

Series in Anxiety and Related Disorders

Michael W. Otto  
Stefan G. Hofmann  
*Editors*

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# Avoiding Treatment Failures in the Anxiety Disorders

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 Springer

# Series in Anxiety and Related Disorders

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# Avoiding Treatment Failures in the Anxiety Disorders

 Springer

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ISBN 978-1-4419-0611-3                      e-ISBN 978-1-4419-0612-0  
DOI 10.1007/978-1-4419-0612-0  
Springer New York Dordrecht Heidelberg London

Library of Congress Control Number: 2009938520

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*MWO: To my son, Jackson, for being who you are.*

*SGH: To Lukas, Benjamin, and Rosemary, for making it easy to resolve life's complications.*

# Preface

Over the last two decades, the field has witnessed dramatic advances in the conceptualization and treatment of anxiety disorders. Conditions, such as panic disorder, that were once considered the domain of pharmacotherapy have now been shown to be responsive to even ultra brief treatment protocols of cognitive-behavioral therapy (CBT). Indeed, the success of CBT across the anxiety disorders, and across the diverse patients afflicted by these conditions, inspires confidence in both the principles of CBT and the models of the disorders on which they are based. Yet, in the face of these clear advances, and the status of CBT as a first-line intervention for all of the anxiety disorders, clinicians continue to be challenged by the persistence of limited or poor treatment response among some patients. This volume is devoted to addressing these challenges.

Authors of the chapters herein were responsible for integrating the latest in empirical research on the nature and treatment of anxiety disorders with lessons learned from actively working with patients in specialty clinic settings. The result is a scholarly based, yet clinically applicable, discussion of issues of treatment non-response and the strategies used to address these clinical challenges. Interventions that define empirically supported CBT for anxiety disorders are described in detail and are complemented by novel clinical strategies and the variations in treatment delivery that expert clinicians have applied with success. Accordingly, this volume is designed to be of use to clinicians seeking to understand more about how CBT is applied in the clinic as well as experienced clinicians seeking to find new strategies to apply when standard interventions fail. This volume is also designed to provide a state-of-the-art account of what is known and is not known about psychosocial treatment outcome for the anxiety disorders, thereby providing clinical researchers with the latest evidence on principles of treatment, core intervention strategies, predictors of nonresponse, and compensatory or novel interventions. By identifying treatment complications and discussing strategies for resolving them, it is our hope that this text will contribute to the further advancement of CBT for anxiety disorders.

Boston, Massachusetts

Michael W. Otto  
Stefan G. Hofmann

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**Part I**  
**General Aspects of Treatment**  
**Complications**

# Introduction

**Stefan G. Hofmann and Michael W. Otto**

Anxiety disorders are prevalent and disabling mental disorders. Although cognitive-behavioral therapy (CBT) is clearly effective, there is still room for improvement. In this volume, the editors recruited a number of noted clinical researchers with extensive experience to contribute chapters covering the state-of-the-art CBT for anxiety disorders, treatment complications encountered when applying these interventions in clinical practice, and strategies for overcoming treatment failures when they are encountered.

By definition, anxiety disorders are associated with significant distress or interference with the patient's life and are associated with significant role disability. In addition, anxiety disorders impose significant direct and indirect costs on the nation in terms of medical resources as well as in reduced or lost productivity. In fact, it is estimated that anxiety disorders are associated with greater economic costs than any other mental disorder (DuPont, DuPont, & Rice, 2002).

Cognitive-behavioral therapy (CBT) is a gold standard treatment for the anxiety disorders. Based on empirically supported models of the disorder, CBT provides a tailored set of interventions – often emphasizing exposure combined with cognitive restructuring but also including other cognitive- and skill-based techniques (e.g., relaxation training or assertiveness training) – designed to modify the self-perpetuating cycles that characterize the anxiety disorders.

CBT is clearly effective for anxiety disorders and offers direct benefits in terms of symptom reduction, reduction in role disability, and enhancement of quality of life, as demonstrated in numerous clinical trials. For example, a recent meta-analytic review of randomized placebo-controlled CBT trials showed a mean effect size of 0.73 of CBT over psychosocial or pill placebo comparison conditions (Hofmann & Smits, 2008). This body of work demonstrates that CBT strategies for anxiety disorders are more effective than the nonspecific effects of credible placebo interventions, thus refuting the often-held belief that all psychotherapies are of equal effectiveness with common (nonspecific) factors constituting the primary mechanism of action.

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However, it is also clear that there is still considerable room for improvement to help more patients achieve beneficial outcomes.

This book is designed to provide clinicians with detailed information on (1) state-of-the-art CBT for anxiety disorders, (2) treatment complications encountered when applying these interventions in clinical practice, and (3) strategies for overcoming treatment failures when they are encountered. This is achieved by bringing together noted clinical researchers who have extensive experience with CBT in the context of clinical practice, clinical supervision, and clinical trials. The chapters herein combine scholarly review of the literature with clinical experience and provides readers an empirically supported perspective that is grounded in experience with providing targeted care to individuals in need. As such, the text provides guidance useful for clinical practice as well as clinical research.

The book is separated into three parts. Part I reviews the general aspects of treatment complications. In order to facilitate the identification of treatment challenges, Teachman and Clerkin provide a case formulation approach in Chapter 2. Powers, Vervliet, Smits, and Otto provide a learning theory perspective on treatment resistance and relapse in Chapter 3, and Halperin, Weitzman, and Otto discuss therapeutic alliance and common treatment factors in Chapter 4. Issues of combined CBT and pharmacologic treatment are targeted by Chapter 5 by Smits, Reese, Powers, and Otto. The contribution of cultural issues to treatment complications is examined by Spendlove, Jackson, and Borrego in the final chapter of Part I.

Part II examines disorder-specific strategies for avoiding treatment failures. Readers are provided with core strategies linked to case examples and accompanied by strategies to apply should treatment nonresponse be encountered. This section opens with a focus on the treatment of panic disorder (Chapter 7 by Murray, McHugh, and Otto), and is followed by chapters focused on the treatment of obsessive-compulsive disorder (Chapter 8 by Marques, Chosak, Phan, Fama, Franklin, and Wilhelm), post-traumatic stress disorder (Chapter 9 by Zayfert and DeViva), social anxiety disorder (Chapter 10 by Hofmann, Richey, Asnaani, and Sawyer), generalized anxiety disorder (Chapter 11 by Behar and Borkovec), and specific phobias (Chapter 12 by Alpers).

A special feature of the book is Part III on special populations, which covers the common problems of anxiety disorders that are comorbid with depression (Chapter 13 by Deveney and Otto), personality disorders (Chapter 14 by Nock, Deliberto, and Hollander), substance use disorders (Chapter 15 by Reynolds, Tull, Shalev, and Lejuez), eating disorders (Chapter 16 by Becker, Zayfert, and Pratt), medical conditions (Chapter 17 by Greer, Graham, and Safren), and treatment complications specific to children and adolescents (Chapter 18 by Micco and Ehrenreich).

Our hope is that this text will provide the CBT practitioner with useful clinical guidance to overcoming obstacles and impasses when treating some of the most debilitating and costly mental health problems.

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# A Case Formulation Approach to Resolve Treatment Complications

Bethany A. Teachman and Elise M. Clerkin

There is a tendency to think of case formulation as an activity that occurs at the outset of therapy to guide initial clinical decision-making, but which plays little role once therapy is underway. However, we believe that case formulation is most useful when viewed as a dynamic, iterative process that invites frequent revisiting of hypotheses as new client data become available. As Eells describes in her influential handbook, “A psychotherapy case formulation is a hypothesis about the causes, precipitants, and maintaining influences of a person’s psychological, interpersonal, and behavioral problems. A case formulation helps organize information about a person, particularly when that information contains contradictions or inconsistencies in behavior, emotion, and thought content.” (Eells, 2007, p. 4). By viewing case formulation as an animate hypothesis-testing enterprise, the process becomes very useful for resolving treatment complications. In particular, it helps with identifying potential “stuck points” by generating alternative approaches and possible explanations for treatment stagnation.

In the current chapter, we consider some of the many ways that case formulations can help enhance treatment outcome for anxiety disorders. We will focus predominantly on case formulation from a cognitive behavioral perspective because this approach reflects the dominant treatment perspective for anxiety disorders (see list of empirically supported treatments; e.g., Chambless & Ollendick, 2001). However, we also consider recommendations from alternate therapeutic orientations. In particular, underlying the proposals we offer is a perspective borrowed from Motivational Interviewing (Miller & Rollnick, 2002), which suggests that apparent “resistance” in therapy is better understood as ambivalence about making changes. Moreover, ambivalence is expected when we ask clients to give up well-established (albeit maladaptive) ways of thinking, behaving, and relating to others. Thus, complications in treatment present puzzles for therapists and clients to investigate, rather than purposeful defiance on the client’s part. Case formulation is a valuable tool that can help put the pieces of the puzzle (back) together when a treatment is floundering.

