overview of associations between SES and emotion regulation; (2) discuss what types of emotion regulation strategies are relevant to physiological outcomes; (3) discuss physiological markers relevant to disease; (4) provide an overview of previous research on links between emotion regulation and physiological outcomes; and (5) describe studies that have tested emotion regulation as a mediator of SES and physiology relationships.

SES and Emotion Regulation

Emotion regulation refers to strategies to increase, decrease, or maintain emotional responses (Gross, 2001). Gross's process model of emotion regulation states that there are various types of emotion regulation strategies, including *antecedent-focused emotion regulation*, that is, strategies employed before emotional responses become fully activated, and *response-focused emotion regulation*, that is, strategies employed after emotion response tendencies have been activated (Gross, 1998).

In the context of SES, extensive evidence documents that low-SES individuals are more prone to experience negative emotions, and in turn that these negative emotions are detrimental for health (for a review, see Gallo & Matthews, 2003; but note that the evidence of negative emotions actually serving as a mediator of the SES–health relationship is mixed; Matthews & Gallo, 2011). Nonetheless, in this context, effective

emotion regulation strategies should reduce experiences of negative emotions.

Implications of Emotion Regulation Strategies for Physiological Responses

Gross (1998) has documented that efforts that fall under antecedent-focused emotion regulation, such as reappraisal, have fewer physiological costs than response-focused emotion regulation strategies, such as suppression. Hence, in the next sections, we focus on the role that antecedent-focused emotion regulation plays in linking SES to physiological and health outcomes.

Antecedent-focused emotion regulation involves strategies such as reappraisal—that is, reevaluating a stressful situation in a way that seeks to reduce its emotional impact. It can also include strategies such as situation selection, situation modification, and attention deployment; together with reappraisal, all of these strategies occur temporally before emotional responses are generated and can alter behavioral, emotional, and physiological response tendencies. Reappraisal is the strategy that has been studied most frequently with respect to affective, cognitive, and social consequences (Gross, 2001), so in the section below we focus below largely on links between reappraisal and physiological processes implicated in disease.

Relevant Physiological Markers for Disease

Conceptualizing pathways to disease on the biological end entails consideration of both acute and longer-term physiological responses. Below we provide a brief overview of the types of systems and processes implicated in chronic diseases—with a focus on diseases linked to inflammation, such as cardiovascular disease and asthma, so that readers will be familiar with the outcomes we present later on in studies of emotion regulation and physiological processes. In this section, we focus on physiological systems that are capable of being altered by psychosocial factors (e.g., stress) and hence could be plausibly linked to variables such as emotion regulation.

Acutely the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system (SNS) become activated with many psychosocial stressors, releasing hormones such as cortisol, epinephrine, and norepinephrine (Cannon, 1932; Kemeny, 2003). These hormones bind to receptors located on a variety of bodily tissues, exerting effects on the heart, vasculature, and metabolic and immune systems. With respect to acute physiological responses, profiles indicative of lower disease risk include a reduced magnitude of reactivity of these systems and/or a quicker recovery time (quicker return to baseline levels) (Krantz & Manuck, 1984; Linden, Earle, Gerin, & Christenfeld, 1997; Linden, Gerin, & Davidson, 2003; McEwen, 1998; Schwartz et al., 2003). We note that much of the literature reviewed below does not directly measure SNS and HPA activity, but instead focuses on the responses of end organs such as the heart and blood vessels. However, because these organs are influenced by SNS and HPA activity, and are the source of eventual manifestations of cardiovascular disease (CVD), their responses to acute stressors are relevant here.

Over the long term, with excessive and prolonged exposure to the hormones mentioned earlier, the structure and function of tissues and organs are thought to be altered, giving rise to pathogenic processes that drive CVD, such as obesity, insulin resistance, systemic inflammation, high blood pressure, endothelial dysfunction, and platelet activation (Brotman, Golden, & Wittstein, 2007; Everson-Rose & Lewis, 2005; Rozanski, Blumenthal, Davidson, Saab, & Kubzansky, 2005). Hence, we also review links between emotion regulation strategies and these longer-term mechanisms.

Longer-term cumulative physiological risk has sometimes been encapsulated in concepts such as *allostatic load* (McEwen, 1998), which is defined as instances when individuals experience stressors repeatedly and have more frequent activation of physiological systems over time; or when physiological systems do not show adaptation of responses after repeated stressors; or when shutdown mechanisms are delayed or insufficient, leading to prolonged physiological responses over time. The strain on these allostatic systems over years may eventually cause a breakdown of these systems that ultimately leads to disease. Empirically, high levels of obesity, insulin resistance, systemic

inflammation, blood pressure, endothelial dysfunction, and platelet activation all predict CVD morbidity and mortality (Danesh et al., 2005; Guh et al., 2009; Lindmark, Diderholm, Wallentin, & Siegbahn, 2001; Ridker, Hennedens, Buring, & Rifai, 2000; Vasan et al., 2001; Yeboah et al., 2009). In addition, the accumulation of these characteristics, in constellations such as allostatic load or metabolic syndrome, predicts even more strongly an increased risk of CVD later in life (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997; Lakka et al., 2002; Morrison, Friedman, & Gray-McGuire, 2007; Ridker, Buring, Cook, & Rifai, 2003; National Cholesterol Education Program, 2002).

Emotion Regulation and Physiological and Disease Outcomes

We focus here on links specifically between the emotion regulation strategy of reappraisal and physiological responses, given the existing evidence with respect to this strategy. Physiologically, reappraisals reduce cardiovascular reactivity to acute stressors. For example, lower reappraisals of threat have been associated with reduced blood pressure reactivity during acute stressors in both children and adults (El Sheikh & Harger, 2001; Maier, Waldstein, & Synowski, 2003), and lower ambulatory blood pressure during daily life social interactions (Chen, Matthews, & Zhou, 2007). Similarly, individuals high in the ability to reappraise stressful situations show reduced vascular reactivity during acutely stressful tasks (Mauss, Cook, Chang, & Gross, 2007), and lower blood pressure and cortisol responses to an acute stressor (Salovey, Stroud, Woolery, & Epel, 2002). Experimental evidence shows that interventions aimed at changing appraisals in patient populations produce increases in benefit finding, as well as decreases in serum cortisol levels from pre- to postintervention (Cruess et al., 2000). Finally, consistent with the idea that underlying positive beliefs about others shape reappraisals and physiological responses, those who believe that the world is fair (high in just world beliefs) reappraise an acute stressor as less threatening and show less vascular reactivity to the stressor (Tomaka & Blascovich, 1994).

Emotion regulation also mitigates longer-term pathogenic processes implicated in CVD. For example, better emotion regulation abilities are linked to lower allostatic load, including higher high-density lipoprotein (HDL) cholesterol, lower triglycerides, and lower basal systolic blood pressure (Kinnunen, Kokkonen, Kaprio, & Pulkkinen, 2005). Similarly, reappraisals have been linked to longer-term markers of immune processes. For example, HIV-positive individuals who reported finding benefit after experiencing a major negative life event showed slower declines in cluster of differentiation 4 (CD4) T cell levels over 2–3 years (indicating a slower progression to the diagnosis of AIDS) (Bower, Kemeny, Taylor, & Fahey, 1998). Finally, functional indicators of poor emotion regulation, such as the experience of high levels of depression and anger, have been associated with higher levels of systemic inflammatory markers that are implicated in CVD, such as interleukin-6 (IL-6) and C-reactive protein (CRP) (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002; Miller, Maletic, & Raison, 2009).

Finally, emotion regulation strategies can also alter clinical disease outcomes. Reappraisals such as finding benefit after a life-threatening event predicts a lower likelihood of having a future heart attack (Affleck, Tennen, Croog, & Levine, 1987). Conversely, the experience of high levels of negative affect (arguably an indicator of inadequate emotion regulation) has robust associations with CVD-related outcomes (Brosschot, Gerin, & Thayer, 2006; Everson-Rose & Lewis, 2005; Krantz & McCeney, 2002; Kubzansky, Kawachi, Weiss, & Sparrow, 1998). In addition, how effectively one can manage emotions (emotional intelligence) is linked to better general indicators of physical health (better self-reported health and fewer illnesses; Goldman, Kraemer, & Salovey, 1996; Schutte, Malouff, Thorsteinsson, Bhullar, & Rooke, 2007). Finally, experimental data suggest that interventions to help individuals process negative emotions effectively, such as through written disclosure, produce fewer symptoms and health center visits in healthy adults, and improve disease indicators in patient populations (Smyth, Stone, Hurewitz, & Kaell, 1999; Smyth, 1998).

Emotion Regulation as a Mediator

The previously discussed literature links emotion regulation strategies to physiological, endocrine, and immune processes implicated in chronic diseases such as CVD. But is there any evidence that emotion regulation strategies actually mediate the relationship between SES and these biological processes? In a series of studies, our research group has shown evidence for such mediation by focusing on how children and adolescents reappraise stressful life situations.

In healthy adolescents, we have shown that lower SES is associated with greater cardiovascular reactivity to acute laboratory stressors. We further showed that low-SES adolescents were less likely to reappraise ambiguous life situations (e.g., shopping with an overly attentive sales clerk nearby) in benign ways. Finally, we documented that reappraisals of threat statistically mediated the relationship between low SES and heightened cardiovascular reactivity (Chen, Langer, Raphaelson, & Matthews, 2004).

In patient populations, we have documented similar patterns with disease-relevant markers. For example, in pediatric patients with asthma, we have demonstrated that low SES is associated with greater asthma inflammation (e.g., greater production of cytokines relevant to asthma, higher eosinophil counts). We further demonstrated that low SES is associated with being less likely to reappraise ambiguous life situations in benign ways in this patient population. Finally, we documented that reappraisals of threat statistically mediated the relationship between low SES and heightened asthma inflammation (Chen, Fisher, Bacharier, & Strunk, 2003; Chen et al., 2006).

We further documented that effects of low SES can be seen at the genomic level. Low SES children with asthma showed indications of increased activity of proinflammatory gene networks, and these associations between low SES and gene expression patterns were no longer significant once reappraisals of threat during ambiguous life situations were statistically controlled (Chen et al., 2009).

Taken together, this set of studies illustrates how low-SES individuals are, on average, less able to engage in reappraisals of life situations effectively. Furthermore, reappraisals of threat form one pathway explaining why low-SES children show heightened inflammatory and cardiovascular responses. In turn, these inflammatory and cardiovascular profiles are predictive of later disease.

The Role of Emotion Regulation as Buffer

In the previous section we discussed how emotion regulation strategies serve as one psychological mediator explaining links between low SES and detrimental profiles of physiological processes relevant to disease. In this section, we turn to the question of whether emotion regulation could also serve as a moderator of SES and health outcomes. Despite the robust associations between low SES and disease, there remains a subset of individuals that displays physiologically healthy profiles despite living under adversity. What can explain this group of individuals? That is, what factors might naturally protect individuals who grow up in low-SES environments from the physiological toll and accumulation of health problems typically exacted by these environments? Our research group has articulated a theory about the psychological characteristics that may be specifically beneficial to low-SES individuals (Chen & Miller, 2012). Emotion regulation plays a key role in this theory; hence we discuss its role as a buffer for low-SES individuals in this chapter. In this section, we first provide an outline of the shift-and-persist theory; then we describe the empirical evidence in support of this theory.

Shift and Persist

The theory begins with the notion that a lifetime of facing constraints with limited options leads those living in a low-SES context to place value on the ability to adjust in response to stressors through emotion regulation strategies such as reappraisals (*shifting)*. At the same time, in this context, successful adaptation entails enduring adversity with strength by finding meaning in difficult situations and maintaining optimism in the face of adversity (*persisting*). We proposed that this combination of approaches to dealing with adversity reduces physiological responses to stressful situations acutely, specifically among those who are low in SES, and over the long term mitigates the

progression of pathogenic processes leading to chronic diseases such as CVD (Chen & Miller, 2012).

Hence, one of the key components of this beneficial psychological profile centers around the ability to regulate one's emotions through reappraisal strategies. Because low-SES individuals on average have fewer opportunities to select or modify their life situations (alternative forms of emotion regulation; Gross, 1998, 2001), reappraisals represent a realistic approach to emotion regulation in this group. That is, given the myriad day-to-day, largely uncontrollable stressors experienced by many low-SES individuals, in many instances their best option may be to control the one thing they can—the self—rather than engage in what may turn out to be futile attempts to control their environment. By controlling the self, they engage in emotion regulation strategies in which they accept that a stressor has occurred and try to change the effect that stressor has on them. They do this by reappraising the meaning of an event, so that the implications for their lives become less negative. And they adjust their emotional reactions, so that the event evokes less distress in them. As they come to see events as having less serious implications and being less upsetting, the physiological responses they elicit are mitigated. Hence, we propose that low-SES individuals uphold as an ideal the goal of utilizing emotion regulation strategies related to reappraisals when dealing with stress. The ability to do this successfully comprises the "shift" part of our shift-and-persist model.

Shifting is hypothesized to be necessary but not sufficient for buffering low-SES individuals from stressors. In addition to shifting, we hypothesize that it will be important to persist—that is, to endure adversity with strength by finding meaning in life and maintaining optimism about the future. Finding meaning allows individuals to understand adversity and to grow from it. Optimism allows individuals to maintain hope about the future, and can be essentially thought of as reappraising the future (as opposed to shifting, which entails reappraisals of events that have already happened). Thus, reappraisals are an important component of both shifting and persisting.

We further postulate that there is something important about the combination—that is, it is not sufficient to be able to engage in emotion regulation to deal with current adversities; one also needs also to find broader meaning in life and be able to reappraise the future. Hence the label that we use, "shift and persist," is intended to connote the fact that it is this combination of characteristics that will be beneficial to low-SES individuals with respect to their health. In the next section, we discuss evidence supporting the notion that when low-SES individual engage in shift and persist, there are benefits to the physiological mechanisms that underlie disease.

Empirical Evidence for Shift and Persist

In two studies from our research group, we have documented the benefits of shift and persist specifically for low-SES individuals. In the first study, we assessed childhood SES in a national sample of adults. We measured cumulative physiological risk via allostatic load, based on 24 different measures across seven physiological systems. Shift and persist was measured using questionnaires probing reappraisal-related coping styles (shift) and future orientation (persist). We found a three-way interaction between childhood SES, shift, and persist in predicting allostatic load in this sample. Breaking down this three-way interaction revealed that there was a significant two-way interaction between shift and persist in those from low childhood SES backgrounds, but no two-way interaction of shift and persist among those from high childhood SES backgrounds. The two-way interaction revealed that those participants with low childhood SES backgrounds who were high on both shifting and persisting had the lowest allostatic load. In contrast, the combination of shift and persist did not predict allostatic load among those from high-SES childhood backgrounds (Chen, Miller, Lachman, Gruenewald, & Seeman, 2012).

In a second study, using a clinical sample, we investigated the effects of shift and persist among children diagnosed with asthma, using questionnaires that tapped both reappraisal styles of coping (for shifting) and optimism about the future (for persisting). Among those low in SES, the higher their shift-and-persist scores, the lower their asthma inflammation. Also, among low-SES children, higher shift-and-persist scores pro-

spectively predicted less functional impairment (fewer school absences, less rescue inhaler use) 6 months later, when we controlled for baseline levels. In fact, low-SES children who scored high on shift and persist had inflammatory and clinical profiles more similar to high-SES children with asthma than to low-SES children who scored low in shift and persist. Shift and persist was not related to inflammatory or clinical profiles in high-SES children with asthma (Chen et al., 2011).

In summary, we find that low-SES individuals who are able to engage in emotion regulation strategies involving reappraisals, in combination with being optimistic and future oriented, are the ones who show the most beneficial physiological profiles and the least clinical disease impairment. Shift and persist is only beneficial to those who are low in SES, not to those who are high in SES, suggesting that there are context-specific determinants of what types of strategies will be beneficial physiologically for whom. In particular, for low-SES individuals, engaging in reappraisals and focusing on the future when encountering current daily stressors that are largely uncontrollable may be beneficial. In contrast, for those who are high in SES, proactive attempts to eliminate or mitigate stressful situations may be a more beneficial approach given the greater resources these individuals tend to have.

In addition, it is important to note that the combination of shift and persist is critical to physiological profiles among low-SES individuals. That is, neither shifting nor persisting alone was predictive of physiological outcomes; rather, it is only when individuals combine shifting and persisting that one sees physiological benefits. This suggests that emotion regulation strategies of reappraisals on their own are not sufficient; rather, they need to be combined with a focus on the future that emphasizes optimism and meaning in order to derive physiological benefits among those who are low in SES.

Conclusions

In summary, health disparities are a pressing issue in our society, and researchers have been working to understand what factors account for such striking differences in health outcomes across the SES gradient. One possibility we suggest here is that on the psychological end, low-SES children may experience greater difficulties with emotion regulation. In particular, low-SES children may find themselves less able to reappraise stressful situations in positive ways. In turn, this leads them to be more likely to experience negative emotions and physiological costs, both acutely and cumulatively, that may contribute to risk for disease over the long term. We note, however, that much of this work is correlational, and cannot be used to draw conclusions about causality.

We also document that emotion regulation serves an important function in terms of buffering low-SES children from detrimental physiological profiles. That is, those low-SES children who are able to engage in shift and persist (utilizing effective emotion regulation strategies, such as reappraisals in combination with persisting with hopes and finding meaning with respect to one's future) exhibit physiological profiles that are more similar to profiles of high-SES children than to those of low-SES children who do not engage in shift and persist. These children also showed less clinical disease impairment compared to low-SES children who did not engage in shift and persist. These findings suggest that shift and persist serves as a natural protective factor in low- but not high-SES children.

Emotion regulation strategies are an important factor to consider when investigating individual-level psychological mechanisms underlying SES disparities in health. These strategies provide an interface between children and their broader social environments, and play an important role in shaping hormonal and inflammatory responses to stress. In turn, these acute responses appear to have longer-term implications for the pathogenic processes that underlie chronic diseases across the lifespan.

References

Adler, N. E., Boyce, W. T., Chesney, M. A., Folkman, S., & Syme, S. L. (1993). Socioeconomic inequalities in health: No easy solution. *Journal of the American Medical Association 269*, 3140–3145.

Affleck, G., Tennen, H., Croog, S., & Levine, S.

(1987). Causal attribution, perceived benefits, and morbidity after a heart attack: An 8-year study. *Journal of Consulting and Clinical Psychology, 55*, 29–35.

Bower, J. E., Kemeny, M. E., Taylor, S. E., & Fahey, J. L. (1998). Cognitive processing, discovery of meaning, CD4 decline, and AIDS-related mortality among bereaved HIV-seropositive men. *Journal of Consulting and Clinical Psychology, 66*, 979–986.

Braveman, P. A., Cubbin, C., Egerter, S., Williams, D. R., & Pamuk, E. (2010). Socioeconomic disparities in health in the United States: What the patterns tell us. *American Journal of Public Health, 100*, S186–S196.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research, 60*, 113–124.

Brotman, D. J., Golden, S. H., & Wittstein, I. S. (2007). The cardiovascular toll of stress. *Lancet, 370*, 1089–1100.

Cannon, W. B. (1932). *The wisdom of the body.* New York: Norton.

Chen, E., Fisher, E. B., Jr., Bacharier, L. B., & Strunk, R. C. (2003). Socioeconomic status, stress, and immune markers in adolescents with asthma. *Psychosomatic Medicine, 65*, 984–992.

Chen, E., Hanson, M. D., Paterson, L. Q., Griffin, M. J., Walker, H. A., & Miller, G. E. (2006). Socioeconomic status and inflammatory processes in childhood asthma: The role of psychological stress. *Journal of Allergy and Clinical Immunology, 117*, 1014–1020.

Chen, E., Langer, D. A., Raphaelson, Y. E., & Matthews, K. A. (2004). Socioeconomic status and health in adolescents: The role of stress interpretations. *Child Development, 75*, 1039–1052.

Chen, E., Matthews, K. A., & Boyce, W. T. (2002). Socioeconomic differences in children's health: How and why do these relationships change with age? *Psychological Bulletin, 128*, 295–329.

Chen, E., Matthews, K. A., & Zhou, F. (2007). Interpretations of ambiguous social situations and cardiovascular responses in adolescents. *Annals of Behavioral Medicine, 34*, 26–36.

Chen, E., & Miller, G. E. (2012). "Shift-and-persist" strategies: Why being low in socioeconomic status isn't always bad for health. *Perspectives on Psychological Science, 7*, 135–158.

Chen, E., Miller, G. E., Lachman, M. E., Gruenewald, T. L., & Seeman, T. E. (2012). Protective factors for adults from low childhood socioeconomic circumstances: The benefits of shift-and-persist for allostatic load. *Psychosomatic Medicine, 74*, 178–186.

Chen, E., Miller, G. E., Walker, H. A., Arevalo, J. M., Sung, C. Y., & Cole, S. W. (2009). Genome-wide transcriptional profiling linked to social class in asthma. *Thorax, 64*, 38–43.

Chen, E., Strunk, R. C., Trethewey, A., Schreier, H. M., Maharaj, N., & Miller, G. E. (2011). Resilience in low-socioeconomic-status children with asthma: Adaptations to stress. *Journal of Allergy and Clinical Immunology, 128*, 970–976.

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2004). Childhood socioeconomic status and host resistance to infectious illness in adulthood. *Psychosomatic Medicine, 66*, 553–558.

Cruess, D. G., Antoni, M. H., McGregor, B. A., Kilbourn, K. M., Boyers, A. E., Alferi, S. M., et al. (2000). Cognitive-behavioral stress management reduces serum cortisol by enhancing benefit finding among women being treated for early stage breast cancer. *Psychosomatic Medicine, 62*, 304–308.

Danesh, J., Lewington, S., Thompson, S. G., Lowe, G. D., Collins, R., Kostis, J. B., et al. (2005). Plasma fibrinogen level and the risk of major cardiovascular diseases and nonvascular mortality: An individual participant meta-analysis. *Journal of the American Medical Association, 294*, 1799–1809.

Dankwa-Mullan, I., Rhee, K. B., Williams, K., Sanchez, I., Sy, F. S., Stinson, N., et al. (2010). The science of eliminating health disparities: Summary and analysis of the NIH Summit recommendations. *American Journal of Public Health, 100*, S12–S18.

El Sheikh, M., & Harger, J. (2001). Appraisals of marital conflict and children's adjustment, health, and physiological reactivity. *Developmental Psychology, 37*, 875–885.

Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health, 26*, 469–500.

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin, 129*, 10–51.

Galobardes, B., Lynch, J. W., & Smith, G. D.

(2004). Childhood socioeconomic circumstances and cause-specific mortality in adulthood: Systematic review and interpretation. *Epidemiologic Reviews, 26*, 7–21.

Galobardes, B., Lynch, J. W., & Smith, G. D. (2008). Is the association between childhood socioeconomic circumstances and cause-specific mortality established?: Update of a systematic review. *Journal of Epidemiology and Community Health, 62*, 387–390.

Galobardes, B., Shaw, M., Lawlor, D. A., Lynch, J. W., & Smith, G. D. (2006). Indicators of socioeconomic position (Part 2). *Journal of Epidemiology and Community Health, 60*, 95–101.

Goldman, S. L., Kraemer, D. T., & Salovey, P. (1996). Beliefs about mood moderate the relationship of stress to illness and symptom reporting. *Journal of Psychosomatic Research, 41*, 115–128.

Goodman, E. (1999). The role of socioeconomic status gradients in explaining differences in US adolescents' health. *American Journal of Public Health, 89*, 1522–1528.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*, 271–299.

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224–237.

Gross, J. J. (2001). Emotion regulation in adulthood: Timing is everything. *Current Directions in Psychological Science, 10*, 214–219.

Guh, D. P., Zhang, W., Bansback, N., Amarsi, Z., Birmingham, C. L., & Anis, A. H. (2009). The incidence of co-morbidities related to obesity and overweight: A systematic review and meta-analysis. *BMC Public Health, 9*, 88.

Kemeny, M. E. (2003). The psychobiology of stress. *Current Directions in Psychological Science, 12*, 124–129.

Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Emotions, morbidity, and mortality: New perspectives from psychoneuroimmunology. *Annual Review of Psychology, 53*, 83–107.

Kinnunen, M. L., Kokkonen, M., Kaprio, J., & Pulkkinen, L. (2005). The associations of emotion regulation and dysregulation with the metabolic syndrome factor. *Journal of Psychosomatic Research, 58*, 513–521.

Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodological critique. *Psychological Bulletin, 96*, 435–464.

Krantz, D. S., & McCeney, M. K. (2002). Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. *Annual Review of Psychology, 53*, 341–369.

Kubzansky, L. D., Kawachi, I., Weiss, S. T., & Sparrow, D. (1998). Anxiety and coronary heart disease: A synthesis of epidemiological, psychological, and experimental evidence. *Annals of Behavioral Medicine, 20*, 47–58.

Lakka, H. M., Laaksonen, D. E., Lakka, T. A., Niskanen, L. K., Kumpusalo, E., Tuomilehto, J., et al. (2002). The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. *Journal of the American Medical Association, 288*, 2709–2716.

Linden, W., Earle, T. L., Gerin, W., & Christenfeld, N. (1997). Physiological stress reactivity and recovery: Conceptual siblings separated at birth? *Journal of Psychosomatic Research, 42*, 117–135.

Linden, W., Gerin, W., & Davidson, K. (2003). Cardiovascular reactivity: Status quo and a research agenda for the new millennium. *Psychosomatic Medicine, 65*, 5–8.

Lindmark, E., Diderholm, E., Wallentin, L., & Siegbahn, A. (2001). Relationship between interleukin 6 and mortality in patients with unstable coronary artery disease: Effects of an early invasive or noninvasive strategy. *Journal of the American Medical Association, 286*, 2107–2113.

Maier, K. J., Waldstein, S. R., & Synowski, S. J. (2003). Relation of cognitive appraisal to cardiovascular reactivity, affect, and task engagement. *Annals of Behavioral Medicine, 26*, 32–41.

Matthews, K. A., & Gallo, L. C. (2011). Psychological perspectives on pathways linking socioeconomic status and physical health. *Annual Review of Psychology, 62*, 501–530.

Mauss, I. B., Cook, C. L., Cheng, J. Y., & Gross, J. J. (2007). Individual differences in cognitive reappraisal: Experiential and physiological responses to an anger provocation. *International Journal of Psychophysiology, 66*, 116–124.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*, 171–179.

Miller, A. H., Maletic, V., & Raison, C. L. (2009). Inflammation and its discontents: The

role of cytokines in the pathophysiology of major depression. *Biological Psychiatry, 65*, 732–741.

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin, 137*, 959–997.

Morrison, J. A., Friedman, L. A., & Gray-McGuire, C. (2007). Metabolic syndrome in childhood predicts adult cardiovascular disease 25 years later: The Princeton Lipid Research Clinics Follow-Up Study. *Pediatrics, 120*, 340–345.

National Center for Health Statistics. (2010). *Health, United States, 2009*. Hyattsville, MD: Author.

National Cholesterol Education Program. (2002). *Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III)*. Bethesda, MD: National Heart, Lung, and Blood Institute.

Ridker, P. M., Buring, J. E., Cook, N. R., & Rifai, N. (2003). C-reactive protein, the metabolic syndrome, and risk of incident cardiovascular events. *Circulation, 107*, 391–397.

Ridker, P. M., Hennekens, C. H., Buring, J. E., & Rifai, N. (2000). C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. *New England Journal of Medicine, 342*, 836–843.

Rozanski, A., Blumenthal, J. A., Davidson, K. W., Saab, P. G., & Kubzansky, L. (2005). The epidemiology, pathophysiology, and management of psychosocial risk factors in cardiac practice: The emerging field of behavioral cardiology. *Journal of the American College of Cardiology, 45*, 637–651.

Salovey, P., Stroud, L. R., Woolery, A., & Epel, E. S. (2002). Perceived emotional intelligence, stress reactivity, and symptom reports: Further explorations using the Trait Meta-Mood Scale. *Psychology and Health, 17*, 611–627.

Schutte, N. S., Malouff, J. M., Thorsteinsson, E. B., Bhullar, N., & Rooke, S. E. (2007). A meta-analytic investigation of the relationship between emotional intelligence and health. *Personality and Individual Differences, 42*, 921–933.

Schwartz, A. R., Gerin, W., Davidson, K. W., Pickering, T. G., Brosschot, J. F., Thayer, J. F., et al. (2003). Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosomatic Medicine, 65*, 22–35.

Seeman, T. E., Singer, B. H., Rowe, J. W., Horwitz, R. I., & McEwen, B. S. (1997). Price of adaptation—allostatic load and its health consequences: MacArthur studies of successful aging. *Archives of Internal Medicine, 157*, 2259–2268.

Smyth, J. M. (1998). Written emotional expression: Effect sizes, outcome types, and moderating variables. *Journal of Consulting and Clinical Psychology, 66*, 174–184.

Smyth, J. M., Stone, A. A., Hurewitz, A., & Kaell, A. (1999). Effects of writing about stressful experiences on symptom reduction in patients with asthma or rheumatoid arthritis: A randomized trial. *Journal of the American Medical Association, 281*, 1304–1309.

Starfield, B., Riley, A. W., Witt, W. P., & Robertson, J. (2002). Social class gradients in health during adolescence. *Journal of Epidemiology and Community Health, 56*, 354–361.

Starfield, B., Robertson, J., & Riley, A. W. (2002). Social class gradients and health in childhood. *Ambulatory Pediatrics, 2*, 238–246.

Thomson, G. E., Mitchell, F., & Williams, M. (2006). *Examining the Health Disparities Research Plan of the National Institutes of Health: Unfinished business*. Washington, DC: National Academies Press.

Tomaka, J., & Blascovich, J. (1994). Effects of justice beliefs on cognitive appraisal of and subjective, physiological, and behavioral responses to potential stress. *Journal of Personality and Social Psychology, 67*, 732–740.

U.S. Department of Health and Human Services. (2000). *Healthy People, 2010: Understanding and improving health*. Washington, DC: U.S. Government Printing Office.

Vasan, R. S., Larson, M. G., Leip, E. P., Evans, J. C., O'Donnell, C. J., Kannel, W. B., et al. (2001). Impact of high-normal blood pressure on the risk of cardiovascular disease. *New England Journal of Medicine, 345*, 1291–1297.

Yeboah, J., Folsom, A. R., Burke, G. L., Johnson, C., Polak, J. F., Post, W., et al. (2009). Predictive value of brachial flow-mediated dilation for incident cardiovascular events in a population-based study: The multi-ethnic study of atherosclerosis. *Circulation, 120*, 502–509.

CHAPTER 35

Emotion Regulation and Cardiovascular Disease Risk

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My tongue will tell the anger of my heart,
Or else my heart concealing it will break.
—WILLIAM SHAKESPEARE,
The Taming of the Shrew
(Act 4, Scene 3)

Cardiovascular disease (CVD) is a leading cause of morbidity and mortality worldwide (Mendis, Puska, & Norrving, 2011), and it is increasingly clear that disease processes initiate in childhood (Berenson & Srnivasan, 2005; Lloyd-Jones et al., 2009; National Heart, Lung, and Blood Institute, 2007). *Poor cardiovascular health* is defined as having a combination of unhealthy levels of lipids, blood pressure, and glucose, being overweight or obese, smoking, being sedentary, and having an unhealthy diet. This profile is highly prevalent among middle-aged adults in the United States (Folsom et al., 2011; Lloyd-Jones et al., 2010) and once risk factors are elevated, they are difficult to ameliorate (Lloyd-Jones et al., 2010). With the growing awareness that major CVD risk factors such as atherosclerosis and hypertension are often identifiable many years before the disease is fully manifest, the American Heart Association recently revised its national goals for cardiovascular health promotion to emphasize the identification and prevention of early life risk factors for CVD (Lloyd-Jones et al., 2010). As such, considering CVD from a developmental or life course perspective may provide new insights into disease etiology and suggest novel avenues for prevention and intervention efforts.

The notion that emotions are inextricably linked to heart health has been part of popular discourse for centuries. Terms such as "broken heart" and "heartsick" invoke the well-understood sentiment that while emotions may emanate from within the psyche, emotions move the heart to behave and respond in predictable ways. Moreover, the idea that the control or *regulation* of emotions also matter for heart health has popularly endured as well, as evidenced in the opening quotation. However, while emotions and cardiovascular health have long been recognized as intertwined, the exact nature of the relationship is not well understood. Much epidemiological and psychological work describes cardiovascular risk in association with negative emotions (for a review, see Suls & Bunde, 2005), and a growing body of work also describes the potentially protective effects of positive emotions on cardiovascular health (for

a review, see Boehm & Kubzansky, 2012). Despite this accumulation of evidence, key questions remain, including whether or not emotions truly cause CVD, or whether specific emotions are a product of the illness. Additional questions revolve around understanding the different roles of positive and negative emotion in determining CVD risk. We suggest that examining CVD risk in relation to emotion regulation will help to address these issues. Thus, in this review we consider what the extant literature tells us about a relationship between emotion regulation and CVD risk, and identify the potentially harmful and health-promoting impact of various emotion regulation strategies in this context.

A large literature indicates that both positive and negative emotions are relevant in terms of maintaining cardiovascular health or developing CVD (Boehm & Kubzansky, 2012; Suls & Bunde, 2005). Moreover, research has increasingly indicated that positive emotional functioning entails more than the absence of emotional distress, and that physical and mental health may depend in part on our ability to meet environmental demands (Kubzansky, Park, Peterson, Vokonas, & Sparrow, 2011). Drawing on these findings, investigators have begun to speculate that beyond the effects of any specific emotion, it is the regulation of emotion that is critical. Emotion regulation is a higher order feature of emotional functioning that involves the monitoring and management of emotional experience and response (Gross, this volume). Emotion regulation can be conscious or unconscious and involve both up- and down-regulation of positive and negative emotions. Emotion regulation is learned through socialization and experience over time, with childhood being an important period of development as temperament, biology, and social factors interact to build regulatory skills and strategies that are then used across the life course (Calkins & Hill, 2007; John & Gross, 2004; Rothbart, Sheese, & Posner, and Thompson, this volume). The research findings linking emotions and CVD risk, the importance of early life experience in learning to regulate emotion, and the recent evidence indicating that deterioration of cardiovascular health begins in childhood (Berenson & Srnivasan, 2005), have raised the question of whether emotion regulation may play an important role in determining CVD risk over the life course.

In this chapter, we first consider the laboratory and population-based evidence suggesting that emotion regulation may contribute to CVD risk. The literature linking emotion regulation to CVD has considered cardiovascular conditions (e.g., cardiovascular disease, hypertension) and the biological markers that are considered indicative of risk of developing CVD in individuals who are too young to have developed actual disease. These include indicators of cardiovascular function and risk that are measured in response to acute stress, generally obtained from laboratory-based studies (e.g., blood pressure reactivity), predisease conditions and markers of risk (e.g., hypertension, C-reactive protein, metabolic syndrome), and actual measures of disease (e.g., myocardial infarction, death). The term *cardiovascular disease* refers to a group of disorders of the heart and blood vessels, and encompasses both coronary heart disease and cerebrovascular disease (Mendis et al., 2011), the two cardiovascular disease outcomes most commonly considered in relation to emotion. We review work in these areas, while limiting our discussion to exemplar studies that focus on objectively measured outcomes. Doing so mitigates a common concern about these associations that self-report bias inflates or falsely suggests that emotional factors are causally related to health (i.e., more distressed individuals report more symptoms). Also, we primarily consider prospective studies that examine longitudinal data in which emotion regulation is assessed initially among individuals who are disease-free, prior to development of CVD. These study designs help to mitigate another common concern about these associations: that health conditions actually drive emotions rather than the reverse. We also discuss and apply a developmental perspective to emotion regulation and CVD risk associations, and consider the evidence linking childhood emotion regulation to CVD risk in adulthood. Finally, we close with recommendations for future work. Due to space constraints we do not consider studies of emotion regulation and health risk behaviors (e.g., smoking) in detail, although we note that such behaviors are considered

potential pathways through which emotion regulation may influence CVD, and recommend these as areas for future research. Also, while we focus on studies of emotion regulation in relation to CVD etiology (i.e., development of disease), we recognize that emotion regulation is also likely related to CVD progression and survival. Finally, it is important to note that while the effects of emotion regulation are likely involved in the pathophysiology of many diseases, the evidence to date is strongest for CVD and, as such, is reviewed specifically in this chapter.

Evidence Linking Emotion Regulation to Cardiovascular Function and Disease

Over the past several decades, the study of emotion regulation and cardiovascular function and disease has been taken up by related but traditionally distinct disciplines of psychology and epidemiology. Psychological laboratory-based and epidemiological population-based studies have documented associations between emotion regulation and cardiovascular function and risk. However, as each discipline has different goals and generally relies on different methodologies, the evidence linking emotion regulation to CVD has developed largely in two different literatures, with limited crossover. Laboratory-based studies of emotion regulation and cardiovascular function tend to have small sample sizes and focus on identifying relevant biological alterations occurring in association with experimentally manipulated use of regulatory strategies. Epidemiological studies tend to be observational and employ large population-based samples, and focus more on the diagnostic and health risk implications of dysregulated emotion. Moreover, epidemiological work includes studies that explicitly measure emotion regulation and test associations with cardiovascular risk (i.e., direct evidence), but these are limited in number. However, more numerous are studies of cardiovascular risk that do not measure emotion regulation directly but assess aspects of emotional functioning (e.g., negative emotions) that may provide indication of poor emotion regulation. We include these studies, because they provide relevant but indirect evidence of the relationships of interest. In this section, we bring together important work from each area, integrating evidence across disciplines in order to expand the evidence base and move the science forward.