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We will outline the seven steps advocated by Persons and Tompkins (2007) for clinicians to follow to develop an effective cognitive behavioral therapy (CBT) case formulation. These include (1) creating a problem list, (2) assigning DSM diagnoses, (3) selecting a primary diagnosis, (4) applying an empirically supported, nomothetic formulation, (5) integrating individual client characteristics, (6) hypothesizing about mechanisms maintaining the disorder, and (7) considering antecedents for the current onset of illness. Our goal in outlining these steps is not to espouse one “correct” way for devising a case formulation. Rather, we use these steps as a springboard to evaluate the multiple ways that case formulation can help identify problems and potential solutions to aid in treatment planning and implementation. We will close the chapter by discussing the use of different modalities of case formulation and alternative treatment strategies to resolve typical complications that arise in therapy.

## **Using Cognitive Behavioral Case Formulation to Resolve Treatment Complications**

Just as there is not a single cognitive behavioral treatment for anxiety disorders, there is not a single CBT case formulation. Those working from a primarily cognitive orientation are likely to form hypotheses about a client’s maladaptive beliefs that contribute to the development and maintenance of their disorder. For instance, clinicians providing cognitive therapy for obsessive compulsive disorder (OCD) will emphasize a client’s unhealthy interpretations about the meaning of their intrusive thoughts (see Frost & Steketee, 2002; Rachman, 1998). Analogously, a therapist working from cognitive models of social phobia will highlight the onset of fears of negative evaluation and beliefs that one will fall short of an expected standard (see Clark & Wells, 1995; Rapee & Heimberg, 1997). On the other hand, when working primarily from a behavioral perspective, maladaptive behaviors and environmental contingencies, such as reinforcements for avoidance, will likely feature prominently in case formulation.

Notwithstanding the many varieties of possible *content* in CBT case formulations, Persons and Tompkins (2007) outline a series of seven *steps* that are common to developing effective formulations. As we outline, each of these seven steps provide opportunity for the clinician to re-evaluate a case that is not proceeding smoothly.

### ***Problem List***

It is critical to compile a comprehensive biopsychosocial problem list that characterizes the range of different problem areas in a client’s life. Although it is unlikely that all of the different problems will be targeted in treatment, having this list (and updating it throughout treatment) will help ensure that a feasible approach to treatment is

selected, and can help both the client and the therapist anticipate barriers to change. In addition to assessing the difficulties associated with the presenting clinical problems, Woody, Detweiler-Bedell, Teachman, and O'Hearn (2002) recommend evaluating the following domains as a start to creating the problem list: Injurious Behavior (e.g., suicidal ideation or actions), School/Occupational Functioning (e.g., job or school stability, financial status), Family Functioning (e.g., relationships with key family members, parenting skills), Other Interpersonal Functioning (e.g., frequency and quality of social supports), Behavioral Health (e.g., medical history and physical fitness), Risky Behaviors (e.g., alcohol abuse, legal difficulties), and Culture, Spirituality, and Moral Development (e.g., involvement with religious institutions, level of acculturation). In addition, evaluation of motivational factors, such as readiness for change, can be useful for anticipating therapy-interfering behaviors.

It is often tempting to skip doing a full assessment of functioning across different life domains when treating an anxiety disorder because there are clear and effective treatments available for most anxiety diagnoses; thus, the full problem list may seem superfluous if therapists feel they already know what type of treatment they will provide. However, not being aware of the broader picture of a client's functioning can easily sabotage a treatment. For instance, it is common for well-intentioned therapists to suggest all manner of creative exposures that turn out not to be viable because of financial problems (attending movies for clients with agoraphobia and taking long excursions for persons with driving phobias are common examples of impractical exposures for clients with limited means). Similarly, identifying challenges related to diversity issues is critical, whether tied to cultural background and difficulties related to acculturation or concerns stemming from prejudice tied to physical challenges, religion, ethnicity, social status, or sexual orientation (see Hays, 1995). By referring to the problem list and recognizing how these other life challenges may impact implementation of standard CBT strategies, homework adherence can often be greatly enhanced.

Using the problem list component of case formulation is also very useful for recognizing complications in treatment that follow from difficult interpersonal relationships. We see this frequently in the context of OCD and generalized anxiety disorder (GAD) where couples may develop an unhealthy dyad with one individual repeatedly requesting reassurance from their partner. Changing this reassurance-seeking ritual is often important for success in treatment, but without some knowledge about the relationship dynamics (including those with a partner, parent, teacher, friend, or other "support" person), interpersonally oriented interventions can easily backfire.

As a general guideline, when treatment is not proceeding as planned, especially when adherence to treatment recommendations is low, returning to the problem list and considering other problems that may be operating as barriers to strategy implementation is an easy and often fruitful first step to resolving the impasse. Additionally, it is important to check if new problems have arisen since the last evaluation or if any major domains were not evaluated. This list can also help clinicians determine if one reason for a stalled treatment is that the wrong problems were prioritized. One of the challenges when working with complicated clients who

have high levels of comorbidity and related impairments in functioning is making an educated guess about a good place to start when forming an initial treatment plan. The problem list and its role in case formulation are helpful in this regard by highlighting how one problem area may fuel another, and in turn, how change in one area may alleviate difficulties in another.

### ***Five-Axis DSM Diagnoses***

Once the problem list has been compiled, DSM diagnoses are assigned along the five axes (Axis I: clinical disorders, including major mental disorders; Axis II: pervasive or personality conditions; Axis III: medical conditions; Axis IV: psychosocial and environmental factors; Axis V: Global Assessment of Functioning). Often it is not feasible to complete a full, structured Axis II personality disorder evaluation. In this case, it can still be beneficial to note difficult personality dimensions that may interfere with treatment because they may be helpful when returning to the case formulation to generate hypotheses about why treatment may be stalled. Common situations where this arises in anxiety treatment concern clients with an overly dependent personality (who may then want the therapist to directly give advice and make decisions for them), or clients with an avoidant personality style (who have few social relationships and, in some cases, limited social skills). Recognizing that change in personality disorders tends to occur more slowly than change in Axis I problems is important.

Similarly, even though a complete medical evaluation for Axis III rarely coincides with a mental health intake evaluation, asking about medical problems is critical for treatment planning. For instance, we frequently learn about medical conditions, such as asthma or neck pain, that influence the type of interoceptive exposures (exercises that bring on physical sensations relevant to anxiety and panic) we recommend for individuals with panic attacks. This is also the time where clients will often discuss upcoming medical interventions that may interfere with treatment attendance.

Psychosocial and environmental difficulties outlined on Axis IV, such as unemployment, and the more general assessment of functioning on Axis V are useful for setting realistic goals for different stages of therapy. It is not unusual for clients (and new therapists) to feel discouraged about progress in therapy because they had unreasonable expectations about the extent and speed of recovery related to the focal disorder (e.g., that a client with OCD would become free of obsessions; that symptoms of panic would never again come out of the blue). Clients sometimes imagine that change in one area of functioning will miraculously solve all other problems. Expectations for a reasonable pace of change are essential for keeping both the client and therapist motivated. Having some idea about other areas of limited functioning will help determine the resources the person has available to aid with the hard work necessary for progress in treatment. Along these lines, it is helpful to get an evaluation of premorbid functioning (i.e., a sense of a client's skills and lifestyle before onset of the disorder).

## ***Primary Diagnosis***

Persons and Tompkins (2007) advocate that the therapist should select a primary or “anchoring” diagnosis at this point in CBT case formulation. Frequently this is determined by the diagnosis that causes the most distress for the individual or contributes to the most difficulties on the problem list. This is ideally a collaborative decision so that the client and therapist are working toward similar goals in therapy. The selection of the primary diagnosis is used to guide which nomothetic template will be used as the basis for the individual formulation.

Challenges with this step of the process that can contribute to later treatment complications include: (1) disagreement between the therapist and client about the appropriate diagnosis to prioritize, (2) selection of a diagnosis that is not primary or sufficiently important to the individual’s overall functioning, or (3) other comorbid diagnoses interfering with progress on the selected diagnosis. When problems emerge in therapy where the therapist feels like the therapy is repeatedly being pulled off-track because the client regularly wants to work on issues unrelated to the selected focus, it is worth revisiting the case formulation to consider whether one of the above difficulties with selecting the primary diagnosis has occurred.

In the case of complications due to disagreement about the appropriate diagnostic focus, we recommend a careful functional analysis for the client and therapist to evaluate how the different problems are related. For instance, a client may wish to focus on panic attacks even though they occur very infrequently and seem secondary (according to the therapist’s perspective) to social anxiety that is limiting social interactions on a daily basis. In this case, the client and therapist might consider how avoidance due to fear of future panic versus avoidance due to fear of negative evaluation from others is most impairing. Did one versus the other lead to missed opportunities at work, or to more conflict with a spouse? When problems are highly connected, as in this example, it is often difficult to tease apart which anxiety problem is contributing most to the overall impairment (e.g., a promotion at work might be passed up because it would involve public speaking where the individual feared having a panic attack). Nevertheless, in the area of anxiety disorders (unlike other problem areas, such as substance abuse), we find that it is usually not very difficult for the client and therapist to come to agreement about an order in which they will tackle different problems so long as the decision is made collaboratively. Deciding on a phase approach to therapy (see Woody et al., 2002), where problems will be broken down and then undertaken in sequence can make this decision process easier. Clearly, some agreement on the treatment plan is needed if “collaborative empiricism” and willingness to try exposures (in a non-coercive environment) is to succeed.