It is important to note that whether the use of an emotion regulation strategy is adaptive or maladaptive is context-dependent. However, based on the empirical findings to date, we argue that the predominant use of a particular set of strategies (e.g., suppression, inhibition) may carry more cardiovascular health risks than other strategies (e.g., reappraisal, disclosure), even though on occasion the use of such strategies may well be adaptive. Thus, we acknowledge that there may be contexts in which strategies such as reappraisal may not always be beneficial, and strategies such as suppression may be useful. The larger point is that effective regulation (regardless of what regulatory strategy is used in any given situation) versus emotion dysregulation may have differential effects on cardiovascular disease risk.

Laboratory-Based Research

Laboratory-based work has found emotion regulation strategies to be associated with identifiable patterns in cardiovascular function and response to regulatory demands that have implications for disease risk (see also Chen & Miller, this volume). The general hypothesis guiding work on emotion regulation in this context is that failing to manage emotions effectively requires a certain degree of psychological and physiological exertion, and is essentially a form of stress exposure. Thus, such exertion is thought to exact a physiological cost on the body, which leads to "wear and tear" and thereby increases vulnerability to disease over time (Consedine, Magai, & Bonanno, 2002; Pennebaker & Beall, 1986).

Population-based research tends to focus on clinically relevant risk markers (typically measured with resting levels) and incidence of disease. In contrast, laboratory-based work often focuses on indicators of cardiovascular function and risk that are measured in response to acute stress, such as heart rate and blood pressure reactivity, pulse transmission time, finger pulse amplitude, and cardiac interbeat interval. Stressful or demanding situations activate the

hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system (SNS), resulting in a cascade of related hormones that in turn influence a range of end organ responses, including heart rate and blood pressure. High reactivity is typically inferred by higher heart rate and blood pressure levels, greater increases in sympathetic activation during acute stress, and slower return to baseline levels of these parameters during a recovery period, after the acute stress experience has ended. Reactivity has also been measured by examining cortisol response to stress (a marker of HPA activation) (Chida & Hamer, 2008). Heart rate variability is another parameter this work has considered; at rest, lower levels of heart rate variability have been linked with increased risk of developing CVD (Thayer & Lane, 2007), while reduced heart rate variability in response to stress or slower return to resting levels have been linked with autonomic dysregulation (Cohen et al., 2000). Other experimental work manipulating emotion regulation has also considered effects on immune-related markers or alterations in relevant health conditions.

Much work considering emotion suppression and inhibition in these settings has found some support for the idea that suppression may alter acute cardiac response in ways that could impair cardiovascular health. For example, in two key studies, participants were instructed to suppress emotion expression when watching films designed to elicit diverse emotions (Gross & Levenson, 1993, 1997). Compared to controls, those who actively suppressed both positive (amusement) and negative emotion (disgust, sadness) experienced significantly increased activation of the autonomic system, as measured by a composite variable of pulse transit time to the finger, finger pulse amplitude, pulse transit time to the ear, and figure temperature. Similar patterns of association have been observed across a range of physiological and health indicators, with more reactivity or risk-related outcomes associated with strategies of suppression and inhibition (Consedine et al., 2002). One recent study found divergent associations of emotion suppression and reappraisal with heart rate variability in response to an emotion regulation task (Denson, Grisham, & Moulds, 2011). Among 131 women instructed to suppress, reappraise, or remain neutral when viewing an anger-eliciting film, those in the suppression condition exhibited significantly reduced heart rate variability compared with baseline, whereas those in the reappraisal condition showed increased heart rate variability. These studies suggest that actively inhibiting or suppressing emotional expression and experience is associated with deleterious autonomic functioning, which over time may take a toll on the cardiovascular system.

Other laboratory-based work has considered whether effects of emotion regulation strategies over a longer period of time may be associated with beneficial effects on a variety of other markers of cardiovascular health. Many studies have found regulatory strategies such as emotional disclosure, or the ability to discuss or disclose emotions verbally or in writing, to have positive effects on a range of health outcomes, although fewer studies have directly assessed outcomes related to cardiovascular health. In an early experimental study of trauma disclosure, Pennebaker and Beall (1986) instructed disease-free subjects to write in journals over 4 consecutive days about either a traumatic experience or a neutral-control event. Compared to controls, those who wrote about a trauma and related emotions made significantly fewer health center visits in the 6 months following the disclosure experiment. Such benefits of emotional disclosure have been replicated across a variety of samples and with cardiovascular-related health outcomes. For example, in a randomized controlled trial of expressive writing among 179 individuals who recently experienced a first-time myocardial infarction, those randomized to the expressive writing/emotional disclosure condition had significantly lower blood pressure, reported fewer cardiac symptoms, had fewer medical appointments, and used less medication than controls 5 months postintervention (Willmott, Harris, Gellaitry, Cooper, & Horne, 2011). A recent meta-analysis of 146 experimental disclosure studies among 10,994 participants found a positive and significant effect (small to moderate in size) of disclosure specifically on cardiovascular indicators related to CVD, such as lipids and inflammation (Frattaroli, 2006). Among the physiological parameters studied, the stron-

gest beneficial effect of emotional disclosure was evident for specific immune function parameters. For example, experimental disclosure was associated with lower levels of proinflammatory cytokines and C-reactive protein, which in turn predict reduced risk of CVD (Danesh et al., 2004; Pearson et al., 2003).

While these studies generally suggest potential divergent effects of inhibitive and expressive forms of regulatory strategies on cardiovascular function, other studies do not find such differences, and suggest that use of either type of strategy requires physiological exertion. For example, in a study of 190 female college students, women were instructed to suppress emotion, reappraise emotion, or react naturally (control condition) when discussing an upsetting film with another participant. Participants in both the suppression and reappraisal conditions demonstrated larger increases in heart rate variability compared to controls (Butler, Wilhelm, & Gross, 2006). Some studies linking emotion regulation with altered cortisol responses in response to stress in a laboratory setting have described similar results whereby suppression and reappraisal are each related to higher cortisol response (Lam, Dickerson, Zoccola, & Zaldivar, 2009). However, in other studies reappraisal is associated with less cortisol reactivity (Chen & Miller, this volume; Salovey, Stroud, Woolery, & Epel, 2002). Given that laboratory-based work explicitly considering potential divergent effects of these strategies on a unified and comparable set of cardiovascular-relevant outcomes remains limited, additional work is needed to ascertain whether and how these acute effects might be informative in regard to long-term risk. In addition, prospective work would be useful to determine whether such patterns of exertion affect health over time.

Population-Based Work

Direct Evidence

Though capacity to regulate emotions may influence cardiovascular health, few have explicitly examined this question in population-based research. While highly promising, evidence is limited to a handful of studies, because emotion regulation is not typically assessed in large-scale studies of the determinants of CVD. Such epidemiological studies generally consider CVD risk across a set of related outcomes, including angina (usually ascertained by self-reported symptoms), myocardial infarction (usually ascertained by hospital records and diagnostic procedures), and coronary heart disease death (ascertained by death certificates). Studies of CVD may also consider hypertension and stroke as relevant outcomes, although we know of no epidemiological studies of directly assessed emotion regulation that have considered these conditions. *Incident disease* refers to rate of new cases developing disease rather than the number of cases in a population at any given time.

Existing results are highly congruent with those from laboratory and experimental work, and emerging evidence suggests that emotion regulation strategies that frequently involve suppression or inhibition may increase CVD risk, whereas use of other regulatory strategies may reduce CVD risk. For example, one prospective study of 1,122 older male participants considered the relation between self-regulation and the development of CVD. Self-regulation was assessed when men were disease-free, using items from the Minnesota Multiphasic Personality Inventory assessing one's ability to manage impulses, feelings, and behaviors; emotion regulation was identified as a central feature of this measure of self-regulation. Exemplary items used to construct the Self-Regulation scale included "I control my emotion"; "I am not easily angered"; and "I am usually calm and not easily upset." Compared with men who had the lowest levels of self-regulation, those with the highest levels had 62% reduced risk of experiencing a nonfatal myocardial infarction or coronary heart disease (CHD) death over 13 years of follow-up (Kubzansky et al., 2011). Moreover, the association appeared to be additive, because each standard deviation increase in self-regulation was associated with a 20% reduced risk of incident angina, nonfatal myocardial infarction, and CHD death over the follow-up period. Findings were maintained after adjusting for known coronary risk factors, as well as main effects of positive and negative affect.

Another study identified *control* over anger as one mechanism linking anger to

cardiovascular risk (for a review, see Dedert, Calhoun, Watkins, Sherwood, & Beckham, 2010). In a study of Finnish adults (n = 7,933), several dimensions of anger expression and control were assessed in relation to incident CVD events (myocardial infarction, stroke) over 10–15 years of follow-up (Haukkala, Konttinen, Laatikainen, Kawachi, & Uutela, 2010). Anger control was characterized by items related to the extent to which anger is regulated (e.g., "I control my temper"), and anger expression was characterized by how people generally react when feeling angry (e.g., "I express my anger"). While experiences of anger per se were not associated with CVD, participants reporting the lowest levels of anger control had 35% significantly higher risk of experiencing a fatal or nonfatal cardiovascular event in the subsequent 10–15 years compared to those with the highest levels of anger control (Haukkala et al., 2010). These findings were maintained even after researchers took into account demographic and coronary risk factors, as well as depressive symptoms. While not specifically addressing *how* the anger is regulated (e.g., suppressed, situation reappraised), this study suggests that the occurrence of negative emotions such as anger may not be sufficient to induce risk. Instead, it is the failure to regulate the emotion effectively that may help explain anger and CVD risk associations.

Another study of 181 Finnish men and women observed cross-sectional associations of adaptive and maladaptive emotion regulation with metabolic syndrome (Kinnunen, Kokkonen, Kaprio, & Pulkkinen, 2005). *Metabolic syndrome*, which is characterized by hypertension, elevated lipid levels, central adiposity (a measure of body fat distribution), and insulin resistance, is a well-established risk marker for CVD (Expert Panel on Detection Evaluation and Treatment of High Blood Cholesterol in Adults, 2001). In this study, the authors examined whether use of certain emotion regulation strategies including mood repair (e.g., "I am imagining something nice to keep my mood up") and mood maintenance (e.g., "I would not want to change this mood") were associated with metabolic syndrome at age 42. The authors also examined whether emotional ambivalence (a specific form of emotion dysregulation; "I try to suppress my anger but would like to let others know how I feel") was associated with midlife metabolic syndrome. The authors found that mood repair and mood maintenance were significantly associated with reduced risk of metabolic syndrome, whereas emotional ambivalence was associated with higher risk of metabolic syndrome at age 42. This study suggests that effective regulation of emotion may protect cardiovascular health, and emotion dysregulation may contribute to CVD risk. However, this study did not account for prior physical health or other relevant covariates such as socioeconomic status that could provide alternative explanations for the associations observed.

A recent study by our group also observed divergent associations of different emotion regulation strategies as assessed by the Emotion Regulation Questionnaire (Gross & John, 2003) with C-reactive protein (CRP). CRP, an inflammatory risk marker for CVD, is associated with atherosclerosis and incident coronary events (Danesh et al., 2004; Pearson et al., 2003). CRP is often used as a predisease marker of CVD by referencing a diagnostic cut point identified by the Centers for Disease Control and Prevention (CDC)/American Heart Association (Pearson et al., 2003). We examined reappraisal (i.e., altering how one thinks about an emotion-eliciting situation in order to change its emotional impact) and suppression (i.e., inhibiting emotional expression in response to an emotion eliciting event). Among 379 U.S. adults, an increase of one standard deviation in reappraisal was significantly associated with 20% *lower* odds of having levels of CRP at or above the CDC/American Heart Association's high risk cut point (Appleton, Buka, Loucks, Gilman, & Kubzansky, 2013). Conversely, a one standard deviation increase in suppression was significantly associated with 44% *higher* odds of having systemic inflammation levels indicating high CVD risk. Similarly, some preliminary analyses by our research group using the same sample found reappraisal and suppression also be patterned differentially with 10-year risk of developing CVD in midlife (Appleton, Loucks, Buka, & Kubzansky, unpublished data). This emerging body of work provides the first direct population-based evidence that emotion regula-

tion may contribute significantly to cardiovascular health.

Indirect Evidence

While the evidence base explicitly linking emotion regulation to CVD in the general population is small, much indirect evidence comes from studies of poor emotional functioning and negative affective states, which are characterized in part by dysregulated emotion (Kubzansky et al., 2011; Taylor, Lerner, Sage, Lehman, & Seeman, 2004). Chronic distress is thought to influence cardiovascular health by way of repeated and excessive activation of the stress response and impaired adaptation, which over time may contribute to damaging the cardiovascular system (McEwen, 2003), and also risk behaviors associated with poor mental health (e.g., smoking) (Everson-Rose & Lewis, 2005). Work in this area has primarily focused on three negative affective states in association with CVD risk: anger, anxiety, and depression (Chida & Steptoe, 2009; Suls & Bunde, 2005). Though epidemiological studies often consider depression and anxiety as representing single emotions, in fact they reflect complex constellations of chronic elevations of maladaptive cognitions, behaviors, and emotions, of which dysregulated emotion is one feature (Lazarus, 1991). However, given the importance of dysregulated emotion in these affective states, we review studies linking them to CVD, because we speculate that an emotion regulation perspective could add insight to understanding their observed associations. Studies reviewed in this section do not explicitly measure emotion regulation; we therefore characterize them as providing indirect evidence that dysregulated emotion may contribute to CVD.

In a review of 37 studies examining prospective associations of negative emotions with incident CHD events in initially healthy individuals, high anger was associated with 1.5- to threefold higher risk of CHD; chronic anxiety was associated with 1.5- to sevenfold higher risk of CHD; and clinically relevant levels of depression was associated with more than a 2.5-fold elevated risk, with gradations in risk evident according to levels of depressive symptoms (Kubzansky, 2007). Posttraumatic stress disorder, a severe form of distress marked by dysregulated emotion in response to trauma, has also been linked to incident cardiovascular events in military veteran and community-based civilian populations (for a review, see Dedert et al., 2010). Taken together, these studies suggest significantly increased cardiovascular risk associated with experiencing chronic negative emotion and poor emotional functioning that likely derives in part from maladaptive emotion regulation.

Whereas experiencing chronic negative emotions may indicate dysregulation, positive emotional functioning may be considered a marker of effective emotion regulation. A recent review of studies of positive psychological well-being and cardiovascular health identified a consistent protective effect of positive emotions and psychological attributes related to more frequent occurrence of positive emotions (e.g., optimism); findings also suggested that positive emotions and related factors are associated with health-related behaviors that reduce risk of CVD (e.g., increased physical activity) and more resilient biological function (e.g., less systemic inflammation) (Boehm & Kubzansky, 2012). The evidence of CVD buffering attributable to positive functioning is most well established for optimism (Boehm & Kubzansky, 2012; Peterson & Bossio, 2000). For example, in a study of 97,253 participants of the Women's Health Initiative, women with high levels of dispositional optimism had significantly lower risk of incident CHD and CHD mortality compared to those with low levels of optimism over 8 years of follow-up, even after controlling for health risk behaviors, obesity, lipids, and depressive symptoms (Tindle et al., 2009).

Another domain of healthy psychological functioning that may promote cardiovascular health is *emotional vitality*, which can be defined as feeling energetic or full of pep, and having a sense of positive well-being and emotional self-control characterized in part by effective emotion regulation (Kubzansky & Thurston, 2007; Rozanski & Kubzansky, 2005). In a study of 6,025 healthy men and women ages 25–74 years at baseline, those with the highest levels of emotional vitality had 19% reduced risk of developing CHD over 15 years of follow-up compared to those with the lowest levels of emotional vitality (Kubzansky & Thurston, 2007). Effects

were maintained after researchers took into account known coronary risk factors, as well as depression and psychological problems. Taken together, these studies suggest that positive emotional and psychological functioning protect cardiovascular health, providing indirect evidence that effective emotion regulation may increase cardiovascular resilience.

Mechanisms by Which Emotion Regulation Influences CVD

Emotion regulation may affect CVD risk through both physiological and behavioral pathways. As noted earlier, dysregulated emotion is associated with increased likelihood of engaging in deleterious cardiovascular-related health behaviors such as smoking, unhealthy diet, and low physical activity (Kiecolt-Glaser, McGuire, Robles, & Glaser, 2002). Emotion dysregulation is also associated with deteriorative biological functioning in terms of the development of obesity (Blaine, 2008), high levels of cholesterol and blood pressure, atherosclerosis, and inflammation (Appleton, Buka, et al., 2013; Boehm & Kubzansky, 2012). Laboratory-based studies (see earlier sections for examples) suggest that effects may occur via heightened or repeated activation of stress–response systems, including the HPA and SNS. Such overactivation has been linked to a variety of potentially damaging biological alterations, including hemodynamic forces such as increased turbulence and shear stress leading to impaired endothelial function, and flow-related arterial injury leading to increased atherogenesis, as well as altered electrical stability of the heart and increased inflammation (Kubzansky & Kawachi, 2000). For example, heightened HPA and SNS activity and resultant stress hormones (e.g., corticosteroids, catecholamines) initiate an inflammatory response characterized by the production of a number of inflammatory markers, including CRP (Black & Garbutt, 2002). Inflammation is of increased interest in studies of emotion regulation and CVD, because it is strongly implicated in the pathophysiology of CVD (Danesh et al., 2004; Pearson et al., 2003) and has also been linked with several factors related to emotion regulation, including depression (Howren, Lamkin, & Suls, 2009), and chronic and acute stress (Janicki-Deverts, Cohen, Matthews, & Cullen, 2008; Steptoe, Hamer, & Chida, 2007).

Moreover, beyond simply indicating the absence of deterioration, the ability to regulate emotions effectively appears to enhance cardiovascular health actively. Similar to mechanisms associated with dysregulation, effective emotion regulation may promote cardiovascular health by not only preventing deteriorative processes but also promoting restorative health behaviors and biological functioning. Important to note is that deteriorative and restorative processes are not always on a continuum (for a more detailed discussion, see Boehm & Kubzansky, 2012). Relevant health-promoting behaviors include engaging in more opportunities for rest and restoration (Rozanski & Kubzansky, 2005), frequent consumption of fruits and vegetables, improved problem solving, and mobilization of social support (Eisenberg, Hofer, & Vaughan, 2007). Whereas dysregulated emotion may heighten HPA and SNS activity and initiate a cascade of physiological risks for CVD, effectively regulated emotion may prevent such activity and further enhance healthy cardiovascular functioning, although few studies have directly tested this hypothesis. In one of the few empirical tests conducted, a recent study (reviewed earlier) found support for this hypothesis by demonstrating that reappraisal was associated with lower inflammatory risk and suppression was associated with elevated inflammatory risk (Appleton, Buka, et al., 2013). Other forms of healthy psychological functioning that have been identified as possible markers of adaptive emotion regulation (e.g., positive well-being, optimism) have also been linked to relevant biological markers, such as slower rates of progression of carotid atherosclerosis and better endothelial function (Boehm & Kubzansky, 2012; Ikeda et al., 2011).

Important to consider is that emotion regulation strategies are used at different points during the emotion-generative process (Gross, 2001; Gross & John, 2003; John & Gross, 2004), and strategies can be employed at any stage. Thus, it may be that the effect of the emotion regulatory strategy on physiological activation, inflammation, and subsequent CVD risk is due in part to

the timing of use of each strategy. For example, reappraisal is antecedent-focused, which means that it is employed *before* the emotion occurs and involves changing cognitive appraisals about the situation, which then prevents or reduces the intensity of negative emotions (Gross, 2001; Gross & John, 2003; John & Gross, 2004). Therefore, HPA or SNS dysregulation may be avoided, potentially leading to lower levels of systemic inflammation, which in turn may lower CVD risk. On the other hand, suppression, a response-focused strategy employed *after* the emotion has occurred, involves the modification of behavioral manifestations of the emotion (Gross, 2001; Gross & John, 2003; John & Gross, 2004). Though individuals may appear outwardly tranquil, chronic negative emotions and associated HPA and SNS dysregulation may continue internally unchecked, ultimately contributing to increased systemic inflammation and CVD risk. Moreover, suppression has been found to require significant mental exertion (Consedine et al., 2002; Gross, 2001). Thus, the act of suppression may further tax body systems and contribute to elevations in systemic inflammation and CVD risk. Emotion regulation theory and research suggest that regulating the emotion earlier in the emotion-generative process may be more effective than doing so in the later, response-focused stage of the process (Gross, 2001). As such, while the appropriateness of any given strategy is context dependent, *consistent* reliance on response-focused (e.g., suppression) versus antecedent-oriented (e.g., reappraisal) regulation may differentially impact biological processes and long-term CVD risk.

The Way Forward

The confluence of evidence from laboratory- and population-based research strongly suggests that the association between emotion regulation and CVD is not spurious. Moreover, risk and protective associations have been consistently observed across a variety of emotion regulation indicators, signifying that emotion dysregulation may increase CVD risk, whereas effective emotion regulation may lower it. While all the evidence reviewed thus far has been obtained with adults during their middle to later adulthood, a time when CVD is likely to become manifest, many studies indicate that loss of cardiovascular health can begin in childhood and progress over the life course (Berenson & Srnivasan, 2005). Studies of emotion regulation and CVD among adults are ill equipped to answer important etiological questions as to *when* these processes emerge and begin to exert damaging or health-promoting effects. Given that emotion regulation strategies are learned over time, with childhood being a major period of development (Calkins & Hill, 2007; John & Gross, 2004), a more explicit consideration of these relationships over the life course may be highly fruitful. In this section, we argue that the way forward in emotion regulation and CVD research is to move toward framing and evaluating hypotheses from a developmental or life course perspective. Doing so may not only answer important scientific questions but also suggest novel avenues for the prevention of CVD.

Applying a Developmental Perspective

Children are typically born with many of the requisite components of ideal cardiovascular health. They generally have healthy blood pressure, lipid, and glucose levels; they do not smoke and have the potential for developing an ideal body weight, and healthy dietary and physical activity practices. So how does the loss of cardiovascular health occur, and when does it begin to become evident? What role does emotion regulation play in the loss or promotion of cardiovascular health over time? We believe the answers to these questions are related in part to achieving a key socioemotional developmental milestone during childhood: attaining emotion regulation skills.

Emotion regulation is not primarily an inborn trait. Rather, it is a set of strategies learned through socialization and experience over time, with childhood being a key period of development (John & Gross, 2004). Temperament, maturation, and social experiences shape core emotion regulation competencies during childhood, with refinements occurring over the lifespan in accordance with new experiences (Calkins & Hill, 2007; John & Gross, 2004; Zeman, Cassano, Perry-Parrish, & Stegall, 2006).

This developmental or life course perspective can help us understand how achieving this developmental milestone specifically influences CVD risk in adulthood. For example, children who develop patterns of dysregulated emotion regulation (e.g., impulsivity, poor attention, depressed mood) generally continue to exhibit such patterns through adolescence and adulthood (Brame, Nagin, & Tremblay, 2001; Caspi, Moffitt, & Newman, 1996; Dekker et al., 2007). Thus, as they age, children with such patterns of regulation are more likely to initiate and maintain a range of risk-related health behaviors (e.g., cigarette smoking, sedentary behavior, poor diet) that over time may contribute to disease development and progression (Shonkoff & Phillips, 2000).

Developmental or *life course epidemiology* is the study of chronic disease risk in terms of the long-term effects of health-relevant experiences that occur during gestation, childhood, adolescence, and adulthood (Ben-Shlomo & Kuh, 2002). Experiences can be biological, behavioral, environmental (physical and social), and psychological in nature. Emotion regulation can be considered one such factor that may have positive or negative health implications over time. From this perspective, emotion regulation may affect disease risk mainly in two ways. First, a latency model suggests that both adverse and health-promoting experiences during particular sensitive or critical periods of development will have lasting effects on health and functioning, with effects emerging years or decades after the initial experience (Ben-Shlomo & Kuh, 2002; Shonkoff, Boyce, & McEwen, 2009). Such a model implies that early prevention and intervention are crucial. Second, an accumulation model specifies that the number and duration of experiences cumulatively add up to affect health, again, often with a significant amount of time passing before the effects manifest as health outcomes (Ben-Shlomo & Kuh, 2002; Shonkoff et al., 2009). This perspective suggests multiple windows of opportunity for prevention and intervention, because an accumulation model would suggest that positive and negative factors cumulatively build to determine disease resilience and risk, respectively.

Because both latency and accumulation of risk explanations are plausible and not mutually exclusive, it is possible that emotion regulation skills originating during childhood shape developing brain architecture and biological systems, as well as contribute to behaviors that together and independently influence later cardiovascular risk. Thus, developing poor emotion regulation skills during childhood may have lifelong cardiovascular consequences by negatively altering biological systems during sensitive periods of development *and* through accumulated damage over time. For example, persistent psychological distress (i.e., dysregulated emotion) is associated with activation of the HPA axis (Luppino et al., 2010). Prolonged HPA activity is thought to up-regulate hormones that influence appetite and promote weight gain (Luppino et al., 2010). As such, poor childhood emotion regulation may alter developing metabolic processes during a sensitive period of development, which in turn may increase risk of obesity and other conditions linked with increased susceptibility to developing CVD (e.g., elevated inflammation, dyslipidemia) later in life. Conversely, learning effective emotion regulation skills early in life may prevent such risk and promote resiliency by helping to buffer such deleterious stress reactivity and prevent such damage to biological systems. Also, psychological distress/dysregulated emotion is associated with behaviors related to higher risk of obesity that may emerge even in childhood (and continue through adolescence and into adulthood), such as emotional eating, consumption of calorie-dense foods, and decreased physical activity (Blaine, 2008), whereas adaptive emotional functioning is associated with greater likelihood of engaging in health-promoting behaviors (Boehm & Kubzansky, 2012).

While the American Heart Association's national goals for cardiovascular health promotion emphasize that cardiovascular risk originates early in life and urge study of early-life antecedents (Lloyd-Jones et al., 2010), considering emotion regulation and CVD risk from a developmental perspective is highly novel. While there is a growing literature on child emotion regulation and adult CVD risk, we do not know how early in life these processes may be evident, because research examining emotion regulation and biological markers of CVD risk are scant. We turn now to the emerging empiri-

cal evidence linking childhood emotion regulation to adult CVD risk.

Evidence Linking Child Emotion Regulation to Adult CVD Risk

Among the limited set of prospective studies on associations between child emotion regulation and adult CVD risk, most have focused on upstream risk markers for CVD, such as obesity and inflammation, rather than on objectively measured clinical endpoints of disease (e.g., myocardial infarction, stroke). This may be due to (1) limited availability of prospectively assessed information on childhood emotional functioning in cohort studies that were initiated decades ago, and (2) the relative youth of participants in the longitudinal cohorts that have collected information on child emotional functioning and adult health, so that actual CVD outcomes have not yet developed. Moreover, most studies assess primarily poor emotional functioning, behavior problems, or psychological disorders. These conditions can be considered markers of or proxies for dysregulated emotion, but they do not provide direct assessments of emotion regulation. As discussed previously in our review of studies of depression and anxiety with CVD risk, we do not conflate poor emotion regulation with psychopathology. Instead, we acknowledge that child behavior disorders and emotional functioning reflect a complex set of factors that include problems with emotion regulation. Thus, these studies are suggestive, and we encourage future work to measure child emotion regulation explicitly in relation to later cardiovascular risk. That said, findings from life course studies of child emotional functioning and behavior problems, and adult CVD risk are congruent with associations observed in studies of adults and suggest that cardiovascular risk may have developmental origins in child emotion regulation.

For example, while the direction of the relation between emotional functioning and obesity is debated and is likely to be bidirectional (Atlantis & Baker, 2008; Luppino et al., 2010), several studies have found that poor child emotional functioning contributes to the development of obesity in adulthood (Duarte et al., 2010; Goodwin et al., 2008; Mamun et al., 2009). Mamun et al., in a prospective study of 2,278 Australian boys and girls, found that child behavior problems (a marker of emotional dysfunction) at ages 5 and 14 years were associated with 4.6 higher risk (95% confidence interval (CI): 2.36, 9.06) of obesity at age 21, once they controlled for maternal demographic and lifestyle variables, child dietary patterns, TV viewing, family meals, and physical activity. In a study of 2,209 Finnish boys, Duarte et al. (2010) found that both moderate and high levels of conduct problems (which may also mark severely dysregulated emotion) at age 8 years were associated with two- to threefold higher risk of overweight and obesity at ages 18–23, compared to low levels of conduct problems. These associations were maintained when the researchers controlled for hyperactivity and sociodemographic variables.

Similar findings have been observed in studies of childhood emotional functioning and systemic inflammation in adulthood. For example, in a prospective study of 379 U.S. adults, multiple domains of child emotional functioning (directly assessed by a study psychologist when participants were age 7) were examined in association with CRP levels in middle adulthood. Poor child self-regulation (i.e., unrestrained, impulsive behavior) and distress proneness (i.e., emotionally labile, easily frustrated) were each associated with elevated CRP levels 35 years later, after researchers controlled for demographics, being born small for gestational age, child health status, child body mass index, and child IQ (Appleton et al., 2011). Associations were robust, because poor child emotional functioning was associated with 2.3- to 3.9-fold greater odds of having CRP levels at or above the CDC/American Heart Association's cut point for being at high-risk for CVD (Pearson et al., 2003). Moreover, there was evidence that these associations were mediated by adult weight status and modified by early life socioeconomic status (Appleton et al., 2011, 2012). In other words, poor childhood emotional functioning increased inflammatory risk in adulthood by way of higher body mass index, and such associations were more robust for children growing up in poorer environments than for those from higher level socioeconomic environments (see also Chen and Miller, this volume, for a more

detailed discussion of the interrelationships between socioeconomic status and adversity, emotion regulation, and health). In a study of 526 male participants of the Dunedin, New Zealand cohort, Odgers et al. (2007) examined childhood conduct problem levels assessed repeatedly from ages 7–26 years in association with various physical health outcomes at age 32, including CVD risk factor clustering (a composite of factors including overweight, high blood pressure, and dysregulated lipid levels, among others) and inflammation, as measured by CRP. Compared to those with low levels, boys with persistently high levels of conduct problems had 2.9 higher odds (95% CI: 1.3–6.2) of having high-risk CRP in adulthood. Adulthood CVD risk factor clustering was also higher in those with persistent conduct problems than in better functioning children, but the association was not significant (Odgers et al., 2007).

While measures of behavior problems can be considered markers of emotion dysregulation, they ultimately provide somewhat limited insight into the broader relationship between emotion regulation and CVD risk, since they measure emotional functioning at only one end of the spectrum rather than considering the range of functioning. Few life course studies have considered whether adaptive or effective emotion regulation in childhood is associated with reduced CVD risk in adulthood. This is a critical gap in the literature. To address this issue, our research group has conducted two preliminary studies that examine the potential cardiovascular benefit of effective emotion regulation in childhood.

First, in a prospective study of 377 U.S. adult men and women, we examined associations of effective and poor emotion functioning, assessed by a study psychologist when participants were age 7, with the 10-year risk of developing CVD in their 40s (Appleton, Loucks, Buka, Rimm, & Kubzansky, 2013). For women, a one standard deviation increase in child attention regulation (ability to stay focused, considered an effective regulatory strategy) was marginally associated with 8% reduced risk ($p = .09$) of developing CVD in the next 10 years, and a one standard deviation increase in distress proneness (considered poor functioning) was significantly associated with 31% higher risk. For men, no association was observed for attention regulation and 10-year CVD risk, but each standard deviation increase in child distress proneness was significantly associated with 17% higher risk. These associations were maintained when we controlled for childhood cardiovascular health, body mass index, chronic conditions, IQ, socioeconomic status, being born small for gestational age, and demographic factors. Next, in another prospective study within this sample, we considered the role of attention regulation in association with the development of a favorable cardiovascular health profile at age 42 (Appleton et al., 2013). This profile is characterized by low blood pressure, low body mass index, healthy lipid levels, not smoking, and not having diabetes (Daviglus et al., 2004; Lloyd-Jones, Dyer, Wang, Daviglus, & Greenland, 2007). A one standard deviation increase in child attention regulation was significantly associated with 40% higher odds of having favorable cardiovascular health in midlife. Of note, this association was maintained when we adjusted for early life cardiovascular health, socioeconomic status, and potential psychosocial and behavioral pathway variables from adulthood. These studies suggest that earlier acquisition of effective emotion regulation may reduce risk of CVD and promote early development of healthy cardiovascular functioning.

Summary and Recommendations for Future Work

Taken together, the evidence from laboratory- and population-based studies in adulthood and over the life course suggest that poor emotion regulation may impair cardiovascular function and increase risk, whereas effective emotion regulation may reduce risk and promote cardiovascular health. Moreover, evidence is accumulating that CVD may have developmental origins in child emotion regulation that influence trajectories of risk or resilience earlier than was previously considered. However, as is evident from this examination of the literature, research in this area is still limited, and additional work is needed to address critical remaining issues. For example, studies that explicitly measure emotion regulation in

conjunction with cardiovascular health and functioning over time (and across a range of outcomes) will help to identify *when* these processes emerge and exert damaging or health-promoting effects. In addition, studies that consider not only dysregulation but also effective emotion regulation in relation to CVD outcomes would provide clearer understanding of the spectrum of effects. Moreover, epidemiological research should be informed by experimental findings, and vice versa, to ensure that causal inferences are accurate and to guide efficient exploration of how effects may occur. Research on pathways, behavioral and biological, also remain limited but could facilitate distinguishing between latency and cumulative effects, thereby indicating key periods for targeting prevention and intervention strategies. In fact, the earlier reported findings that effects of emotion regulation were modified by socioeconomic status are particularly intriguing, suggesting that effects of emotion regulation on health may well be modifiable (Appleton et al., 2012). It may be that when more resources are available to children who are somewhat dysregulated, regulatory capacity can be improved, thereby leading to better health over the life course (for related discussion of this issue, see Chen and Miller, this volume).

We suggest that work in this area is exciting and worth pursuing, because it has a number of important implications, both for understanding emotion regulation more generally and for thinking about it as a lifelong critical asset. Capacity to regulate emotions may have more far-reaching effects than have previously been identified, and timing of acquisition of this capacity may matter for physical health and perhaps other outcomes as well. Thus, capacity to regulate emotion is important not only as an outcome in its own right, but also as a determinant of health, which suggests it could play an important role in health-related prevention strategies.

True prevention of CVD must involve the prevention of the development of CVD risk factors in the first place. This "primordial prevention" has become increasingly more interesting in public health and medicine (Lloyd-Jones et al., 2010). While experts in cardiovascular health largely acknowledge that childhood is a life stage particularly amenable to CVD prevention, efforts at modifying risk during this time are not yet well established. In general, public health and biomedicine have not focused on the sensitivity of child emotion development to help safeguard cardiovascular or other forms of lifelong health, or prioritized emotional development for resource allocation as a way to reduce disease burden in adulthood. Because we believe research in this area will continue to indicate the central importance of developing emotion regulatory capacity, we suggest that greater resources be dedicated to promoting the acquisition of healthy emotion regulation strategies throughout life, with a particular emphasis on early life stages.

However, to our knowledge, programs designed to promote the development of healthy emotion regulation skills among children in the general population are scant. Work in this area is generally intervention-focused rather than prevention-oriented. Thus, efforts target pediatric populations with identified mental health problems, with the aim of building or improving emotion regulation skills. For example, *contextual emotion regulation therapy*, a developmentally based treatment approach for pediatric depression, involves learning to manage emotional distress by identifying contexts that elicit maladaptive emotional responses, then incorporating more adaptive responses that can be used even in challenging situations (Kovacs & Lopez-Duran, 2012). This approach is developmentally focused, which means that therapeutic approaches are age-appropriate and tailored to the child's developmental stage. Moreover, there are a number of effective school-based programs designed to prevent anxiety and promote emotional resilience in children through cognitive-behavioral therapy and other forms of psychotherapy (for a review, see Neil & Christensen, 2009). Such programs might provide the basis for thinking about prevention strategies that could be implemented on a larger scale, either in population-based studies or with high-risk populations. Thus, prevention programs could consider incorporating these and other skills-building techniques typically used in traditional psychological treatments for child emotion dysregulation (e.g., guided practice in using adaptive strategies, role playing, parental involvement) in the design of programs

to build effective emotion regulation skills and emotional resilience for children at all levels of functioning in the general population (Kovacs & Lopez-Duran, 2012; Zeman et al., 2006). With future laboratory- and population-based studies working from a developmental perspective, we can continue to build the evidence base in this area and inform efforts to enhance adaptive emotion regulation capacities at a population level. Doing so will not only improve the emotional health of children but may also protect their health for a lifetime.

References

Appleton, A. A., Buka, S. L., Loucks, E. B., Gilman, S. E., & Kubzansky, L. D. (2013). Divergent associations of adaptive and maladaptive emotion regulation strategies with inflammation. *Health Psychology, 13*, 748–756.

Appleton, A. A., Buka, S. L., Loucks, E. B., Martin, L. T., & Kubzansky, L. D. (2013). A prospective study of positive early life psychosocial factors and favorable cardiovascular risk in adulthood. *Circulation, 12*, 905–912.

Appleton, A. A., Buka, S. L., McCormick, M. C., Koenen, K. C., Loucks, E. B., Gilman, S. E., et al. (2011). Emotional functioning at age 7 years is associated with C-reactive protein in middle adulthood. *Psychosomatic Medicine, 73*(4), 295–303.

Appleton, A. A., Buka, S. L., McCormick, M. C., Koenen, K. C., Loucks, E. B., & Kubzansky, L. D. (2012). The association between childhood emotional functioning and adulthood inflammation is modified by early life socioeconomic status. *Health Psychology, 31*(4), 413–422.