At times, the therapist and client readily agree on an anchoring diagnosis, but they make a poor choice so treatment does not produce the expected gains. This can frequently occur when issues of differential diagnosis are challenging. For instance, in the panic disorder versus social phobia example above, it is not unusual to assume that the presence of panic attacks warrants panic disorder treatment. Frequent panic attacks are without question incredibly distressing, but these can often occur as part

of the presentation of another anxiety disorder, which is in fact primary. Individuals with specific and social phobias, for example, sometimes have panic attacks (or fear they will) when encountering their feared situation. Yet, while there are important overlapping components in the treatments for phobias and for panic disorder, there are also critical differences (in required exposures, catastrophic cognitions, etc.) that may need a targeted treatment. There are numerous challenging differential diagnoses in the context of anxiety disorders: body dysmorphic disorder symptoms can overlap with social phobia, depression symptoms can overlap with GAD, rituals associated with eating disorders can look similar to OCD, to name just a few. When a treatment is not progressing well, considering whether the right treatment targets have been selected is an important step in the problem-solving process, and reconsidering the links between the selected diagnosis and the problem list is a good place to look for solutions.

Re-evaluating the selected primary diagnosis is also useful when a client has other disorders comorbid with the anxiety diagnosis. This is the norm rather than the exception, but the field is still at early stages in terms of research to guide how to select treatment goals with complex clients who have multiple diagnoses. One obvious issue to consider is whether the presence of an additional diagnosis is possibly interfering with the anxiety treatment. This occurs frequently in the context of substance abuse, eating disorders (especially in anorexia nervosa when starvation greatly impairs cognitive processing), psychotic disorders, and severe mood disorders (where lethargy and retardation, unmanaged manic symptoms, or suicidal ideation can all interfere with the ability to engage in treatment). In these cases, it may be necessary to focus on the interfering diagnosis first before returning to treatment for the anxiety problem.

### ***Nomothetic Formulation***

Selecting the anchoring diagnosis provides a useful focal point so that the therapist can then turn to the research literature to determine whether a group-level, nomothetic formulation exists for that diagnosis. While many disorders do not yet have such formulations, there are a number of choices that have a strong empirical foundation for anxiety disorders. This does not mean there will be a readily available formulation to exactly match your particular client; for instance, the field sorely needs formulations for anxiety disorders that recognize cultural differences. These include adaptations of standard CBT formulations that take into account unique treatment needs related to age, race and ethnicity, religious affiliation, sexual orientation, etc. Notwithstanding, there are many useful sources to draw upon even when no exact match exists – imagine, for example, that you want to develop a formulation for a client who is 79 years old and has social phobia as the primary diagnosis. There are multiple CBT formulations for social phobia, including the highly influential models by Clark and Wells (1995) and by Rapee and Heimberg (1997). However, these are not geared toward conceptualizing elderly clients, so if this is likely to be central to the case formulation, seeking out a further nomothetic template that includes

an aging focus would be helpful. While we are unaware of such a template specific to social phobia, valuable models exist for anxiety problems in general among elderly persons (see Beck & Stanley, 1997; McCarthy, Katz, & Foa, 1991; Sheikh & Salzman, 1995, among others) and for some specific anxiety disorders, such as GAD (see Ayers, Sorrell, Thorp, & Wetherell, 2007), which can then be modified to reflect social anxiety concerns.

The purpose of selecting the nomothetic formulation is that it serves as the template for developing hypotheses about the psychological mechanisms that are maintaining the disorder, which can then be targeted in treatment. In the case of social phobia, the nomothetic formulation would likely lead to hypotheses about how fears of negative evaluation result in maladaptive avoidance of social situations (the avoidance relieves anxiety in the short-term, but worsens it in the long-term). In panic disorder with agoraphobia, the formulation might highlight how catastrophic misinterpretations of benign bodily sensations and one's ability to tolerate anxiety contribute to a "fear-of-fear" cycle and avoidance of situations where panic sensations might occur (e.g., drinking caffeine). In OCD, a cognitive formulation would emphasize the misinterpretation of unwanted thoughts as being meaningful or personally significant, while a more behavioral formulation would focus on the impact of doing rituals to reduce the anxiety caused by obsessions. As evident from these examples, the nomothetic formulation is particular to the disorder, but is generic in the sense that it is thought to apply to most individuals who have the disorder, regardless of whether OCD involves hand-washing or checking locks, or whether social phobia involves public speaking or dating fears. It is in the next step that the formulation is adapted to the individual client.

The nomothetic formulation incorporates a number of valuable components for helping resolve treatment complications. In particular, it brings a wealth of empirical support for a given conceptualization and subsequent treatment approach. Using the research literature to understand what approaches have worked well – and equally importantly, worked poorly – saves the clinician an amazing amount of time and trial and error. Notwithstanding, it is difficult to anticipate how an approach that works well for people *on average* will work for a given *individual*, particularly when a case is complex and the client may not match the clinical population used in the research studies on some critical variable (e.g., pattern of comorbidity, ethnic background). To date, there is not sufficient data to guide therapists in deciding when to individually tailor a treatment versus when to adhere closely to an empirically supported treatment (see discussions in Persons & Tompkins, 2007; Schulte, Kunzel, Pepping, & Schulte-Bahrenberg, 1992). We recommend trying the empirically supported approach first but monitoring progress regularly so that a treatment that is not moving ahead is recognized quickly (see Woody et al., 2002). In this way, an unhelpful intervention will not continue unchecked and a change of course can be considered (see discussion of alternate treatment options at the end of this chapter). Thus, individual client-level data should be collected throughout therapy regardless of whether an empirically supported or individually tailored plan is implemented. In this sense, all formulations and treatment plans can be evidence-based.

Perhaps the most useful lesson from this step of case formulation with regards to addressing a stagnant or deteriorating therapy is the focus on empiricism. It is well known that clinicians, like all humans, are vulnerable to biases that distort our memory and interpretations so that we tend to “see what we want to see” (see Garb, 1997; Fiske & Taylor, 1984; Lopez, 1989). This is why including objective measures of progress is so critical. Being an empiricist can occur both at the level of selecting a formulation with compelling research support and at the level of collecting data from the individual client. In turn, these data are often the key to identifying the barriers that are blocking advances in treatment.

### ***Individualize the Formulation***

The next step is to take the nomothetic formulation and apply it to a particular individual, taking into account his or her particular problem list and the posited relationships among the problems. As noted, cognitive behavioral formulations for anxiety tend to be diagnosis-based in that there is a model to guide the conceptualization and treatment of a given disorder. While this model has enormous utility and predictive validity, treatment complications can readily ensue if formulation stops at the level of the diagnosis. Knowing that a person meets criteria for post traumatic stress disorder (PTSD), social phobia, or GAD allows one to make an educated guess about a first line of treatment to try (based on the research literature), but it does not explain how the model is a fit or mismatch for that specific person. As Wilson (1998) noted, “Empirically-supported, manual-based treatments are good, but not good enough” (p. 367).

One does not have to be in practice for long before encountering seeming mismatches. There is the client with OCD who reports no awareness of obsessions tied to his compulsions, or who does not see the compulsions as anxiety-reducing. There is the individual with panic disorder who denies experiencing any catastrophic (mis)interpretations of her bodily sensations. Even when the disorder-based model does readily fit, it details a process but does not explain why or how that particular person came to make those catastrophic misinterpretations, experience those obsessions, etc.

Case formulation can help translate the group-level treatment approach to a given client, and this translation can guide critical decision points in treatment when a standard strategy does not appear to be working. One important place to start is to consider how the various difficulties on the problem list might be inter-related. For instance, a recent client seeking treatment at our clinic for GAD was receiving a standard worry control treatment protocol, but progress was incredibly slow. At first, this was puzzling to the therapist because the client, Steve, a 27-year-old recently married man of Korean descent, appeared highly motivated and committed to the therapy. He was early for each session and not only completed his homework, but had assembled a color-coded binder in which he kept his session notes. Moreover, he had inquired about and purchased self-help books to use as adjuncts to his weekly therapy session. It would be hard to imagine a more “perfect” client.

Not surprisingly (in retrospect!), this turned out to be the very problem that was interfering with the client making progress.