Appleton, A. A., Loucks, E. B., Buka, S. L., Rimm, E., & Kubzansky, L. D. (2013). Childhood emotional functioning and the developmental origins of cardiovascular disease risk. *Journal of Epidemiology and Community Health, 67*, 405–411.

Atlantis, E., & Baker, M. (2008). Obesity effects depression: Systematic review of epidemiological studies. *International Journal of Obesity, 32*, 881–891.

Ben-Shlomo, Y., & Kuh, D. (2002). A life course approach to chronic disease epidemiology: Conceptual models, empirical challenges and interdisciplinary perspectives. *International Journal of Epidemiology, 31*, 285–293.

Berenson, G. S., & Srnivasan, S. R. (2005). Cardiovascular risk factors in youth with implications for aging: The Bogalusa heart study. *Neurobiology of Aging, 26*, 303–307.

Black, P. H., & Garbutt, L. D. (2002). Stress, inflammation and cardiovascular disease. *Journal of Psychosomatic Research, 52*, 1–23.

Blaine, B. (2008). Does depression cause obesity?: A meta-analysis of longitudinal studies of depression and weight control. *Journal of Health Psychology, 13*, 1190–1197.

Boehm, J. K., & Kubzansky, L. D. (2012). The heart's content: The association between positive psychological well-being and cardiovascular health. *Psychological Bulletin, 138*(4), 655–691.

Brame, B., Nagin, D. S., & Tremblay, R. E. (2001). Developmental trajectories of physical aggression from school entry to late adolescence. *Journal of Child Psychology and Psychiatry, 42*(4), 503–512.

Butler, E. A., Wilhelm, F. H., & Gross, J. J. (2006). Respiratory sinus arrhythmia, emotion and emotion regulation during a social interaction. *Psychophysiology, 43*(6), 612–622.

Calkins, S. D., & Hill, A. (2007). Caregiver influences on emerging emotion regulation: Biological and environmental transactions in early development. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 229–248). New York: Guilford Press.

Caspi, A., Moffitt, T. E., & Newman, D. L. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. *Archives of General Psychiatry, 53*, 1033–1039.

Chen, E., & Miller, G. (this volume, 2014). Early-life socioeconomic status, emotion regulation, and the biological mechanisms of disease across the lifespan. In J. J. Gross (Ed.), *Hanbook of emotion regulation* (2nd ed., pp. 586–595). New York: Guilford Press.

Chida, Y., & Hamer, M. (2008). Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: A quantitative review of 30 years of investigations. *Psychological Bulletin, 134*(6), 829–885.

Chida, Y., & Steptoe, A. (2009). The association of anger and hostility with future coronary heart disease: A meta-analytic review of prospective evidence. *Journal of the American College of Cardiology, 53*(11), 936–946.

Cohen, H., Benjamin, J., Geva, A. B., Matgar,

M. A., Kaplan, Z., & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: Application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry Research, 25*, 1–13.

Consedine, N. S., Magai, C., & Bonanno, G. A. (2002). Moderators of the emotion inhibition-health relationship: A review and research agenda. *Review of General Psychology, 6*(2), 204–228.

Danesh, D., Wheeler, J. G., Hirschfield, G. M., Eda, S., Eiriksdottir, G., Rumley, A., et al. (2004). C-reactive protein and other circulating markers of inflammation in the prediction of coronary heart disease. *New England Journal of Medicine, 350*(14), 1387–1397.

Daviglus, M. L., Stamler, J., Pirzada, A., Yan, L. L., Garside, D. B., Liu, K., et al. (2004). Favorable cardiovascular risk profile in young women and long-term risk of cardiovascular and all-cause mortality. *Journal of the American Medical Association, 292*(13), 1588–1592.

Dedert, E. A., Calhoun, P. S., Watkins, L. L., Sherwood, A., & Beckham, J. C. (2010). Post-traumatic stress disorder, cardiovascular, and metabolic disease: A review of the evidence. *Annals of Behavioral Medicine, 39*(1), 61–78.

Dekker, M. C., Ferdinand, R. F., van Lang, N. D., Bongers, I. L., van der Ende, J., & Verhulst, F. C. (2007). Developmental trajectories of depressive symptoms from early childhood to late adolescence: Gender differences and adult outcome. *Journal of Psychology and Psychiatry and Allied Disciplines, 48*(7), 657–666.

Denson, T. F., Grisham, J. R., & Moulds, M. L. (2011). Cognitive reappraisal increases heart rate variability in response to an anger provocation. *Motivation and Emotion, 35*, 14–22.

Duarte, C. S., Sourander, A., Nikolakaros, G., Pihlajamaki, H., Helenius, H., Piha, J., et al. (2010). Child mental health problems and obesity in early adulthood. *Journal of Pediatrics, 156*(1), 93–97.

Eisenberg, N., Hofer, C., & Vaughan, J. (2007). Effortful control and its socioemotional consequences. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 287–306). New York: Guilford Press.

Everson-Rose, S. A., & Lewis, T. T. (2005). Psychosocial factors and cardiovascular disease. *Annual Review of Public Health, 26*, 469–500.

Expert Panel on Detection Evaluation and Treatment of High Blood Cholesterol in Adults. (2001). Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *Journal of the American Medical Association, 285*(19), 2486–2497.

Folsom, A. R., Yatsuya, H., Nettleton, J. A., Lutsey, P. L., Cushman, M., & Rosamond, W. (2011). Community prevalence of ideal cardiovascular health, by the American Heart Association definition, and relationship with cardiovascular disease incidence. *Journal of the American Journal of Cardiology, 57*(16), 1690–1696.

Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin, 132*(6), 823–865.

Goodwin, R. D., Sourander, A., Duarte, C. S., Niemela, S., Multimaki, P., Nikolakaros, G., et al. (2008). Do mental health problems in childhood predict chronic physical conditions among males in early adulthood?: Evidence from a community-based prospective study. *Psychological Medicine, 39*(2), 301–311.

Gross, J. J. (2001). Emotion regulation in adulthood: Timing is everything. *Current Directions in Psychological Science, 10*(6), 214–219.

Gross, J. J. (this volume, 2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3–20). New York: Guilford Press.

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships and well-being. *Journal of Personality and Social Psychology, 85*(2), 348–362.

Gross, J. J., & Levenson, R. W. (1993). Emotion suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64*(6), 970–986.

Gross, J. J., & Levenson, R. W. (1997). Hiding feelings: The acute effects of inhibiting negative and positive emotion. *Journal of Abnormal Psychology, 106*(1), 95–103.

Haukkala, A., Konttinen, H., Laatikainen, T., Kawachi, I., & Uutela, A. (2010). Hostility, anger control, and anger expression as predictors of cardiovascular disease. *Psychosomatic Medicine, 72*, 556–562.

Howren, M. B., Lamkin, D. M., & Suls, J. (2009). Associations of depression with C-reactive

protein, IL-1, and IL-6: A meta-analysis. *Psychosomatic Medicine, 71*, 171–186.

Ikeda, A., Schwartz, J., Peters, J. L., Fang, S., Spiro, A., III, Sparrow, D., et al. (2011). Optimism in relation to inflammation and endothelial dysfunction in older men: The VA Normative Aging Study. *Psychosomatic Medicine, 73*(8), 664–671.

Janicki-Deverts, D., Cohen, S., Matthews, K. A., & Cullen, M. R. (2008). History of unemployment predicts future elevations in C-reactive protein among male participants in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Annals of Behavioral Medicine, 36*, 176–185.

John, O. P., & Gross, J. J. (2004). Healthy and unhealthy emotion regulation: Personality processes, individual differences, and life span development. *Journal of Personality, 72*(6), 1301–1334.

Kiecolt-Glaser, J. K., McGuire, L., Robles, T. F., & Glaser, R. (2002). Emotions, morbidity, and mortality: New perspectives from psychoneuroimmunology. *Annual Review of Psychology, 53*, 87–107.

Kinnunen, M., Kokkonen, M., Kaprio, J., & Pulkkinen, L. (2005). The associations of emotion regulation and dysregulation with the metabolic syndrome factor. *Journal of Psychosomatic Research, 58*, 513–521.

Kovacs, M., & Lopez-Duran, N. L. (2012). Contextual emotion regulation therapy: A developmentally based intervention for pediatric depression. *Child and Adolescent Psychiatric Clinics of North America, 21*(2), 327–343.

Kubzansky, L. D. (2007). Sick at heart: The pathophysiology of negative emotions. *Cleveland Clinic Journal of Medicine, 74*(S1), S67–S72.

Kubzansky, L. D., & Kawachi, I. (2000). Going to the heart of the matter: Do negative emotions cause coronary heart disease? *Journal of Psychosomatic Research, 48*, 323–337.

Kubzansky, L. D., Park, N., Peterson, C., Vokonas, P., & Sparrow, D. (2011). Healthy psychological functioning and incident coronary heart disease: The importance of self-regulation. *Archives of General Psychiatry, 68*(4), 400–408.

Kubzansky, L. D., & Thurston, R. C. (2007). Emotional vitality and incident coronary heart disease: Benefits of healthy psychological functioning. *Archives of General Psychiatry, 64*(12), 1393–1401.

Lam, S., Dickerson, S. S., Zoccola, P. M., & Zaldivar, F. (2009). Emotion regulation and cortisol reactivity to a social-evaluative speech task. *Psychoneuroendocrinology, 34*, 1335–1362.

Lazarus, R. S. (1991). *Emotion and adaptation*. New York: Oxford University Press.

Lloyd-Jones, D. M., Adams, R., Carnethon, M., De Simone, G., Ferguson, T. B., Flegal, K., et al. (2009). Heart disease and stroke statistics 2009 update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation, 119*, e21–e181.

Lloyd-Jones, D. M., Dyer, A. R., Wang, R., Daviglus, M. L., & Greenland, P. (2007). Risk factor burden in middle age and lifetime risks for cardiovascular and non-cardiovascular death (Chicago Heart Association Detection Project in Industry). *American Journal of Cardiology, 99*, 535–540.

Lloyd-Jones, D. M., Hong, Y., Labarthe, D., Mozaffarian, D., Appel, L. J., Van Horn, L., et al. (2010). Defining and setting national goals for cardiovascular health promotion and disease reduction: The American Heart Association's strategic impact goal through 2020 and beyond. *Circulation, 121*, 586–613.

Luppino, F. S., de Wit, L. M., Bouvy, P. F., Stijnen, T., Cuijpers, P., Penninx, B. W., & Zitman, F. G. (2010). Obesity, overweight, and depression: A systematic review and meta-analysis of longitudinal studies. *Archives of General Psychiatry, 67*(3), 220–229.

Mamun, A. A., O'Callaghan, M. J., Cramb, S. M., Najman, J. M., Williams, G. M., & Bor, W. (2009). Childhood behavioral problems predict young adults' BMI and obesity: Evidence from a birth cohort study. *Obesity, 17*, 761–766.

McEwen, B. S. (2003). Mood disorders and allostatic load. *Biological Psychiatry, 54*, 200–207.

Mendis, S., Puska, P., & Norrving, B. (2011). *Global atlas on cardiovascular disease prevention and control*. Geneva: World Health Organization.

National Heart Lung and Blood Institute. (2007). Morbidity and mortality: 2007 chart book on cardiovascular, lung, and blood diseases. Bethesda, MD: Author.

Neil, A. L., & Christensen, H. (2009). Efficacy and effectiveness of school-based prevention and early intervention programs for anxiety. *Clinical Psychology Review, 29*(3), 208–215.

Odgers, C. L., Caspi, A., Broadbent, J. M.,

Dickson, N., Hancox, R. J., Harrington, H., et al. (2007). Prediction of differential adult health burden by conduct problem subtypes in males. *Archives of General Psychiatry, 64*, 476–483.

Pearson, T. A., Mensah, G. A., Alexander, R. W., Anderson, J. L., Cannon, R .O., Criqui, M., et al. (2003). Markers of inflammation and cardiovascular disease: Application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation, 107*, 499–511.

Pennebaker, J. W., & Beall, S. K. (1986). Confronting a traumatic event: Toward an understanding of inhibition and disease. *Journal of Abnormal Psychology, 95*(3), 274–281.

Peterson, C., & Bossio, L. M. (2000). Optimism and physical well-being. In E. C. Chang (Ed.), *Optimism and pessimism: Implications for theory, research, and practice* (pp. 127–146). Washington, DC: American Psychological Association.

Rothbart, M., Sheese, B. E., & Posner, M. I. (this volume, 2014). Temperament and emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 305–320). New York: Guilford Press.

Rozanski, A., & Kubzansky, L. D. (2005). Psychologic functioning and physical health: A paradigm of flexibility. *Psychosomatic Medicine, 67*(Suppl. 1), S47–S53.

Salovey, P., Stroud, L. R., Woolery, A., & Epel, E. S. (2002). Perceived emotional intelligence, stress reactivity, and symptom reports: Further explorations using the trait meta-mood scale. *Psychology and Health, 17*, 611–627.

Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities. *Journal of the American Medical Association, 301*(21), 2252–2259.

Shonkoff, J. P., & Phillips, D. A. (Eds.). (2000). *From neurons to neighborhoods: The science of early childhood development*. Washington, DC: National Academy Press.

Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain, Behavior, and Immunity, 21*, 910–912.

Suls, J., & Bunde, J. (2005). Anger, anxiety, and depression as risk factors for cardiovascular disease: The problems and implications of overlapping affective dispositions. *Psychological Bulletin, 131*(2), 260–300.

Taylor, S. E., Lerner, J. S., Sage, R. M., Lehman, B. J., & Seeman, T. E. (2004). Early environment, emotions, responses to stress, and health. *Journal of Personality 72*(6), 1365–1393.

Thayer, J. F., & Lane, R. D. (2007). The role of vagal function in the risk for cardiovascular disease and mortality. *Biological Psychology, 74*(2), 224–242.

Thompson, R. A. (this volume, 2014). Socialization of emotion and emotion regulation in the family. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 173–186). New York: Guilford Press.

Tindle, H. A., Chang, Y., Kuller, L. H., Manson, J. E., Robinson, J. G., Rosal, M. C., et al. (2009). Optimism, cynical hostility, and incident coronary heart disease and mortality in the Women's Health Initiative. *Circulation, 120*, 656–662.

Willmott, L., Harris, P., Gellaitry, G., Cooper, V., & Horne, R. (2011). The effects of expressive writing following first myocardial infarction: A randomized controlled trial. *Health Psychology, 30*(5), 642–650.

Zeman, J., Cassano, M., Perry-Parrish, C., & Stegall, S. (2006). Emotion regulation in children and adolescents. *Developmental and Behavioral Pediatrics, 27*(2), 155–168.

CHAPTER 36

Emotion and Self-Regulation Failure

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As most people can attest, conquering vices and changing bad habits are difficult. Even when the motivation to change is strong, self-control failures are common. Subjectively, it often feels as though our capacity to self-regulate waxes and wanes in the face of new temptations, changing moods, and fatigue. Contemporary investigations into the causes of self-regulation failure have demonstrated that the ability to self-regulate can be undermined by a variety of threats that act by impairing awareness, exhausting limited resources, or increasing the salience of temptations. Perhaps the most potent of these threats is negative affect. When people experience emotional distress, be it in the form of a bad mood, disappointment, or social rejection, they often find it more difficult to resist temptations or to suppress unwanted impulses and may engage in various forms of self-defeating behaviors (for a review, see Baumeister, 1997). However, the relationship between emotions and self-control is by no means all one-way; too much self-regulation over a period of time can increase emotional reactivity, as well as impair an individual's ability to regulate his or her emotions.

Much like the experience of hunger or thirst, emotions serve to motivate behavior and predispose individuals toward certain actions (e.g., Keltner & Gross, 1999; Levenson, 1994). It has been suggested that one function of emotion is to indicate whether individuals are succeeding or failing to meet their goals (Carver & Scheier, 1990). Therefore, just as it would be fatally maladaptive for an organism to have the ability to turn off thirst, it would be just as detrimental to be able to completely shut down emotions. However, there are times when our emotions interfere with our goals or may lead us to further distress, such as when our mood causes us to violate social norms (i.e., being tragically depressed on a first date or overjoyed at a funeral). Thus, it is often in our best interests to be able to regulate our affective states.

In this chapter, we take the view that regulating emotions relies on much of the same cognitive and neural machinery as regulating other responses (e.g., thoughts, behaviors, impulses) with the understanding that, all things being equal, emotions are more difficult to suppress or inhibit. Unlike behaviors, thoughts, or cravings, all of which tend to have a clear target of regulation (e.g., "I must not grab the cigarette and light it"), affect is typically more diffuse, with no clear action to regulate. Indeed, one of the central findings of research on emotion regulation is that direct, response-focused attempts to suppress the outward expression of emotion are more cognitively taxing and less success-

ful than antecedent-focused methods such as distraction and reappraisal (Richards & Gross, 2000). Although there may very well be advantages to considering emotion regulation as a separate domain (i.e., Gross, 2002), for the present discussion we consider it fruitful to think of emotion regulation as another type of self-regulation, thereby rendering it subject to the same vulnerabilities and threats as other forms of self-control (see Heatherton & Wagner, 2011).

In the following sections, we examine the relationship among emotions, emotion regulation, and self-regulation failure. We focus primarily on intrinsic forms of emotion regulation, in which individuals regulate their own internal affective states, rather than on extrinsic emotion regulation, in which individuals attempt to influence the emotions of others through affect displays or other means. Along with an overview of the mechanisms whereby emotions can derail self-regulation, we also consider the case of misregulation, for example, when individuals seek out temptations (e.g., food, drugs or alcohol) as a misguided strategy to repair their mood or escape from aversive self-awareness resulting from overindulgence of these same temptations. In addition, we review findings that regulation of emotions exerts a cost on self-regulatory capacity and, conversely, that engaging in effortful self-regulation can impair emotion regulation. Finally, we highlight recent research suggesting that emotional reactivity is increased when self-regulatory resources are depleted.

The Role of Negative Affect in Self-Regulation Failure

When dieters, substance abusers, or sexual offenders are asked to describe their reasons for engaging in harmful behaviors (e.g., binge eating, sexual aggression, smoking, and drug use) they overwhelmingly report that their actions were triggered by negative affect (Haedt-Matt & Keel, 2011; Kassel, Stroud, & Paronis, 2003; Pithers, Kashima, Cumming, Beal, & Buell, 1988; Sinha, 2007). Among the general population, negative affect is similarly associated with impulsive and self-defeating behaviors, such as alcohol consumption (Witkiewitz & Villarroel, 2009), gambling (Raviv, 1993), risky sexual behavior (Bousman et al., 2009; Roberts et al., 2012), excessive Internet usage (LaRose, Lin, & Eastin, 2003), and aggression (Berkowitz, 1989). Although multiple emotions can be considered negative or positive in valence, the extant literature on the role of affect in self-regulation seldom dissociates them. Here, too, we employ the broad categories of negative and positive affect, noting that these necessarily subsume many different emotion categories.