When the therapist returned to the problem list, she was reminded of the client's perfectionistic thinking and how this had caused considerable impairment in Steve's job and even in his marriage, because he was so easily discouraged by small setbacks. The clinician had initially missed how this same pattern was playing out in therapy because it had been so pleasurable to work with the client who was clearly trying to please the therapist. Once the clinician recognized the relationship between Steve's perfectionism and his response to treatment, she was able to talk to Steve about his tendency to try to implement the techniques he was learning so rigidly that he felt like a failure whenever he experienced quite minor stumbling blocks (e.g., a single day with increased worry). This was creating a vicious cycle, whereby Steve would experience more worry and then try even more rigidly to follow the program. He became increasingly sensitive to his perceived failings and worried excessively about "screwing up treatment." The therapist suggested Steve put away his binder of session notes and self-help books for a while, and actually do less typical therapy homework for a few weeks. He was encouraged to focus on enhancing the quality of his life versus working on treatment assignments so rigidly. Ironically, by stepping back and reducing his focus on the usual worry control assignments, Steve was able to make far more progress. Of course, Steve was still doing homework for the therapy – the focus of the homework had simply shifted from closely monitoring anxiety and explicitly re-evaluating negative automatic thinking to considering what Steve truly valued in his life and focusing on promoting those goals.

As this case illustrates, the individual's intra- and interpersonal circumstances – and how these interact with the focal diagnosis and its treatment – need to be considered to understand how to apply the nomothetic formulation to help a particular person. This does not mean that clinicians should abandon the evidence-based treatment plan at the first sign of a treatment complication. Instead, it suggests consideration of how the relations among a client's strengths and weaknesses can lead to more effective application of an intervention. Further, the decision to advocate for *less* standard CBT homework (e.g., keeping thought records) in order to challenge perfectionist beliefs highlights the importance of focusing on the principle behind a given strategy, as opposed to rigidly following a script when adapting a formulation to a particular individual.

### ***Hypotheses About the Basis of Mechanisms Maintaining the Disorder***

Once the nomothetic formulation has been adapted for the specific individual, the next stage is to develop hypotheses about the origins of the mechanisms that are thought to maintain the disorder. This involves evaluating the client's social and family history (both in terms of family psychiatric history and information about the client's upbringing). Relevant information for a person with social phobia might include early life experiences with parents and teachers that contributed to fears of

being evaluated negatively by authority figures. In GAD, one might inquire about the development of beliefs that the world is an unpredictable and dangerous place, or that the client is somehow vulnerable and unable to cope. For someone with OCD, were there religious teachings that emphasized the importance of purity of thought? More generally, when did the individual start avoiding situations that made him anxious, and in what ways was this avoidance behavior reinforced?

Ideally, one would generate multiple hypotheses about the etiology of the maladaptive ways of thinking, behaving, and relating to others. Considering more than one hypothesis is important down the line for helping the therapist and client see more than one avenue for intervention. The goal of generating ideas about how the problem developed is not to figure out the “cause” of a disorder – this is rarely definable – but to assist with treatment planning. These hypotheses can help the client develop some kind of narrative about the onset of their problems. In turn, this narrative can make the treatment approach more comprehensible and credible, in part by helping clients appreciate the need to identify and then alter maladaptive patterns that maintain the disorder.

For example, Lily, a 43-year-old woman with PTSD (following a rape that occurred when she was in her 20s), recognized that she was trained at a young age to avoid confrontation at all costs. As a result, she developed a pattern of avoiding all interactions that might elicit negative affect, especially anger (either in herself or in others). Upon considering this explanation for the development of her avoidance behavior, and consequent maintenance of her PTSD, Lily was far more willing to consider prolonged exposures in treatment. At the same time, the client discussed her mother’s constant warnings as she was growing up about the need to be vigilant around men because “they were only after one thing.” Although Lily had enjoyed dating in her teens and early 20s, following the rape she became extremely distrustful of all men who were not family, even ending close male friendships that had been quite supportive. Again, once these early warnings and ensuing beliefs were recognized, the client was able to engage in cognitive restructuring to re-evaluate her over-general conclusions about the dangerousness of men.

This step of the case formulation is often helpful when treatment is stuck because the generation of multiple hypotheses about how the disorder developed (or mechanisms maintaining it) can point to a variety of potential targets for intervention. This is not to say that the solution for a disorder has to be rectifying some factor that contributed to its development; after all, aspirin can help a headache, but the absence of aspirin was not the cause of the headache. However, identifying factors that contributed to the development of a disorder can be motivating for clients when they recognize that the disorder was not predetermined. If they learned dysfunctional ways of thinking or behaving, they also have the ability to learn more adaptive approaches. Analogously, this stage of hypothesis generation can help clients see their role in the onset of a problem; this is not done to assign blame but to empower clients by helping them see that they have choices in how they respond to the events in their lives.

For instance, clients with social phobia will often talk about being teased in childhood, and feel this contributed to the development of their fears. While this

hypothesis is certainly reasonable, it is likely incomplete. Most people experience teasing in childhood to some degree, so the individual is challenged to consider why *their* fears of negative evaluation grew and persisted. This may lead to hypotheses about a parent who was overly critical and emphasized the need to “put on a good face”, which in turn contributed to the development of a core belief of inadequacy. By recognizing that others might not have accepted the teasing as valid (i.e., as a sign of their inadequacy), the client then has a choice to consider other ways of responding to criticism.

This step of the case formulation can be a powerful one when treatment is going poorly because it can help the client better understand why their problems developed, and show them that they can now work towards a different way of responding to the world.

### ***Precipitants of Illness***

The final step in CBT case formulation involves considering possible precipitants for the current period of illness. As with the previous step, the goal is not to figure out the cause of the illness, but to recognize possible triggers and activating situations so clients can learn how to minimize these situations in the future. When antecedents are recognized, clients also learn that seemingly unpredictable anxiety reactions can often be understood more fully (and seem more predictable and controllable). Further, this step can be helpful when a treatment feels stuck because it allows the client and therapist to consider whether the same triggers are still in place and may explain the difficulty in breaking old patterns and alleviating symptoms. For instance, discovering that increased fighting in the marriage preceded a surge in panic attacks can allow the client to work on changing his interpersonal environment (e.g., consider couple’s counseling to reduce the marital conflict) while simultaneously using the conflict trigger as an opportunity for exposures to elicit panic symptoms.

Each of these seven steps in CBT case formulation can play a valuable role in resolving treatment complications. In particular, as noted by Eells (2007), the formulation can help make sense of seeming inconsistencies in a client’s presentation across thoughts, feelings, and behaviors. To illustrate how the formulation can be used, we introduce a case example that highlights a common inconsistency; the discrepancy between a client’s stated treatment goals and her behavior. The “yes, but” refrain in response to one treatment recommendation after another can be frustrating for even the most experienced clinician. By returning to (and potentially reconsidering) the posited origins, mechanisms, and precipitants of the anxiety problem, it often becomes clear why an apparent discrepancy is occurring. Once a possible explanation has been identified, paths to resolve the impasse are much easier to identify.

*Case example.* Kelly was a 51-year-old mother of two college-aged daughters who contacted our clinic after seeing an advertisement for one of our research studies, which provided free treatment for panic disorder. Upon calling, she reported that

she had a 10-year history of occasional panic attacks but that they had significantly worsened following September 11, 2001. She felt a strong sense of loss of control following 9/11, and was terrified that her college-aged daughters, who no longer lived in their home town, might come to harm. Despite this terror, she rarely spoke of her concerns, wanting to maintain her reputation among her friends as a “woman who had it together.” Her panic attacks typically occurred in the evenings and were triggered by small signs of gastrointestinal distress, which escalated into fears that she might lose bladder control and make a fool of herself.

The initial case formulation focused on the standard nomothetic one for panic attacks following Clark’s (1986) model – that she was catastrophically misinterpreting changes in bodily sensations, resulting in a rapidly worsening fear-of-fear cycle. At the idiographic level, the fears tied to 9/11 were emphasized as a precipitant for the worsening of symptoms, and her fears about losing control were thought to be critical maintaining factors. At the outset of the treatment, Kelly appeared highly engaged, asking questions during the initial psychoeducation component and commenting frequently that she was so glad to be “helping with a research study” because one of her daughters was working in a research lab at college.

However, when it came time to start doing exposures to elicit panic sensations, Kelly regularly had reasons why a given exposure was not likely to help her. For instance, she initially denied any avoidance behaviors, but subsequent probing revealed that she would not drink caffeine because of the jittery sensations and occasional upset stomach it brought on, and would not eat a full meal after 6:00 pm because of concerns that she would have indigestion. When Kelly was encouraged to consider trying these activities, she suggested that caffeine was not good for you and eating large meals at night was unhealthy because her metabolism worked more slowly then. All the while, she kept reiterating that she was very happy to be in treatment because she believed research was important, and was glad to be contributing. After numerous circular discussions and unsuccessful brainstorming about alternate exposure options, the therapist felt stuck. Kelly regularly said she was happy to be in the study and even agreed that exposures were likely very helpful for reducing panic, but rejected all suggestions for personally tailored exposures.

At this stage, the therapist revisited the case formulation and hypothesized that the critical link that she had been missing was the common denominator that tied together the client’s secrecy about her fears amongst her friends (in order to appear “together”) and her insistence that she was participating in treatment to help with the research study. The therapist tried focusing less on fears of losing control tied to bodily changes or harm coming to others (e.g., the fears related to 9/11), and reconceptualized the problem around the client’s difficulties admitting to a weakness. Just as Kelly had not wanted others to know of her fears about her family or about her panic attacks, she also did not want to think of herself as someone who needed therapy. Focusing on the research component of her treatment participation was interfering with her engaging in the therapy as someone who actually needed help. With this new hypothesis, the therapist was then able to understand the client’s seeming resistance and use cognitive restructuring techniques to help the

client re-evaluate her beliefs about the unacceptability of needing help. With this revised case formulation, the client eventually recognized that she was participating to get help for panic as well as to help with research, and became more willing to try exposures.

The following dialog illustrates the therapist and client working together to try to connect the seemingly discrepant components of her presentation:

Therapist: I am wondering how to make sense of the different things you're telling me. On the one hand, you've repeatedly said that you want to be in the group and that you agree that exposures are likely helpful for panic. On the other hand, it feels like whenever we suggest trying an exposure, you have a reason why it won't work for you.

Kelly: I just really want to support the research you're doing, and I've told my daughter all about the study.

Therapist: That's great that you've shared your experience with your daughter and that she has been supportive. I've noticed that you keep referring to our work here as a research study, rather than as therapy. While it's certainly true that we're conducting research, the goal is also to provide you with treatment for your panic disorder. What do you think it would be like to tell your daughter that you are in therapy, instead of in a research study?

Kelly: Then she would think something was wrong with me.

Therapist: I see how that could be difficult, but why would it be bad for her to find out that you needed some help right now?

Kelly: I'm the Mom.

Therapist: And what does it mean for you to be the Mom?

Kelly: It means I should be the one who is always in control.

Therapist: That's quite a tough standard to meet. Is that also the standard you set for yourself with your friends? You told me that you try not to share your problems with them either, and that they don't know about your fears for your family or about your panic attacks.

Kelly: I guess so, although it's not something I really think much about. I suppose I've just always prided myself on being the one who everyone else can count on. It scares me to be the one who has a problem – I've always been the one who fixed other people's problems. Who will they turn to if I'm all screwed up?

Therapist: I understand that acknowledging your own problems can be very scary. I wonder, though, whether having a problem like panic attacks really means that you're "all screwed up" and can no longer help other people. . .

As this case illustrates, revisiting the formulation to look for clues about seeming inconsistencies – in this case, the discrepancy between the client's apparent engagement in treatment yet unwillingness to try exposures – can highlight patterns and likely explanations for treatment barriers that might not be apparent without the formulation's guideposts.

## Using Different Modalities of Case Formulation to Resolve Treatment Complications

The above discussion has focused on CBT formulation, but case formulation can take many forms. In some traditions, formulation is done primarily at the outset of therapy (e.g., interpersonal psychotherapy where a focal problem area is selected early on; Markowitz & Swartz, 2007), while for other orientations the formulation occurs later in treatment (e.g., emotion-focused therapy where critical information is not thought to emerge without experience in the “safe context of the therapeutic environment”; Greenberg & Goldman, 2007). Regardless, in all instances the formulation is assumed to be a dynamic hypothesis. As we have outlined, in the case of anxiety disorders, a disorder-specific nomothetic formulation usually serves as the starting point. While this formulation is determined at the outset of therapy as soon as a primary diagnosis is selected, the refining of this formulation to include client-specific data should occur and recur throughout treatment. Moreover, using a DSM disorder-based, nomothetic formulation is not the only approach. For example, the substantial rates of comorbidity across mood and anxiety problems have led to recent advances that focus on treating “negative affect syndrome,” rather than isolating single disorders (see Barlow, Allen, & Choate, 2004). Others have advocated focusing on more general, underlying emotional processing difficulties that emerge during therapy, such as fear of abandonment, rather than emphasizing specific disorders at the outset of treatment (e.g., Greenberg & Goldman, 2007).

Another important distinction across different case formulation traditions includes adherence to a categorical versus dimensional model. The categorical approach follows the so-called medical model, which views disorders as “discrete pathological entities” with predictable causes and prognoses (Eells, 2007, p. 9). In contrast, the dimensional approach focuses less on classification, instead viewing psychopathology on a continuum from normal to pathological. CBT formulations for anxiety typically follow a categorical approach, emphasizing a particular disorder to understand a person’s problems. This approach is very useful for selecting a treatment plan that corresponds with the categorical decision, but can reify the category (treating an abstract concept, like a disorder, as though it were a concrete entity) and can leave out important non-categorical influences on the origin and course of the disorder. For instance, intra- and interpersonal dimensions, such as openness to experience and ability to form alliances with others, do not fit into neat categories (until you reach the Axis II personality disorders, and even then the categorical nature of these disorders is controversial; Widiger, 1992), but undoubtedly influence treatment outcome (see Martin, Garske, & Davis, 2000).

Related to the focus on dimensions versus categories, case formulation approaches also vary in their emphasis on a person’s weaknesses versus strengths and on change versus acceptance (e.g., cognitive-behavioral and interpersonal formulations focus more on fixing problems and skill deficits, relative to more humanistic approaches, such as an emotion-focused therapy formulation). While CBT approaches make some reference to the value of acceptance, this is far more explicit in other approaches, such as case formulation in dialectical behavior therapy

(DBT; see Koerner, 2007). This focus on working to accept some distress and negative circumstances can be extremely valuable with anxiety-disordered clients, particularly when perfectionist tendencies lead to overly rigid applications of change strategies (as the earlier case example with Steve's GAD treatment illustrated). The recent emphasis on mindfulness strategies (see later section) attests to these potential benefits. In fact, many of the dialectics outlined in DBT case formulation can be extremely useful for generating hypotheses to understand treatment complications in anxiety disorders. For example, recognizing both sides of the emotional vulnerability dialectic – inability or unwillingness to regulate emotions during emotional extremes versus attempts to deny or ignore vulnerability – can help make sense of clients' inconsistencies in their readiness to confront avoidance behaviors in anxiety. Similarly, being aware of the active–passive dialectic, where the individual oscillates between passive, acting incompetent behaviors versus trying to appear overly together, can aid in catching over- or under-predictions of fear that can interfere with successful learning during exposures.

Thus, while we advocate starting with a cognitive and/or behavioral case formulation for most anxiety problems because of the extensive research base supporting these treatment modalities, we also note the importance of drawing upon other case formulation approaches when clients are not making the expected progress. Analogously, when starting with an alternate treatment approach (e.g., a recent clinical trial suggests non-CBT approaches, such as psychodynamic therapy, may also be efficacious in the treatment of panic disorders; Milrod et al., 2007), the clinician is advised to consider CBT or other case formulation steps to revise a floundering treatment.

## **Using Case Formulation to Decide to Add or Change Treatment Strategies**

Before closing, we would like to comment on a number of additional treatment strategies that are not explicit components of most standard CBT approaches, which may help address some of the common difficulties that case formulation can reveal. Unfortunately, matching data that indicate specific treatment alternatives to use based on unique client characteristics are limited, both generally (see Project MATCH Research Group, 1993) and for specific anxiety problems (see Clarkin & Levy, 2004; Dusseldorp, Spinhoven, Bakker, Van Dyck, & Van Balkom, 2007). Thus, it is unclear at this point whether these suggested alternatives will ultimately garner empirical support as effective treatment matches for the presenting complications. However, the following suggestions address the theoretical mechanisms thought to underlie the given treatment complication, so are a reasonable place to start. It is important to note that this list is by no means exhaustive, but is included as an initial guide to respond to common problems that can arise in the treatment of anxiety disorders. For further information on combined treatment strategies, see Chapter 5.

### ***Treatment Complication 1: Lack of Motivation or Difficulty with Follow-Through***

One of the biggest challenges in psychotherapy is working with clients who have difficulty following through on treatment plans and homework assignments, who have many prior failed treatments, or who experience hopelessness about their ability to change. This problem can sometimes be recognized early in the case formulation process by either learning about a long history of unsuccessful past treatments (especially multiple cases of dropping out of treatment), or by giving a mini-homework assignment and evaluating whether or not this is completed between sessions. We frequently ask clients to spend some time between the first couple of sessions thinking about their specific goals for therapy and trying to identify concrete ways their life would be different if treatment were effective. Whether and how this assignment is completed may give early clues about whether motivation and follow-through are likely to be treatment barriers.

Fortunately, there are a number of specialized treatment approaches that have been developed to address these difficulties. For instance, Motivational Interviewing (MI) (Miller & Rollnick, 2002) is a widely used technique that draws from the transtheoretical model of change (Prochaska & DiClemente, 1992) to highlight the differences between clients' goals and their current behavior, and tries to reduce these discrepancies. Motivational Interviewing has achieved considerable success in helping clients overcome difficult motivational problems in order to profit from treatment, particularly within the realm of substance abuse (Burke, Arkowitz, & Dunn, 2002). Recent evidence indicates that Motivational Interviewing may be beneficial for clients with anxiety problems as well. For example, Westra and Dozois (2006) found that individuals with a principal anxiety diagnosis displayed greater benefits from CBT if they first participated in Motivational Interviewing sessions.