The role of negative affect in initiating self-regulation failure has also been studied in experimental settings using a variety of affect induction procedures. Generally, these procedures involve exposure to sad or aversive stimuli in the form of images, music, or movie clips. Other commonly used techniques include writing about negative life events (i.e., a funeral) or reading a series of increasingly negative self-referential statements, such as in the Velten (1968) mood induction procedure. Much of this work has been conducted within the realm of drug and alcohol addiction, where negative affect has long been known to be the most potent cause of relapse (e.g., Marlatt & J. Gordon, 1985). In smokers and other substance abusers, inducing negative affect in the laboratory has been shown to increase substance cravings (Childress et al., 1994; Fox, Bergquist, Hong, & Sinha, 2007; Tiffany & Drobes, 1990; Willner & Jones, 1996) and the intensity of substance use (McKee et al., 2011) when compared to neutral or positive mood inductions.

Chronic dieters are another population that has received considerable attention with regards to the relationship between negative affect and self-regulation failure (i.e., disinhibited eating). Inducing negative emotional states in dieters increases eating both in comparison to non-dieters, but also to dieters in a neutral mood (e.g., Baucom & Aiken, 1981; Heatherton, Striepe, & Wittenberg, 1998; Herman & Polivy, 1975). For instance, Heatherton, Herman, and Polivy (1991) induced negative affect either by giving participants negative performance feedback during a cognitive task or by instructing them that they would have to prepare a speech that would be given in the presence of their peers. In order to measure overeating, participants were asked to participate in an unrelated taste-test of various flavors

of ice cream, which, unbeknownst to them, had been weighed prior to the task so that the amount of ice cream consumed could be calculated afterwards. In both cases, dieters ate significantly more ice cream than non-dysphoric subjects. Moreover, this occurred even when negative affect was induced by having participants anticipate a future task (i.e., public speaking). Similar findings have been found for social drinkers, in whom fear of future social evaluations increases alcohol consumption relative to participants who were not going to be evaluated (Higgins & Marlatt, 1975).

Social rejection is another method of inducing negative affect that has a rich history of being associated with behavioral disinhibition, aggression, and violent crimes (e.g., high school shootings; Leary, Kowalski, Smith, & Phillips, 2003). Laboratory studies in which subjects are induced to feel socially excluded have found that being rejected increases aggression (DeWall, Twenge, Bushman, Im, & Williams, 2010; Twenge, Baumeister, Tice, & Stucke, 2001; Warburton, Williams, & Cairns, 2006) and reduces the willingness to help others (Twenge, Baumeister, DeWall, Ciarocco, & Bartels, 2007). For example, when given the opportunity to decide how much hot sauce to administer to a group of people who had previously been identified as disliking spicy foods, rejected individuals assigned four times more hot sauce than nonrejected peers (Warburton et al., 2006). With respect to self-regulation failures, social exclusion has been shown to have the same effects as other negative affect inductions, leading to overeating (Baumeister, DeWall, Ciarocco, & Twenge, 2005; Oaten, Williams, Jones, & Zadro, 2008) and reduced persistence on difficult tasks (Baumeister et al., 2005). Thus it seems that, as with negative affect, social rejection can lead to self-regulation failure, although with an added dose of aggression.

Why Does Negative Affect Impair Self-Control?

So far we have reviewed various lines of evidence demonstrating that negative affect can precipitate a variety of maladaptive behaviors, most of which are indicative of poor self-control. In the following sections we consider a number of mechanisms that have been proposed to explain the disinhibiting effects of negative affect. Many of these mechanisms target specific aspects of self-regulation, so it is important at this stage to briefly review some of the core components involved in theories of self-regulation.

Although the details vary, most models of self-regulation can be said to deal with three basic components. The first involves a target state that is to be attained. This can be a goal, such as the goal to quit smoking or to avoid contaminating the palate with cheap wines, but this may also be a set of standards, such as rules of conduct (i.e., whenever possible, avoid drinking and teaching). The second component involves an awareness of one's actions, often referred to as *monitoring*. In cybernetic models of self-regulation (e.g., Carver & Scheier, 1981), monitoring involves comparing current behavior with the desired goal state and signaling any discrepancy. Monitoring is a particularly vulnerable component of self-regulation, as a failure to monitor ongoing behavior necessarily entails an inability to catch (and therefore control) unwanted actions. The final component is regulation itself. Upon identifying a thought or an emotion that conflicts with his or her goals, that person must be capable of implementing a strategy to inhibit or otherwise disarm the unwanted impulse. Limited capacity models of self-regulation (Baumeister & Heatherton, 1996) emphasize the ways in which this component operates like a muscle and is therefore subject to improvement through training and also to breakdowns through fatigue. Figure 36.1A depicts a model of self-regulation wherein monitoring, limited capacity resources, and goals interact with the strength of impulses and temptations, ultimately determining self-regulatory success or failure.

In the following sections we turn to some of the proposed mechanisms for how negative affect may impair self-control. As we shall see, negative affect appears to have the pernicious ability to operate on each of the aforementioned components of self-regulation. Acting like poison tendrils, it reaches into all aspects of self-control (Figure 36.1B), interfering with monitoring, exhausting the capacity to regulate behavior, and increasing the strength of desires and temptations.

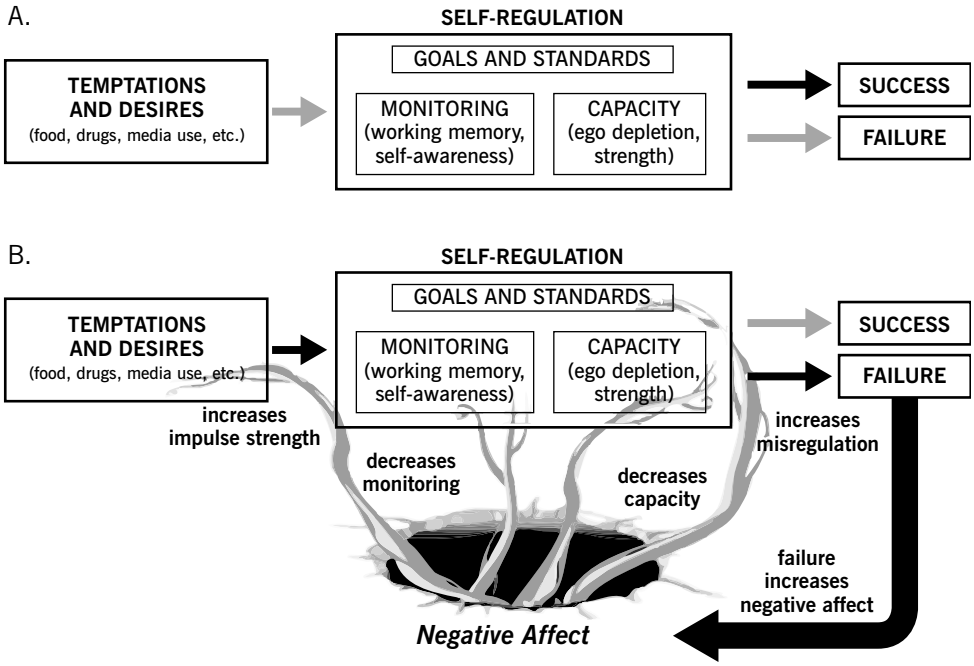

FIGURE 36.1. (A) Under normal circumstances, when a person is faced with temptations, successful self-regulation involves a balance between the strength of impulses and the capacity to monitor and regulate behavior in the service of goals and standards. (B) Negative affect spreads poison tendrils into every aspect of self-regulation, amplifying desires, decreasing monitoring, depleting limited capacity, and encouraging misregulation strategies (e.g., mood repair and escape from aversive self-awareness), which can relieve negative affect in the short term but often lead to further negative affect upon failure to meet one's goals.

How Might Negative Affect Impair Self-Control?

Negative Affect Increases the Strength of Temptations

In their dual-system model of self-control, Metcalf and Mischel (1999) theorize that negative affect may impair the cool system involved in executing control, while simultaneously increasing the hot system that responds to temptations and rewards. In support of this, studies of delay of gratification have shown that negative affect increases the frequency with which people accept immediate gratification instead of waiting for larger, delayed rewards (Mischel, Ebbesen, & Zeiss, 1973; Seeman & Schwarz, 1974; Tice, Bratslavsky, & Baumeister, 2001).

Interestingly, studies of nonhuman animals have revealed similar patterns in the form of increased reward sensitivity in distressed animals. For example, the induction of emotional distress via social isolation increases food and drug consumption (Campbell Teskey, Kavaliers, & Hirst, 1984; Ramsey & Van Ree, 1993). This heightened reward sensitivity is thought to be due to the release of glucocorticoids during distressing situations that serve to sensitize the brain's reward circuitry to appetitive stimuli (Deroche et al., 1995; Piazza & Le Moal, 1996). Although difficult to directly measure in humans, research has demonstrated that when people receive an artificial administration of glucocorticoids, they subsequently eat more relative to a placebo group (Tataranni et al., 1996).

In humans, negative affect has been shown to increase the intensity of smoking in smokers (McKee et al., 2011), as well as how much pleasure people report experiencing when smoking (Zinser, Baker, Sherman,

& Cannon, 1992). Results from a recent functional neuroimaging study of chronic dieters offer further evidence that negative affect may serve to increase the rewarding properties of appetitive stimuli—in this case of appetizing foods. Following a negative or neutral mood induction, chronic dieters were exposed to appetizing food cues during functional neuroimaging (Wagner, Boswell, Kelley, & Heatherton, 2012). Relative to nondieters, dysphoric dieters showed increased brain activity in the orbitofrontal cortex, a brain area implicated in representing the rewarding value of appetizing foods. Moreover, activity in the orbitofrontal cortex and ventral striatum was correlated with a measure of how distressing subjects found the negative mood induction, suggesting that increases in reward-related brain activity to food cues were dependent on the strength of the negative emotional state (Wagner et al., 2012). Taken together, these findings from both human and animal studies suggest that negative affect may sensitize people to rewards, thereby rendering temptations more difficult to regulate.

Negative Affect Reduces Monitoring through Cognitive Load

Another mechanism whereby negative affect may reduce self-control is by impairing the monitoring component of self-regulation. Specifically, research demonstrates that when dieters overeat following a negative mood induction, they are less aware than nondieters of the precise amount of food they have consumed (Heatherton, Polivy, Herman, & Baumeister, 1993). Reasons for this reduced awareness vary from a motivated desire to escape from negative affect (see the next section) to an increase in cognitive load as a result of ruminating over one's mood. In this section we focus on the possibility that experiencing negative affect can lead to increased working memory load as people ruminate over their negative mood and neglect to monitor their behavior (Figure 36.1B).

As monitoring entails the ability to maintain goals and standards in working memory (Hofmann, Schmeichel, & Baddeley, 2012), it follows that a concurrent working memory load (e.g., attempting to regulate mood or ruminating over one's performance anxieties) will impair task-relevant monitoring. Perhaps nowhere has this mechanism been better studied than in research on the phenomenon of stereotype threat. This work has shown that reminding women of negative stereotypes about their gender, or reminding African Americans of negative stereotypes about their race, can lead them to underperform on math or intelligence tests (Spencer, Steele, & Quinn, 1999; Steele & Aronson, 1995). Studies demonstrate that this decrement in performance is at least in part due to a reduction in working memory capacity, which is thought to result from concurrent attempts to regulate affect (Johns, Inzlicht, & Schmader, 2008). This phenomenon has been shown to "spill over" into nonstereotyped domains, as shown in work by Inzlicht and Kang (2010), who demonstrate that inducing stereotype threat in women results in disinhibited eating of unhealthy foods. Further evidence that working memory is impacted by negative affect comes from studies in which inducing emotional distress through the anticipation of an upcoming evaluation (e.g., public speaking) leads to poorer performance on working memory tests (Schoofs, Preuss, & Wolf, 2008). Indeed, one imaging study found that stereotype threat leads to increased activity in brain regions associated with affect (i.e., ventral anterior cingulate cortex) but not in areas that support working memory (Krendl, Richeson, Kelley, & Heatherton, 2008). In this case, the authors argued that it is likely that emotional distress resulting from stereotype threat has downstream effects on working memory.

Outside of the realm of negative affect, research on the effects of cognitive load on self-regulation have shown that cognitive loads impairs a variety of controlled behaviors, from suppressing thoughts (Wegner & Erber, 1992) to inhibiting food consumption in dieters (Ward & Mann, 2000). Mann and Ward (2007) proposed an attentional myopia theory of cognitive load, which posits that cognitive load restricts the range of attention to targets in the immediate environment, thereby rendering individuals vulnerable to a variety of external cues. To the extent that negative affect increases cognitive load through rumination or attempts to regulate affect (Johns et al., 2008), a similar attentional myopia may

occur when people are experiencing a negative emotional state.

Another means by which cognitive load can bring about self-regulation failure comes from research on ironic process theory (Wegner, 1994) demonstrating that when mental capacity is taxed by a concurrent cognitive load, the likelihood of counterintentional behavior increases. Within the realm of emotion regulation, it has been shown that attempting to change one's mood (either from happy to sad or vice versa) while under a concurrent cognitive load leads to an ironic increase in the opposite mood state. Specifically, participants who were instructed to make themselves feel sad, felt happier if they regulated their mood while under cognitive load (Wegner, Erber, & Zanakos, 1993). With respect to the role of affect in bringing about self-regulation failure, what this suggests is that to the degree that negative affect exerts a cognitive load, attempts to regulate behavior simultaneously, such as stopping oneself from smoking, may bring about the exact behavior one is trying to avoid.

Directing people's attention toward the self has a long history of being used as a means to increase monitoring, and thereby self-regulation. With regard to negative affect inductions, exposure to mirrors or to video clips of oneself has been shown to eliminate the effects of negative mood on disinhibited behavior. For example, having dieters watch a video of themselves after a mood induction eliminated the effects of negative affect on overeating as compared to dieters who watched a neutral video (Heatherton et al., 1993). Similarly, being forced to sit in front of a mirror has been shown to eliminate the detrimental effects of social rejection on a self-regulation task (Baumeister et al., 2005). It appears then, that increasing self-awareness tempers the impact of negative affect on self-regulation failures by restoring monitoring and increasing the accessibility of behavioral standards (e.g., Scheier & Carver 1983). However, the timing of increased self-attention may be important; for instance, increasing self-attention during the experience of emotion has been shown to heighten the intensity of emotions (Scheier & Carver, 1983; Fenigstein, 1979) which in turn may exacerbate the effects of negative affect on subsequent self-regulation.

Managing Negative Affect Depletes Self-Regulatory Strength

Over the last decade, considerable evidence has been gathered in favor of a strength model of self-regulation (for a recent meta-analysis, see Hagger, Wood, Stiff, & Chatzisarantis, 2010). This model posits that self-regulation relies on a common resource that can become temporarily exhausted by effortful self-regulation, and that when this resource is exhausted, further attempts at self-regulation are likely to fail (Baumeister & Heatherton, 1996). This phenomenon is typically studied using a sequential task paradigm in which an initial self-regulatory task is used to deplete resources, thereby rendering subsequent self-regulation attempts (even those in different domains) less likely to succeed. Studies have shown that tasks as varied as suppressing thoughts and stereotypes (Gordijn, Hindriks, Koomen, Dijksterhuis, & Van Knippenberg, 2004; Muraven, Collins, & Nienhaus, 2002), making decisions (Vohs et al., 2008), and managing impressions (Richeson & Shelton, 2003) all can lead to self-regulation failure on subsequent tasks. In terms of relevance to this chapter, it is interesting to note that one of the methods most often used for depleting self-regulatory resources is to have participants engage in an emotion regulation task (typically inhibiting emotional responses). For example, regulating emotional responses to an emotionally provocative film reduces persistence on difficult tasks (Baumeister, Bratslavsky, Muraven, & Tice, 1998), impairs performance on executive tasks (Schmeichel, Vohs, & Baumeister, 2003) and leads dieters to break their diets and overeat (Vohs & Heatherton, 2000). These findings strongly suggest that emotion regulation exhausts a common domain-general resource leading to impairments on subsequent self-control tasks.

As we have seen in the previous section, negative affect is often accompanied by concurrent attempts to regulate that affect. To the degree that this emotion regulation strategy is effortful, the strength mode of self-regulation suggests that, above and beyond the cost incurred to monitoring (see previous section), regulating negative affect would also deplete self-regulatory strength, increasing the likelihood of self-regulation failure. Indeed, in the previously mentioned

study by Vohs and Heatherton (2000) this is precisely what was found when dieters who were asked to inhibit their expression of sadness while watching an emotionally evocative video subsequently overindulged themselves in appetizing ice cream.

Although the bulk of the research on self-regulatory depletion and emotion examines how emotion regulation can produce subsequent self-regulation failure on nonemotional tasks, studies shows that this effect goes both ways, such that engaging in effortful self-regulation also impairs subsequent attempts at emotion regulation. The first researchers who looked at this question had participants engage in a thought suppression task, followed by an emotion regulation task that required participants to inhibit expressing emotions evoked by an emotional video (Muraven, Tice, & Baumeister, 1998). They found that participants whose resources had been depleted by the thought suppression task were subsequently less successful at inhibiting their emotions. In another study, Schmeichel (2007) extended these findings to more traditional tasks of executive function. In this experiment, participants completed a complex working memory task, followed by an emotion inhibition task. As in the Muraven et al. (1998) study, participants whose self-regulatory resources were depleted by the complex working memory task were impaired at suppressing their emotions relative to a nondepleted control group (Schmeichel, 2007).

More recently, the interplay between self-regulatory strength and emotional regulation was investigated using functional neuroimaging (Wagner & Heatherton, 2013). In this study, participants were assigned to either a depletion condition, in which they completed a difficult attention control task, or a control condition. Next, all participants viewed a series of emotional scenes differing in valence. Compared to control participants, those who were depleted exhibited greater neural activity in the amygdala, a region involved in the perception and detection of emotion, when viewing negative emotional scenes. Moreover, depleted participants exhibited reduced connectivity between the amygdala and prefrontal regions implicated in top-down control, suggesting a failure to engage in emotion regulation (Wagner & Heatherton, 2013).

Taken together, the results of the studies reviewed in this section offer both behavioral and neural evidence that engaging in self-regulation, be it in the emotional or the cognitive domain, leads to transient impairments in subsequent self-regulation attempts. Thus, with respect to our poison tendrils model of negative affect, ongoing attempts to regulate negative affect serve to reduce self-regulatory capacity, thereby increasing the likelihood of self-regulation failure (Figure 36.1B). Moreover, these findings suggest that it is important to consider the individual's prior self-regulatory context, as having to engage in other forms of self-regulation can lead to transient impairments of emotion regulation, which may exacerbate the effects of negative affect on self-regulation.