Readiness Treatment (VanDyke & Pollard, 2005) is another promising method for working with individuals who have failed to respond to at least one first-line treatment approach. The basic principle underlying Readiness Treatment is that treatment-interfering behaviors (TIBs; i.e., behavior patterns incompatible with successful participation in treatment) may have disrupted therapy. Common TIBs include failure to acknowledge having a problem, difficulty following the treatment plan, or frequently coming late to treatment sessions. Thus, cognitive interventions are primarily designed to focus on readiness for treatment and beliefs associated with the TIBs, as opposed to focusing on beliefs directly related to the anxiety disorder. Ideally, the TIBs should be added to the problem list and become an active part of the case formulation. Originally developed for OCD, initial pilot data suggest that Readiness Treatment may be effective in reducing TIBs so that clients can more fully engage in treatment (VanDyke & Pollard, 2005). At this point, future research is needed to more fully establish the efficacy of Readiness Treatment for anxiety problems more broadly.

### ***Treatment Complication 2: Interpersonal Problems***

Interpersonal issues often disrupt treatment, and may need to be addressed to begin making progress in therapy or to restart a “stuck” treatment. This is part of why it is imperative to create a full biopsychosocial problem list at the outset of treatment, which can help to identify interpersonal problems early on. Additionally, there are times when maladaptive relationship patterns may contribute to the client’s anxiety disorder. Frequent reassurance-seeking was one example mentioned earlier. We also sometimes see interpersonal conflict arise when the client starts to make progress in therapy and their dependence on others is reduced as avoidance behaviors diminish. This often requires redefining roles in the relationship, and can dramatically change power dynamics in the relationship. Although the progress in therapy is clearly positive, treatment can stall if these new relationship demands mean that avoidance behavior (rather than exposure) is being reinforced. In these cases, therapists will want to identify interpersonal problem areas that seem thematically or temporally related to the client’s anxiety disorder and incorporate them into the case formulation.

Applying techniques from interpersonal psychotherapy (IPT; Weissman, Markowitz, & Klerman, 2000) is one approach to handling these problems. Although IPT focuses on identifying and changing interpersonal problems implicated in the development of depressive episodes, these same types of problem areas are often important in pathological anxiety. Traditionally, these include unresolved grief, disputes with friends or relatives, difficulties forming or maintaining relationships, and problems coping with a life transition (e.g., leaving home for college, getting married, having a baby, etc.). Notably, IPT is an empirically supported treatment for both depression (Weissman et al., 2000) and bulimia nervosa (Fairburn, Jones, Peveler, Hope, & O’Connor, 1993), two disorders that share a high rate of comorbidity with anxiety problems. Further, initial pilot studies indicate that IPT may be an effective alternative for treating anxiety problems, including social phobia (Lipsitz, Markowitz, & Cherry, 1999), PTSD (Bleiberg & Markowitz, 2005), and panic disorder (Lipsitz, Gur, & Miller, 2006). Additionally, Crits-Christopher, Gibbons, and Narducci (2005) suggest that clients suffering from GAD may particularly benefit from interpersonally oriented therapy given that relational fears are the predominant worry domain in GAD (Roemer, Molina, & Borkovec, 1997).

### ***Treatment Complication 3: Emotion Regulation Difficulties***

There are a variety of ways that severe emotion regulation difficulties may impact treatment of anxiety problems. For instance, early exposure exercises, ratings of automatic thoughts, or mood evaluations may indicate that a client uses only extreme ends of rating scales, reporting either exceptionally low or high anxiety, regardless of the provocation. This is useful information for the case formulation because it may indicate that the client has trouble feeling or expressing gradations in

emotions, and is experiencing the world in a very all-or-nothing fashion. Similarly, clients may have difficulty identifying a range of different emotions, using anxiety as a default response when the situation may be eliciting sadness, disgust, anger, or other forms of negative affect. We often find this pattern will emerge early in the case formulation process if therapists inquire about triggers for the current episode. In other cases, emotion regulation difficulties present later in treatment, and can then be used to revise the case formulation. For example, we occasionally see clients who reliably over- and then under-predict the fear they expect to experience in various situations, contributing to a recycling pattern of excessive avoidance and lack of self-efficacy, followed by disappointment over a perceived failed exposure.

Fortunately, a number of treatment approaches have been developed to enhance emotion regulation skills, including those specific to treatment-resistant anxiety disorders (see Mennin, 2006). Also, dialectical behavior therapy (DBT) (Linehan, 1993), an empirically supported treatment for Borderline Personality Disorder, includes multiple emotion regulation strategies. Although we are not aware of clinical trials evaluating the efficacy of DBT specific to treating anxiety, there is evidence that integrating aspects of DBT may be useful. For example, Cloitre, Koenen, and Cohen (2002) found that individuals with PTSD benefited from the inclusion of DBT strategies focused on emotion regulation skills prior to exposure work.

When incorporating DBT, clients are taught to observe and describe their current emotional state (without judgment), placing a particular emphasis on separating descriptions of how one is *feeling* from descriptions of the actions that led up to that emotion. DBT techniques include helping clients to identify the precipitating events for emotional reactions, instructing clients to observe ongoing cognitive, physiological, and nonverbal behavior responses, and focusing on what other people might feel in similar situations (Linehan, 1993). For example, the client who consistently fails to predict how fearful she will be in a given situation may be instructed to imagine what others would feel when encountering a similar challenge. Meanwhile, an anxious client who has difficulty identifying a range of emotions may be taught to pay attention to physiological and behavioral reactions for “cues” that highlight the complexity of his or her emotions.

#### ***Treatment Complication 4: Difficulties with Relaxation and Acceptance***

It is not uncommon for the case formulation to reveal difficulties with acceptance of negative affect and arduous life circumstances, or problems with relaxation. During the assessment and initial case formulation phase, clients often report having difficulty relaxing. Alternatively, this problem becomes apparent if the therapist asks how the client spends his or her leisure time. Some individuals may not actually *do* anything to relax, and many anxious persons are unaware that they lack pleasurable activities in their life designed just for fun. In our experience, it is less common for clients to directly report that they have problems with acceptance; yet, this may also

constitute a treatment complication in pathological anxiety. This often emerges during case formulation when a client repeatedly talks about an issue that they cannot seem to “let go” of (e.g., an old relationship or perceived slight). In some cases, the difficulty focuses on rumination over an incident that the client sees as tied to the onset of the anxiety problem, such as an experience of childhood bullying that contributed to excessive worry or fears of negative evaluation. Although difficulties with relaxation and acceptance clearly differ, similar treatments may be helpful for both when standard relaxation techniques that are part of many CBT formulations are not successful at resolving the impasse.

In particular, Mindfulness (e.g., Kabat-Zinn, 1990) and Acceptance and Commitment Therapy (ACT; Hayes, Strosahl, & Wilson, 1999) may be useful approaches to target problems with relaxation or acceptance. For example, Mindfulness, a type of awareness stemming from Eastern traditions, focuses on relaxation techniques, developing an awareness of different possibilities, and altering habitual ways of responding (Martin, 1997). Demonstrating its potential utility, a group intervention based on mindfulness meditation led to significant reductions in anxiety among people with generalized anxiety and panic disorders (Miller, Fletcher, & Kabat-Zinn, 1995). Roemer and Orsillo (2002) advise that when incorporating mindfulness into traditional CBT, clinicians should focus on enhancing awareness of patterns of anxious responding. For instance, they suggest teaching clients to contrast typical patterns of avoidance with mindfulness techniques, such as “noticing and letting go” of tension during progressive muscle relaxation.

ACT, another technique to address difficulties with relaxation and acceptance, has received preliminary empirical support for treating a variety of problems (Hayes, Follette, & Linehan, 2004), including anxiety disorders (Twohig, Masuda, Varra, & Hayes, 2005). ACT is premised on the idea that trying to eliminate the occurrence of negative thoughts and feelings may be counterproductive (Hayes et al., 1999); instead ACT focuses on altering the ways that difficult private experiences function mentally. Researchers stress that a so-called “negative thought” or “negative emotion” that is mindfully observed may no longer *function* in a negative way, even if it might in other contexts (Hayes, Luoma, Bond, Masuda, & Lillis, 2006). Thus, ACT may be particularly useful for addressing acceptance concerns in anxiety given that a core goal of the treatment is to facilitate acceptance and a sense of “psychological flexibility” (Hayes et al., 2004). However, it should be noted that other authors have questioned the claim that ACT is uniquely different from CBT (Hofmann & Asmundson, 2008) and questioned the empirical support for ACT (Öst, 2008).