Depleting Self-Regulatory Strength Intensifies Emotions and Impulses

Although early models of self-regulation tacitly assumed that the strength of impulses and emotions remain essentially unchanged during self-regulation failure, recent theories suggest that impulses and emotions may increase in strength when self-control is depleted (e.g., Heatherton & Wagner, 2011; Schmeichel, Harmon-Jones, & Harmon-Jones, 2010; Vohs et al., submitted). For instance, the recent study by Vohs and colleagues demonstrated that when participants are in a depleted state, they experience stronger cravings for appetizing food, as well as rate emotions and pain as being felt more acutely than do nondepleted participants). The results of the brain imaging study by Wagner and Heatherton (2013) bear some similarities to these findings. Although the results of this study were interpreted in terms of emotion dysregulation following depletion, it is equally plausible that self-regulatory depletion served to amplify people's experience of affect, resulting in greater amygdala activity in response to negative scenes, thus making emotion regulation more difficult.

This relatively new line of research suggests that self-regulatory depletion not only harms subsequent attempts at self-control but also increases the strength of emotions and desires. For the current discussion this suggests that managing ongoing negative

affect may not only reduce self-regulatory capacity but also lead to a concurrent increase in the intensity of currently experienced affect, thereby amplifying its deleterious effects on all aspects of self-regulation, as indicated by the path between depletion and negative affect in Figure 36.1B.

Mood Repair and Escaping Negative Affect Take Precedence over Long-Term Goals

One likely explanation for why people turn to eating, smoking, and drinking in times of emotional distress is that they believe such activities can restore their mood (Sayette, 1993). Indeed, even in nonhuman animals, consuming pleasurable foods has been shown to reduce neuroendocrine markers of distress (Foster et al., 2009). In humans, research has shown that merely being exposed to appetizing stimuli (e.g., food, drugs, alcohol) can activate positive hedonic thoughts (Hofmann, van Koningsbruggen, Stroebe, Ramanathan, & Aarts, 2010; Sayette & Hufford, 1997). For instance, in dieters, exposure to descriptions of appetizing foods has been shown to spontaneously activate more hedonic thoughts about the pleasurable aspects of eating than descriptions of neutral foods (Papies, Stroebe, & Aarts, 2007). This attention to the pleasurable aspects of temptations can have serious consequences, especially in cases of alcohol and substance use in which withdrawal from the substance elicits negative affect that is relieved only by further substance use. It has been suggested that this need to cope with—and escape from—negative affect produced by withdrawal is one of the central motivations underlying addiction (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004). Moreover, through prolonged conditioning of substance use and improved mood, any experience of negative affect—whether due to specific triggers or merely the vicissitudes of daily life—can trigger the desire for drugs or alcohol (Baker et al., 2004; Childress et al., 1994).

Given people's belief that consuming food and alcohol or engaging in pleasurable activities will improve their mood, is there any empirical evidence that people employ this strategy when experiencing negative affect? Studies examining a wide range of behaviors show that, upon experiencing negative affect, people report consuming their favorite substance (e.g., food, alcohol, or drugs) or engaging in their favorite activity (e.g., shopping, television, gambling) precisely because they believe it will make them feel better (e.g., Faber & Christenson, 1996; Rook, 1987; Sayette, 1993). Thus, when stuck in a negative emotional state, people will shirk their long-term goals in order to address the more immediate need to feel better. One particularly disconcerting example of mood repair following negative affect comes from a study in which participants were asked to rate the personalities of ethnic ingroup and outgroup members. Participants who were induced to experience negative affect—as a result of negative performance feedback on an ostensibly unrelated test—subsequently rated the ethnic outgroup members more negatively than did control subjects (Fein & Spencer, 1997). Moreover, it was found that this tendency to denigrate outgroup members led to increased self-esteem in the negative affect group, suggesting that participants denigrated outgroup members in order to make themselves feel better. Likewise, when people experience social rejection, they become more willing to take illicit drugs or waste money on goods that are liked by their peers, but not necessarily by themselves, if doing so increases their chances at fitting in (Mead, Baumeister, Stillman, Rawn, & Vohs, 2011). Other examples involve the effect of negative affect on spending. In these studies, negative affect has been shown to increase participant's willingness to pay more for goods, as well as sell things they already own for less money (Lerner, Small, & Loewenstein, 2004). These findings were taken to indicate that people see buying new things or selling old things as a way of changing their current state and thereby escape negative affect.

Although attempting to repair one's mood through engaging in pleasurable activities such as eating may seem like a good temporary strategy to alleviate negative affect, studies show that this form of mood repair is often a case of *misregulation*. Unlike other forms of self-regulation failure, misregulation is not so much a lack of self-control as the use of self-control in a misguided—and often futile—attempt to improve one's state (Baumeister & Heatherton, 1996). For instance, studies show that although dieters may binge-eat in an attempt to improve their mood, they often end up feeling worse

after eating than they did before (for a meta-analysis, see Haedt-Matt & Keel, 2011).

Finally, a study by Tice and colleagues (2001) elegantly demonstrated that certain cases of disinhibited behavior are due entirely to a motivated attempt to improve affective states. In this study, participants were induced to experience negative affect, but in one group they were given a pill and told that it would freeze their mood. Whereas participants who did not receive the mood-freezing pill exhibited typical signs of self-regulation failure, such as eating unhealthy foods or procrastinating with pleasurable activity before a task, those who took the mood-freezing pill showed no signs of disinhibition (Tice et al., 2001). Thus, when participants believed engaging in pleasurable activities could not improve their negative emotional state, they ceased trying to change their mood by indulging in these activities.

The case of misregulation is particularly interesting, because it suggests that self-regulation failure following negative affect is not always due to a failure in the machinery of self-regulation; rather it reflects a conscious shift in priorities as people focus on improving their immediate affective state at the expense of their long-term regulatory goals. Indeed, it has been suggested that sometimes people actually use self-control to bring about personal harm, such as when they smoke or use drugs to fit in and make friends (Rawn & Vohs, 2011). However, these short-term strategies often lead to more negative affect as people move further and further away from their goals, such as when dieters overeat to improve their mood but then finds themselves further from their goal of weight loss (Heatherton & Polivy, 1992).

Intimately related to the notion of misregulation is research on escape from self-awareness (Baumeister, 1991; Heatherton & Baumeister, 1991). In contrast to the mood repair hypothesis outlined earlier, escape theory posits that self-awareness is aversive for people who possess negative self-views (see Higgins, 1987). Unfortunately, for those with negative self-views, increasing self-awareness does not help them regulate, because self-awareness itself can lead to negative affect as people's attention becomes focused on their perceived shortcomings (Mor & Winquist, 2002). For these people, escaping self-awareness becomes an effective strategy for reducing negative affect. However, attempts to reduce self-awareness come at the expense of the ability to focus on long-term goals and an increased vulnerability to temptations in the immediate environment. For example, dieters who experience a self-esteem threat are more likely than nondieters to overeat (Heatherton et al., 1993). Likewise, following self-esteem threats, people drink more alcohol (Hull & Young, 1983), watch more television (Moskalenko & Heine, 2003) and avoid sitting in front of mirrors (Twenge, Catanese, & Baumeister, 2003).

With respect to our model, this suggests that, in some instances, negative affect interferes with the machinery of self-regulation (e.g., monitoring or capacity), but in other circumstances, people chose to indulge in temptations as a means of repairing their mood. This form of misregulation may temporarily relieve negative affect, but once the awareness sets in that people have failed at their regulatory goals, further negative affect ensues, thus jeopardizing future attempts at self-regulation (see also Heatherton and Polivy's [1992] spiral model describing the relationship between negative affect and chronic dieting).

Are All Negative Emotions Equally Likely to Cause Self-Regulation Failure?

In many of the studies we have reviewed, *negative affect* refers primarily to any unpleasurable or aversive emotional state, thus subsuming a variety of emotional categories—from frustration to shame to social rejection. In some cases, negative affect is assessed through self-reports, whereas in others the emotional state is experimentally induced, affording more control over the specific emotions produced. That being said, differences between negative emotion types are seldom reported, with two exceptions. The first involves research in chronic dieters demonstrating that negative affect inductions that specifically target an individual's self-esteem (e.g., negative performance feedback, social rejection) are more effective at producing disinhibited eating (Heatherton et al., 1991, 1993, 1998; Lattimore & Maxwell, 2004) than induc-

tions targeting physical distress (e.g., fear of electric shock; Heatherton et al., 1991; Herman & Polivy, 1975).

The second exception involves research on the consequences of being socially excluded. As described earlier, social rejection can lead to poorer task performance, selfishness, aggression, and increased eating in dieters. Paradoxically, however, other studies indicate that inducing feelings of social rejection can increase affiliative behaviors (Maner, DeWall, Baumeister, & Schaller, 2007; Williams & Sommer, 1997), as well as improve memory for social information (Gardner, Pickett, & Brewer, 2000). One explanation for this discrepancy is that socially rejected people are adaptively deploying both protective and affiliative strategies, such that when the opportunity for forging new social bonds appears low (as in some experiments) they react with selfish and occasionally hostile behaviors, whereas when there is an opportunity for meaningful social contact, they instead switch to an affiliative strategy in an attempt to repair their self-esteem (e.g., Baumeister, Brewer, Tice, & Twenge, 2007; Maner et al., 2007; Smart Richman & Leary, 2009). Indeed, a recent imaging study found that interpersonal distress led to increased mental engagement for positive social stimuli (as indexed by activity in medial prefrontal cortical regions associated with mentalizing) and decreased engagement for negative social stimuli (Powers, Wagner, Norris, & Heatherton, 2013). Although these reactions to social rejection have only tangential bearing on self-regulation failure, they do highlight the complexity of the relationship between negative affect and self-regulation, demonstrating that reactions to negative affect can strategically vary according to the individual's goals. As described in the previous sections, what can appear to be a failure to maintain self-control may instead be a strategy to repair mood through consuming foods or alcohol, or engaging in pleasurable activities at the expense of other regulatory goals.

Can Negative Affect Ever Increase Self-Regulation Success?

Much of the work discussed here has examined the influence of affect on self-regulation in "hot" contexts, such as when faced with the opportunity to indulge in temptations or pleasurable activities. Emotion can, however, color behavior in other ways. For instance, in Schwarz's (1990) feelings as information model, an individual's current emotional state can be used as information in making evaluative judgments or resolving ambiguities. For example, following a negative mood induction, people tend to judge their current happiness and life satisfaction less positively than when in a positive mood (Schwarz & Clore, 1983). Other research shows that people in a negative mood tend to elaborate on information, demonstrating more accurate performance on tasks requiring attention to details (reviewed in Schwarz, 1990). Other theorists have suggested that emotions signal the need for self-regulation. For instance, Carver and Scheier (1990) argued that positive emotions signal that a person is achieving his or her goal (e.g., successfully dieting), whereas negative emotions arise when a person is moving away from his or her goal (e.g., gaining weight during a diet). From this perspective, then, negative affect should serve to increase rather than thwart efforts at self-regulation. The discrepancy between the preceding theories and the evidence reviewed in this chapter can be resolved if we consider when in time these effects are likely to take place. In this chapter we have primarily focused on the role of negative affect in eliciting self-regulation failure "in the moment," when people are confronted with the immediate need to regulate themselves, such as when faced with temptations. Over longer time frames, outside of these "hot" moments, negative affect may very well serve to motivate individuals to change their current state for the better (Heatherton & Polivy, 1992).

The Role of Positive Affect in Self-Regulation Success and Failure

In the preceding sections we have focused solely on the role of negative affect in self-regulation failure, with scant mention of its opposite, positive affect. This is largely for two reasons: First, researchers have often neglected positive affect in favor of negative affect (see Ashby, Isen, & Turken, 1999); second, of the research that does consider positive affect, there is less evidence that it

plays a strong part in self-regulation failures or successes, at least in the types of contexts discussed in this chapter. For example, compared to positive moods, negative moods and events are typically found to be more memorable and are experienced more intensely (see Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001). Moreover, in the mood induction studies reviewed earlier, of those that have directly compared negative and positive affect, negative always trump positive (Willner & Jones, 1996; Tiffanny & Drobes, 1990). Thus, it appears that negative affect is generally a much more potent force than positive affect in self-regulation failure.

Does positive affect promote self-regulation success? There is some research suggesting that positive affect can serve to momentarily increase self-regulation strength. For instance, when people hold the goal of self-improvement in mind, positive affect inductions lead to increased use of self-control (Fishbach & Labroo, 2007). Based on these results, Fishbach and Labroo suggest that positive affect pushes people to strive toward a currently activated goal. If that goal happens to be about self-improvement, then self-regulation may follow; however, if the goal is mood regulation, then self-regulation may suffer to the degree that it is opposed to maintaining a positive mood. Another study showing beneficial effect of positive affect on self-regulation found that inducing positive affect restored self-regulation capacity following self-regulatory depletion (Tice, Baumeister, Shmueli, & Muraven, 2007). This finding suggests that the capacity to engage in self-control is at least partially moderated by affect. In fact, to the degree that participants in the Tice et al. study hold the goal to succeed at the tasks laid out by the experimenters, then the results of Fishbach and Labroo (2007) would suggest that the positive affect induction reversed the effects of self-regulatory depletion by pushing subjects to expend whatever resources they had left in the service of the goal of succeeding at the task. It is worth pointing out that although these studies suggest that positive affect can increase self-control under certain circumstances, it is likely that under a different set of circumstances (e.g., at a bar), with a different goal in mind (e.g., celebrating with friends), positive affect may lead to greater celebratory excess.

Conclusion

Negative affect may very well be the most potent disinhibitor of restrained behavior. When people feel worthless, depressed, or rejected, they are more likely to engage in a variety of self-defeating behaviors. Following negative affect, dieters overeat, former smokers smoke, and alcoholics fall into relapse. More generally, people become more likely to procrastinate, to be selfish or hostile, and even go so far as to denigrate outgroup members. Negative affect appears to accomplish this pernicious feat by poisoning all facets of self-regulation (see Figure 36.1): sensitizing people to rewards and temptations, decreasing monitoring of behavior, reducing self-regulatory capacity, and causing people to focus on repairing their mood at the expense of abandoning their goals and giving in to their impulses and desires.

Although it often appears that our self-control is entirely at the mercy of our emotions, it is also the case that emotion regulation can be derailed by prior attempts at self-regulation in the cognitive domain. For instance, engaging in effortful self-regulation tasks can exhaust self-regulatory resources, thereby jeopardizing subsequent emotion regulation attempts. Moreover, recent research has shown that when people are cognitively depleted by prior attempts at self-regulation, emotions and impulses appear to increase in potency, rendering them a more forceful adversary to self-control.

The interplay among emotions, emotion regulation, and self-regulation has been discussed as though each of the myriad ways that emotions can hijack self-control work independently of the others. However, it is far more likely that the multiple routes to self-regulation failure are themselves interactive. For instance, negative affect can lead to a desire to escape negative states by reducing self-awareness and also to engage in pleasurable activities, even if these activities conflict with long-term goals. Together, these may serve to increase the pull of immediately available rewards, because the ability to monitor behavior is lessened as the focus shifts to pleasure. Negative affect may also place a load on working memory independent of the desire to escape self-awareness, thus further reducing the ability to monitor behavior. Throw in the fact that prior self-regulatory effort may leave the individual

in a depleted state in which both resources for further self-control are lacking and the strength of impulses and temptations is increased, and it is a small miracle that people are not constantly acting out their fantasies, drinking, smoking, or indulging every gastronomic desire. If the studies reviewed in this chapter paint a dire picture, it is important to note that emotion is not all bad: For instance, it has been demonstrated that experiencing positive emotions can improve self-regulation by helping to buffer against the depleting effects of prior tasks (Tice et al., 2007).

In this chapter we reviewed evidence suggesting that negative emotions are a potent cause of self-regulation failure and we have proposed a simple poison tendrils model in which negative affect invades and disrupts nearly every aspect of self-regulatory function. Moreover, results from a variety of behavioral and neural studies suggest that the relationship between emotions and self-regulation is dynamic and interactive, such that emotion-induced self-regulation failure serves not only to intensify currently experienced emotions and desires but also to increase negative affect as the individual moves further and further away from his or her goals. Negative affect is thus a particularly potent threat to self-regulation, because it not only reduces the capacity for control (increased working memory load, reduced self-awareness and monitoring) but it may also lead to increases in the strength of experienced desires and emotions, rendering them all the more difficult to resist.

References

Ashby, F. G., Isen, A. M., & Turken, A. U. (1999). A neuropsychological theory of positive affect and its influence on cognition. *Psychological Review, 106*(3), 529–550.

Baker, T. B., Piper, M. E., McCarthy, D. E., Majeskie, M. R., & Fiore, M. C. (2004). Addiction motivation reformulated: An affective processing model of negative reinforcement. *Psychological Review, 111*(1), 33–51.

Baucom, D. H., & Aiken, P. A. (1981). Effect of depressed mood in eating among obese and nonobese dieting and nondieting persons. *Journal of Personality and Social Psychology, 41*(3), 577–585.

Baumeister, R. F. (1991). *Escaping the self: Alcoholism, spirituality, masochism, and other flights from the burden of selfhood*. New York: Basic Books.

Baumeister, R. F. (1997). Esteem threat, self-regulatory breakdown, and emotional distress as factors in self-defeating behavior. *Review of General Psychology, 1*(2), 145–174.

Baumeister, R. F., Bratslavsky, E., Finkenauer, C., & Vohs, K. D. (2001). Bad is stronger than good. *Review of General Psychology, 5*(4), 323–370.

Baumeister, R. F., Bratslavsky, E., Muraven, M., & Tice, D. M. (1998). Ego depletion: Is the active self a limited resource? *Journal of Personality and Social Psychology, 74*(5), 1252–1265.

Baumeister, R. F., Brewer, L. E., Tice, D. M., & Twenge, J. M. (2007). Thwarting the need to belong: Understanding the interpersonal and inner effects of social exclusion. *Social and Personality Psychology Compass, 1*(1), 506–520.

Baumeister, R. F., DeWall, C. N., Ciarocco, N. J., & Twenge, J. M. (2005). Social exclusion impairs self-regulation. *Journal of Personality and Social Psychology, 88*(4), 589–604.

Baumeister, R. F., & Heatherton, T. (1996). Self-regulation failure: An overview. *Psychological Inquiry, 7*(1), 1–15.

Berkowitz, L. (1989). Frustration–aggression hypothesis: Examination and reformulation. *Psychological Bulletin, 106*(1), 59–73.

Bousman, C. A., Cherner, M., Ake, C., Letendre, S., Atkinson, J. H., Patterson, T. L., et al. (2009). Negative mood and sexual behavior among non-monogamous men who have sex with men in the context of methamphetamine and HIV. *Journal of Affective Disorders, 119*(1–3), 84–91.

Campbell Teskey, G., Kavaliers, M., & Hirst, M. (1984). Social conflict activates opioid analgesic and ingestive behaviors in male mice. *Life Sciences, 35*(3), 303–315.