### ***Treatment Complication 5: Information Processing Biases and Rigid Thinking***

Cognitive models of anxiety disorders have increasingly relied on information processing paradigms to better understand the maintenance and development of maladaptive anxiety and avoidance (Beck & Clark, 1997). These paradigms suggest that reductions in the tendency to preferentially process potentially threatening

information may decrease anxiety symptoms (see Williams, Watts, MacLeod, & Mathews, 1997). Not surprisingly, biases in the ways clients attend to, interpret, and recall threat cues often figure prominently in case formulation, and can be detected in a variety of ways. For instance, during the initial assessment, accounts of prior fear-relevant interactions can be challenged to examine the rigidity with which a client clings to overly negative interpretations of the encounters.

When the case formulation reveals a rigid pattern of selectively processing threat material, an experimental treatment approach known as “information processing training” may be considered. During information processing training, researchers are attempting to reduce anxiety by literally “re-training” biases in interpretation of and attention to danger cues. Although still preliminary, results are promising that these techniques may effectively shift processing biases in healthy (see MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002; Mathews & MacLeod, 2005) and anxious populations (e.g., Amir, Weber, Beard, Bomyea, & Taylor, 2008; Teachman & Addison, 2008). Further, information processing training may be used to augment existing, empirically supported approaches to help clients consolidate their treatment gains more rapidly. Note, though, that the ultimate impact of these types of interventions for reducing anxiety symptoms is not yet known.

### ***Treatment Complication 6: Low Self-Efficacy and Losing Treatment Gains***

In some instances, an empirically supported treatment is showing signs of progress, but the gains are painfully slow, suggesting additional treatment may be necessary. If the case formulation reveals that a client has extremely low self-efficacy (e.g., about the ability to implement treatment strategies), or the client is regularly losing gains between sessions or having trouble practicing on his or her own, more intensive treatment may be indicated. Whenever possible, decisions about enhanced treatment should be made collaboratively by the client and therapist. Further, more intensive treatment should be framed as additional support as opposed to a failure on the client’s part.

Introducing more intensive treatment can be as simple as adding a few “booster” sessions, or increasing sessions from once to twice a week. For certain clients, particularly those whose issues are most salient in their homes, adding home visits may also be helpful. OCD clients with hoarding problems, for example, may greatly benefit from having a therapist come to their house to help begin the exposure exercises necessary to get rid of excessive belongings. Alternatively, when pathology is so severe that significant treatment gains are unrealistic in a standard setting, inpatient care may be recommended. This more intensive form of treatment offers several advantages over traditional outpatient care, including enhanced structure, support, and therapeutic contact (VanDyke & Pollard, 2005). For example, Abramowitz, Foa, and Franklin (2003) found that, although treatment effects for twice-weekly outpatient (versus inpatient) work were similar for clients with OCD, there was a trend for clients in the more intensive setting to show greater symptom improvement.

## Conclusion

In this chapter, we have outlined just a sampling of the myriad ways that case formulation can help rejuvenate a flailing anxiety treatment. While the chapter focused mainly on the steps used to develop a CBT case formulation and ways that these steps can aid in identifying potential treatment complications early on, it is clear that case formulation across many different treatment modalities can help manage difficulties in anxiety disorder treatments. In most cases, CBT case formulation will initially be matched with CBT approaches. However, we have tried to show that when these first-line approaches are not successful, alternative treatment options may be helpful. Case formulation is especially useful for highlighting likely treatment barriers early in the evaluation and therapy process, so that minimal time is wasted on strategies that are not likely to bear fruit. Further, because case formulation involves generating multiple hypotheses about the factors that led to the development and maintenance of the mechanisms fueling the disorder, the process naturally leads to multiple, creative solutions to address problems. Case formulation is a dynamic process that encourages clinicians to be similarly dynamic in their treatment planning. When case formulation becomes stagnant, so too will treatment. Whether using CBT or another modality, case formulation is most successful in helping to resolve treatment complications when it encourages therapists to think outside the box and truly use collaborative empiricism – iteratively trying and then evaluating new ideas to move therapy forward.

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# Helping Exposure Succeed: Learning Theory Perspectives on Treatment Resistance and Relapse

Mark B. Powers, Bram Vervliet, Jasper A.J. Smits, and Michael W. Otto

Exposure-based interventions are core to the psychosocial treatment of anxiety disorders. Protocols utilizing these elements are associated with some of the highest effect sizes in the anxiety treatment literature (e.g., Hofmann & Smits, 2008). These interventions rely on the use of experience to aid the learning of safety in relation to the core fears underlying anxiety disorders; yet, the efficacy of these interventions may be limited by a number of contextual and procedural challenges. This chapter provides a review of studies of these procedures and parameters and provides a conceptual overview of a heuristic for guiding exposure interventions. Relative to this heuristic, we attend to the stimulus properties, duration, spacing, gradation, and, particularly, context of exposure interventions.

## Exposure Is New Learning

Perhaps the most important concept in helping clinicians arrange effective exposure is the evidence that extinction learning is *new learning* rather than the weakening of previous associative learning (Bouton, Woods, & Pineno, 2004; Rescorla, 2001). Because the old learning remains intact, the new learning of safety competes with the old fear learning (Bouton, 2002). Successful treatment results when the new learning becomes the dominant association to the once-feared cues (e.g., an expectant audience in the case of social anxiety disorder). Said differently, after exposure treatment, the meaning of fear cues becomes ambiguous, with both anxiety-relevant and safety meanings associated with the cue. The task of the cognitive behavior therapist is to ensure that safety learning dominates this competition between the memories, so that cues once core to the anxiety disorder no longer elicit anxiety and avoidance when encountered. As such, the question for the clinician is how to arrange exposure so that a patient best learns that the phobic cues underlying the disorder are safe.

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To help achieve this aim, clinicians need to consider how to link the feared cues with memories of successful exposure. As will be reviewed below, this is done by completion of exposures across the range of the most salient cues and contexts, and the use of as many recall and generalization cues as possible. In short, the more salient that the extinction learning can be made to the patient around the most relevant cue (“this is exactly what I have been fearing; I expected a bad outcome, but it did not occur”) the more relevant the learning. Also, the more that a patient can rehearse and remember this learning, the better this learning will be available for recall. Our own heuristic for achieving these ends are drawn richly from the animal learning research and experimental studies discussed below. Nonetheless, it is important to note that the result of the heuristic is similar to those derived from a cognitive perspective that seeks to use experience from “behavioral experiments” to provide unambiguous opportunities to change beliefs underlying anxiety disorders (e.g., Wells et al., 1995). Indeed, when extinction is considered appropriately as new learning, and when a variety of retention and recall aids are adopted for this new learning (including accurate cognitive cues, “I can do this” “these situations are no longer bothersome”) much of the distinction between behavioral experiments used in cognitive therapy and exposure interventions plus cognitive generalization cues is rendered moot.

In arranging exposure therapy for anxiety patients, we believe clinicians have three central tasks: (1) helping the patient identify the core fears underlying the anxiety disorder, (2) arranging exposure interventions to disconfirm those fears by providing the patient with direct experiences with their ability to safely confront and cope adequately with the feared cues, and (3) demonstrate this ability to themselves in enough diverse contexts so that this ability is strongly remembered for application in the future. Concerning the identification of the core fears, we recommend careful evaluation of the individual patient in relation to the likely patterns identified by psychopathology research. For example, psychopathology research provides strong evidence that panic disorder is characterized by fears of and catastrophic interpretations of anxiety and panic symptoms, whereas social anxiety disorder is characterized by fears of and catastrophic interpretations of humiliation or embarrassment (Barlow, 2002). Nonetheless, despite the commonality of these core fears among individuals with these disorders, there are important individual variations on how these core fears are experienced. For example, in panic disorder, individuals fear different anxiety sensations and have different catastrophic interpretations of these sensations. Targeting exposure to the exact fears experienced by the patient will provide the most relevant learning of safety by elucidating the exact contingencies to be broken by the exposure (non-occurrence of aversive outcomes). For example, a concern about symptoms of dizziness in panic disorder, due to fears of fainting, may be linked to any of a number of additional concerns such as the fear of falling, fears of scrutiny (e.g., “I will look stupid if I am woozy”), or fears of being incapacitated (“others would take my wallet if I faint”). Elucidation of the specific concern, for example with a traditional “downward arrow” questioning technique (Burns, 1980), is helpful in specifying the correct conditions for targeted exposure.

Although the induction of dizziness with interoceptive exposure (e.g., head-rolling or spinning in a chair) would be a core intervention for panic disorder

(Barlow & Craske, 2007), but it would be delivered differently to address the core concerns of a specific patient. For example, for core fears linked to falling, interoceptive exposure to dizziness while standing may be especially relevant; for fears of scrutiny, performance of a social task while dizzy may be important; and for fears of being incapacitated, induction of dizziness around strangers may be the exposure of choice. In all these instances, the goal is to provide therapeutic learning by showing the patient that no aversive outcome follows exposure to the fear cues. Exposure exercises need not start with this bull's eye target, but it will be important for the therapist to work toward this top-of-the-hierarchy set of cues to achieve full extinction of the core fear.