Carver, C. S., & Scheier, M. (1981). *Attention and self-regulation: A control-theory approach to human behavior.* New York: Springer-Verlag.

Carver, C. S., & Scheier, M. F. (1990). Origins and functions of positive and negative affect: A control-process view. *Psychological Review, 97*(1), 19–35.

Childress, A. R., Ehrman, R., McLellan, A. T., MacRae, J., Natale, M., & O'Brien, C. P. (1994). Can induced moods trigger drug-related responses in opiate abuse patients?

Journal of Substance Abuse Treatment, 11(1), 17–23.

Deroche, V., Marinelli, M., Maccari, S., Le Moal, M., Simon, H., & Piazza, P. V. (1995). Stress-induced sensitization and glucocorticoids: I. Sensitization of dopamine-dependent locomotor effects of amphetamine and morphine depends on stress-induced corticosterone secretion. *Journal of Neuroscience, 15*(11), 7181–7188.

DeWall, C. N., Twenge, J. M., Bushman, B., Im, C., & Williams, K. (2010). A little acceptance goes a long way applying social impact theory to the rejection–aggression link. *Social Psychological and Personality Science, 1*(2), 168–174.

Faber, R. J., & Christenson, G. A. (1996). In the mood to buy: Differences in the mood states experienced by compulsive buyers and other consumers. *Psychology and Marketing, 13*(8), 803–819.

Fein, S., & Spencer, S. J. (1997). Prejudice as self-image maintenance: Affirming the self through derogating others. *Journal of Personality and Social Psychology, 73*(1), 31–44.

Fenigstein, A. (1979). Self-consciousness, self-attention, and social interaction. *Journal of Personality and Social Psychology, 37*(1), 75–86.

Fishbach, A., & Labroo, A. A. (2007). Be better or be merry: How mood affects self-control. *Journal of Personality and Social Psychology, 93*(2), 158–173.

Foster, M. T., Warne, J. P., Ginsberg, A. B., Horneman, H. F., Pecoraro, N. C., Akana, S. F., et al. (2009). Palatable foods, stress, and energy stores sculpt corticotropin-releasing factor, adrenocorticotropin, and corticosterone concentrations after restraint. *Endocrinology, 150*(5), 2325–2333.

Fox, H. C., Bergquist, K. L., Hong, K., & Sinha, R. (2007). Stress-induced and alcohol cue-induced craving in recently abstinent alcohol-dependent individuals. *Alcoholism: Clinical and Experimental Research, 31*(3), 395–403.

Gardner, W. L., Pickett, C. L., & Brewer, M. B. (2000). Social exclusion and selective memory: How the need to belong influences memory for social events. *Personality and Social Psychology Bulletin, 26*(4), 486–496.

Gordijn, E. H., Hindriks, I., Koomen, W., Dijksterhuis, A., & Van Knippenberg, A. (2004). Consequences of stereotype suppression and internal suppression motivation: A self-regulation approach. *Personality and Social Psychology Bulletin, 30*(2), 212–224.

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39*(3), 281–291.

Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge eating: A meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin, 137*(4), 660–681.

Hagger, M. S., Wood, C., Stiff, C., & Chatzisarantis, N. L. D. (2010). Ego depletion and the strength model of self-control: A meta-analysis. *Psychological Bulletin, 136*(4), 495–525.

Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. *Psychological Bulletin, 110*(1), 86–108.

Heatherton, T. F., Herman, C. P., & Polivy, J. (1991). Effects of physical threat and ego threat on eating behavior. *Journal of Personality and Social Psychology, 60*(1), 138–143.

Heatherton, T. F., & Polivy, J. (1992). Chronic dieting and eating disorders: A spiral model. In J. H. Crowther, D. L. Tennenbaum, S. E. Hobfoll, & M. A. P. Stephens (Eds.), *The etiology of bulimia nervosa: The individual and familial context* (pp. 133–155). Washington, DC: Hemisphere.

Heatherton, T. F., Polivy, J., Herman, C. P., & Baumeister, R. F. (1993). Self-awareness, task failure, and disinhibition: How attentional focus affects eating. *Journal of Personality, 61*(1), 49–61.

Heatherton, T. F., Striepe, M., & Wittenberg, L. (1998). Emotional distress and disinhibited eating: The role of self. *Personality and Social Psychology Bulletin, 24*(3), 301–313.

Heatherton, T. F., & Wagner, D. D. (2011). Cognitive neuroscience of self-regulation failure. *Trends in Cognitive Sciences, 15*(3), 132–139.

Herman, C. P., & Polivy, J. (1975). Anxiety, restraint, and eating behavior. *Journal of Abnormal Psychology, 84*(6), 666–672.

Higgins, E. T. (1987). Self-discrepancy: A theory relating self and affect. *Psychological Review, 94*(3), 319–340.

Higgins, R. L., & Marlatt, G. A. (1975). Fear of interpersonal evaluation as a determinant of alcohol consumption in male social drinkers. *Journal of Abnormal Psychology, 84*(6), 644–651.

Hofmann, W., Schmeichel, B. J., & Baddeley, A. D. (2012). Executive functions and self-regulation. *Trends in Cognitive Sciences, 16*(3), 174–180

Hofmann, W., van Koningsbruggen, G. M.,

Stroebe, W., Ramanathan, S., & Aarts, H. (2010). As pleasure unfolds: Hedonic responses to tempting food. *Psychological Science, 21*(12), 1863–1870.

Hull, J. G., & Young, R. D. (1983). Self-consciousness, self-esteem, and success-failure as determinants of alcohol consumption in male social drinkers. *Journal of Personality and Social Psychology, 44*(6), 1097–1109.

Inzlicht, M., & Kang, S. K. (2010). Stereotype threat spillover: How coping with threats to social identity affects aggression, eating, decision making, and attention. *Journal of Personality and Social Psychology, 99*(3), 467–481.

Johns, M., Inzlicht, M., & Schmader, T. (2008). Stereotype threat and executive resource depletion: Examining the influence of emotion regulation. *Journal of Experimental Psychology: General, 137*(4), 691–705.

Kassel, J. D., Stroud, L. R., & Paronis, C. A. (2003). Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychological Bulletin, 129*(2), 270–304.

Keltner, D., & Gross, J. J. (1999). Functional accounts of emotions. *Cognition and Emotion, 13*(5), 467–480.

Krendl, A. C., Richeson, J. A., Kelley, W. M., & Heatherton, T. F. (2008). The negative consequences of threat: A functional magnetic resonance imaging investigation of the neural mechanisms underlying women's underperformance in math. *Psychological Science, 19*(2), 168–175.

LaRose, R., Lin, C., & Eastin, M. (2003). Unregulated Internet usage: Addiction, habit, or deficient self-regulation? *Media Psychology, 5*(3), 225–253.

Lattimore, P., & Maxwell, L. (2004). Cognitive load, stress, and disinhibited eating. *Eating Behaviors, 5*(4), 315–324.

Leary, M. R., Kowalski, R. M., Smith, L., & Phillips, S. (2003). Teasing, rejection, and violence: Case studies of the school shootings. *Aggressive Behavior, 29*(3), 202–214.

Lerner, J. S., Small, D. A., & Loewenstein, G. (2004). Heart strings and purse strings: Carryover effects of emotions on economic decisions. *Psychological Science, 15*(5), 337–341.

Levenson, R. W. (1994). Human emotion: A functional view. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion: Fundamental questions* (pp. 123–126). New York: Oxford University Press.

Maner, J. K., DeWall, C. N., Baumeister, R. F., & Schaller, M. (2007). Does social exclusion motivate interpersonal reconnection?: Resolving the "porcupine problem." *Journal of Personality and Social Psychology, 92*(1), 42–55.

Mann, T., & Ward, A. (2007). Attention, self-control, and health behaviors. *Current Directions in Psychological Science, 16*(5), 280–283.

Marlatt, G. A., & Gordon, J. (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors*. New York: Guilford Press.

McKee, S. A., Sinha, R., Weinberger, A. H., Sofuoglu, M., Harrison, E. L., Lavery, M., et al. (2011). Stress decreases the ability to resist smoking and potentiates smoking intensity and reward. *Journal of Psychopharmacology, 25*(4), 490–502.

Mead, N. L., Baumeister, R. F., Stillman, T. F., Rawn, C. D., & Vohs, K. D. (2011). Social exclusion causes people to spend and consume strategically in the service of affiliation. *Journal of Consumer Research, 37*(5), 902–919.

Metcalfe, J., & Mischel, W. (1999). A hot/cool-system analysis of delay of gratification: Dynamics of willpower. *Psychological Review, 106*(1), 3–19.

Mischel, W., Ebbesen, E. B., & Zeiss, A. R. (1973). Selective attention to the self: Situational and dispositional determinants. *Journal of Personality and Social Psychology, 27*(1), 129–142.

Mor, N., & Winquist, J. (2002). Self-focused attention and negative affect: A meta-analysis. *Psychological Bulletin, 128*(4), 638–662.

Moskalenko, S., & Heine, S. J. (2003). Watching your troubles away: Television viewing as a stimulus for subjective self-awareness. *Personality and Social Psychology Bulletin, 29*(1), 76–85.

Muraven, M., Collins, R. L., & Nienhaus, K. (2002). Self-control and alcohol restraint: An initial application of the self-control strength model. *Psychology of Addictive Behaviors, 16*(2), 113–120.

Muraven, M., Tice, D. M., & Baumeister, R. F. (1998). Self-control as limited resource: Regulatory depletion patterns. *Journal of Personality and Social Psychology, 74*(3), 774–789.

Oaten, M., Williams, K. D., Jones, A., & Zadro, L. (2008). The effects of ostracism on self-regulation in the socially anxious. *Journal of Social and Clinical Psychology, 27*(5), 471–504.

Papies, E., Stroebe, W., & Aarts, H. (2007). Pleasure in the mind: Restrained eating and spon-

taneous hedonic thoughts about food. *Journal of Experimental Social Psychology, 43*(5), 810–817.

Piazza, P. V., & Le Moal, M. L. (1996). Pathophysiological basis of vulnerability to drug abuse: Role of an interaction between stress, glucocorticoids, and dopaminergic neurons. *Annual Review of Pharmacology and Toxicology, 36*, 359–378.

Pithers, W. D., Kashima, K. M., Cumming, G. F., Beal, L. S., & Buell, M. M. (1988). Relapse prevention of sexual aggression. *Annals of the New York Academy of Sciences, 528*(1), 244–260.

Powers, K. E., Wagner, D. D., Norris, C. J., & Heatherton, T. F. (2013). Socially excluded individuals fail to recruit medial prefrontal cortex for negative social scenes. *Social Cognitive and Affective Neuroscience, 8*(2), 151–157.

Ramsey, N. F., & Van Ree, J. M. (1993). Emotional but not physical stress enhances intravenous cocaine self-administration in drug-naive rats. *Brain Research, 608*(2), 216–222.

Raviv, M. (1993). Personality characteristics of sexual addicts and pathological gamblers. *Journal of Gambling Studies, 9*(1), 17–30.

Rawn, C. D., & Vohs, K. D. (2011). People use self-control to risk personal harm: An intra-interpersonal dilemma. *Personality and Social Psychology Review, 15*(3), 267–289.

Richards, J. M., & Gross, J. J. (2000). Emotion regulation and memory: The cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology, 79*(3), 410–424.

Richeson, J. A., & Shelton, J. N. (2003). When prejudice does not pay: Effects of interracial contact on executive function. *Psychological Science, 14*(3), 287–290.

Roberts, M. E., Gibbons, F. X., Gerrard, M., Weng, C.-Y., Murry, V. M., Simons, L. G., et al. (2012). From racial discrimination to risky sex: Prospective relations involving peers and parents. *Developmental Psychology, 48*(1), 89–102.

Rook, D. W. (1987). The buying impulse. *Journal of Consumer Research, 14*(2), 189–199.

Sayette, M. A. (1993). An appraisal-disruption model of alcohol's effects on stress responses in social drinkers. *Psychological Bulletin, 114*(3), 459–476.

Sayette, M. A., & Hufford, M. R. (1997). Effects of smoking urge on generation of smoking-related information. *Journal of Applied Social Psychology, 27*(16), 1395–1405.

Scheier, M. F., & Carver, C. S. (1983). Self-directed attention and the comparison of self with standards. *Journal of Experimental Social Psychology, 19*(3), 205–222.

Schmeichel, B. J. (2007). Attention control, memory updating, and emotion regulation temporarily reduce the capacity for executive control. *Journal of Experimental Psychology: General, 136*(2), 241–255.

Schmeichel, B. J., Harmon-Jones, C., & Harmon-Jones, E. (2010). Exercising self-control increases approach motivation. *Journal of Personality and Social Psychology, 99*(1), 162–173.

Schmeichel, B. J., Vohs, K. D., & Baumeister, R. F. (2003). Intellectual performance and ego depletion: Role of the self in logical reasoning and other information processing. *Journal of Personality and Social Psychology, 85*(1), 33–46.

Schoofs, D., Preuss, D., & Wolf, O. T. (2008). Psychosocial stress induces working memory impairments in an *n*-back paradigm. *Psychoneuroendocrinology, 33*(5), 643–653.

Schwarz, N. (1990). Feelings as information: Informational and motivational functions of affective states. In E. T. Higgins & R. M. Sorrentino (Eds.), *Handbook of motivation and cognition: Foundations of social behavior* (Vol. 2, pp. 527–561). New York: Guilford Press.

Schwarz, N., & Clore, G. L. (1983). Mood, misattribution, and judgments of well-being: Informative and directive functions of affective states. *Journal of Personality and Social Psychology, 45*(3), 513–523.

Seeman, G., & Schwarz, J. C. (1974). Affective state and preference for immediate versus delayed reward. *Journal of Research in Personality, 7*(4), 384–394.

Sinha, R. (2007). The role of stress in addiction relapse. *Current Psychiatry Reports, 9*(5), 388–395.

Smart Richman, L., & Leary, M. R. (2009). Reactions to discrimination, stigmatization, ostracism, and other forms of interpersonal rejection: A multimotive model. *Psychological Review, 116*(2), 365–383.

Spencer, S. J., Steele, C. M., & Quinn, D. M. (1999). Stereotype Threat and Women's Math Performance. *Journal of Experimental Social Psychology, 35*(1), 4–28.

Steele, C. M., & Aronson, J. (1995). Stereotype threat and the intellectual test performance of African Americans. *Journal of Personality and Social Psychology, 69*(5), 797–811.

Tataranni, P. A., Larson, D. E., Snitker, S., Young, J. B., Flatt, J. P., & Ravussin, E. (1996). Effects of glucocorticoids on energy metabolism and food intake in humans. *American Journal of Physiology: Endocrinology and Metabolism, 271*(2), E317–E325.

Tice, D. M., Baumeister, R. F., Shmueli, D., & Muraven, M. (2007). Restoring the self: Positive affect helps improve self-regulation following ego depletion. *Journal of Experimental Social Psychology, 43*(3), 379–384.

Tice, D. M., Bratslavsky, E., & Baumeister, R. F. (2001). Emotional distress regulation takes precedence over impulse control: If you feel bad, do it! *Journal of Personality and Social Psychology, 80*(1), 53–67.

Tiffany, S. T., & Drobes, D. J. (1990). Imagery and smoking urges: The manipulation of affective content. *Addictive Behaviors, 15*(6), 531–539.

Twenge, J. M., Baumeister, R. F., DeWall, C. N., Ciarocco, N. J., & Bartels, J. M. (2007). Social exclusion decreases prosocial behavior. *Journal of Personality and Social Psychology, 92*(1), 56–66.

Twenge, J. M., Baumeister, R. F., Tice, D. M., & Stucke, T. S. (2001). If you can't join them, beat them: Effects of social exclusion on aggressive behavior. *Journal of Personality and Social Psychology, 81*(6), 1058–1069.

Twenge, J. M., Catanese, K. R., & Baumeister, R. F. (2003). Social exclusion and the deconstructed state: Time perception, meaninglessness, lethargy, lack of emotion, and self-awareness. *Journal of Personality and Social Psychology, 85*(3), 409–423.

Velten, E. (1968). A laboratory task for induction of mood states. *Behaviour Research and Therapy, 6*(4), 473–482.

Vohs, K. D., Baumeister, R. F., Mead, N. L., Hofmann, W. J., Ramanathan, S., & Schmeichel, B. J. (submitted). *Engaging in self-control heightens urges and feelings.*

Vohs, K. D., Baumeister, R. F., Schmeichel, B. J., Twenge, J. M., Nelson, N. M., & Tice, D. M. (2008). Making choices impairs subsequent self-control: A limited-resource account of decision making, self-regulation, and active initiative. *Journal of Personality and Social Psychology, 94*(5), 883–898.

Vohs, K. D., & Heatherton, T. F. (2000). Self-regulatory failure: A resource-depletion approach. *Psychological Science, 11*(3), 249–254.

Wagner, D. D., Boswell, R. G., Kelley, W. M., & Heatherton, T. F. (2012). Inducing negative affect increases the reward value of appetizing foods in dieters. *Journal of Cognitive Neuroscience, 24*(7), 1625–1633.

Wagner, D. D., & Heatherton, T. F. (2013). Self-regulatory depletion increases emotional reactivity in the amygdala. *Social Cognitive and Affective Neuroscience, 8*(4), 410–417.

Warburton, W. A., Williams, K. D., & Cairns, D. R. (2006). When ostracism leads to aggression: The moderating effects of control deprivation. *Journal of Experimental Social Psychology, 42*(2), 213–220.

Ward, A., & Mann, T. (2000). Don't mind if I do: Disinhibited eating under cognitive load. *Journal of Personality and Social Psychology, 78*(4), 753–763.

Wegner, D. M. (1994). Ironic processes of mental control. *Psychological Review, 101*(1), 34–52.

Wegner, D. M., & Erber, R. (1992). The hyperaccessibility of suppressed thoughts. *Journal of Personality and Social Psychology, 63*(6), 903–912.

Wegner, D. M., Erber, R., & Zanakos, S. (1993). Ironic processes in the mental control of mood and mood-related thought. *Journal of Personality and Social Psychology, 65*(6), 1093–1104.

Williams, K. D., & Sommer, K. L. (1997). Social ostracism by coworkers: Does rejection lead to loafing or compensation? *Personality and Social Psychology Bulletin, 23*(7), 693–706.

Willner, P., & Jones, C. (1996). Effects of mood manipulation on subjective and behavioural measures of cigarette craving. *Behavioural Pharmacology, 7*(4), 355–363.

Witkiewitz, K., & Villarroel, N. A. (2009). Dynamic association between negative affect and alcohol lapses following alcohol treatment. *Journal of Consulting and Clinical Psychology, 77*(4), 633–644.

Zinser, M. C., Baker, T. B., Sherman, J. E., & Cannon, D. S. (1992). Relation between self-reported affect and drug urges and cravings in continuing and withdrawing smokers. *Journal of Abnormal Psychology, 101*(4), 617–629.

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