As part of the task of honing exposure toward core fears, consideration of the additive property of fear cues is important. For example, exposure to memories of an assault in an individual with PTSD may function very differently during an evening session (when it is dark) versus during the day. Likewise, giving a speech as part of an exposure for an individual with social anxiety disorder may be experienced very differently depending whether cues of anxious performance are present (e.g., dry mouth and a pounding heart). It is the clinician's central task to determine the nature of the safety learning that needs to be achieved from the exposure. For example in the treatment of panic disorder, the richness of the cues around driving concerns can help reveal the top-of-the-hierarchy cues for exposure as well as relevant steps along the way in building the hierarchy of exposure successes. Rather than simply focusing on driving in traffic as the relevant exposure, the top-of-the-hierarchy cues should be thought of as the additive influence of all the relevant stimuli: *in car + driving the expressway + rush hour + no exits available + alone + panic symptoms present + the thoughts "I can't make it; I am going to crash the car."*

If extinction represents the active reacquisition of safety in response to these cues, then the clinician needs to arrange for the stepwise exposure to each of these elements either alone or in combination. The degree of combination rests on how high the clinician wants to start in the fear hierarchy (see staging of exposure below) to help ensure stepwise success. For panic disorder, current protocols start this process with exposure to the feared panic sensations using interoceptive exposure techniques (see "Avoiding Treatment Failures in Social Anxiety Disorder" by S. G. Hofmann et al., this volume). The goal is to have the patient *do nothing* in response to these sensations to learn that they are not dangerous or in need of management. Once safety is learned to this element of the larger top-of-the-hierarchy fear, the clinician can begin chaining together the fear cues. Exposure homework away from the therapist can help create safety for the stimulus *panic sensations + alone*. Following success with this stimulus context, the clinician can assign interoceptive exposure in the car to achieve safety learning for *panic sensations + alone + car*. When comfort is achieved with this cue, the next step may be to re-introduce the catastrophic thoughts as part of the extinction stimulus (following cognitive restructuring to help the patient gain perspective on the inaccuracy of these thoughts): *panic sensations + alone + car + "I can't make it; I am going to crash the car."* Interoceptive exposure followed by practice driving safely under conditions of feeling odd may be the next step, and finally, when so many of the cues of the top-of-the-hierarchy concern

have been exposed to safety learning, the clinician is in the position of assigning driving the expressway as an exposure homework. This can be done both with and without interoceptive exposure to help the patient create a true sense of safety in this situation regardless of the presence of anxiety symptoms. With the completion of this work driving the expressway should become incapable of eliciting a panic attack (Hofmann, Richey, Asnaani, Sawyer, this volume).

The chaining together of feared cues can be crucial for helping the clinician target the most durable and relevant learning for the anxiety disorder. In the case of social anxiety disorder, it is easy to focus on the acquisition of safety around a well-done social performance. Under these conditions the clinician should expect that the patient has learned relative safety under conditions of a *well-done social performance*. Fuller acquisition of safety may require the acquisition of safety under poor performance conditions. Indeed the focus on “social mishap” exposures in the treatment of social anxiety disorder (see Hofmann & Otto, 2008; Hofmann, Richey, Asnaani, & Sawyer, this volume) is focused on helping patients undo the true core fear of the consequences of poor social performance (not just the lower level fear of *any* social performance). Hence, programming in social difficult exposure (e.g., spilling a coffee; see Hofmann & Otto) should lead to fuller safety learning than a focus on adequate performance exposures alone.

Finally, to help lock in this safety learning, patients need to have confidence in that negative outcomes do not occur despite variations in the conditions in which they confront their feared cues (for animal studies see Gunther, Denniston, & Miller, 1998; Chelonis, Calton, Hart, & Schachtman, 1999, but see Bouton, Garcia-Gutierrez, Zilski, & Moody, 2006; for clinical studies see Rowe & Craske, 1998b; Vansteenwegen et al., 2007). As clinicians know well, a patient’s phobic concern may differ greatly depending on the context in which it is encountered, e.g., on a *good* versus *bad* day.<sup>1</sup> These contextual factors have a powerful influence not only on the degree of anxiety elicited by a feared cue, but, as reviewed below, can serve as a powerful factor in incomplete treatment and relapse. To provide a full accounting of the power of context in extinction, we turn toward both animal and applied research.

## Context Effects

The conceptualization of extinction as new learning rather than the erasure of old learning is supported by findings from a range of animal, analogue, and clinical studies that illustrate the return of fear with a shift in contextual (recall) cues for either extinction or original fear learning (for review see Bouton, 2002;

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<sup>1</sup>Although clinicians may not be privy to the full learning history of their patients, and hence may not know fully what cues are simply a setting condition (not directly feared by signaling conditions under which the phobic cues are more concerning) or are a fear cue in their own right, this conceptual confusion rarely translates to clinical confusion when it comes to arranging exposure.

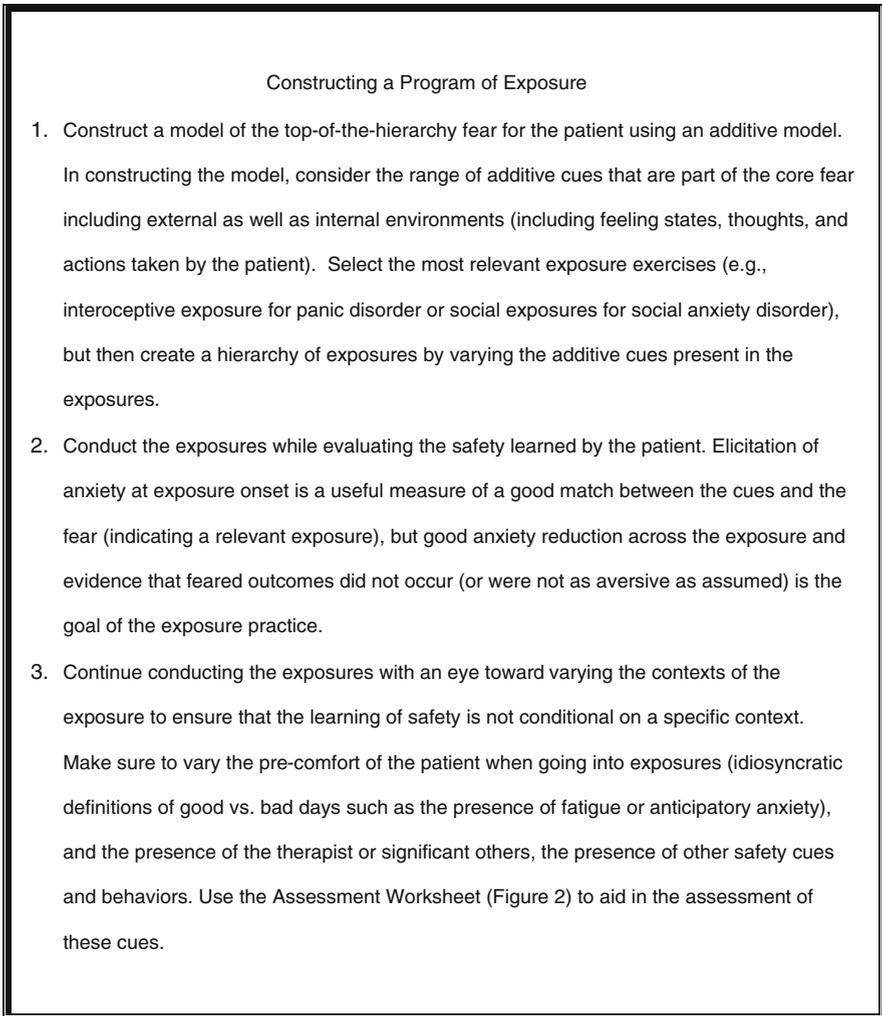
Craske, Hermans, & Vansteenwegen, 2006). In terms of clinical application, fear *renewal* refers to the return of the fear response when there is a context shift after treatment. Contexts can include both internal (i.e., drug state) and external (i.e., darkness) factors. The most widely accepted evidence for the role of contexts in animal and human fear acquisition, extinction, and relapse is provided by the contextual renewal paradigm. The contextual renewal paradigm includes an acquisition (fear learning) phase in one context (A), an extinction phase in a second context (B), and finally an evaluation of which memory (fear or safety learning) is dominant in a test context (the fear context, A; the extinction/safety context, B; or a novel context C). For example, a patient may develop claustrophobia in one context A (i.e., a closet as a child), then receive treatment in a second context B (i.e., claustrophobic treatment chamber), and the “test” then is after treatment in a third context C (an elevator). Unfortunately for clinical treatment, there is ample evidence that extinction learning, as compared to the original fear learning, is much more sensitive to context effects. This conclusion, and the return of a fear after a shift in context (fear renewal), is supported by (a) animal studies with an ABA design (e.g., Bouton & King, 1983), an ABC design (e.g., Bouton & Brooks, 1993) and an AAB design (e.g., Bouton & Ricker, 1994), (b) human conditioning with an ABA design (Vansteenwegen et al., 2005; Milad, Orr, Pitman, & Rauch, 2005) and an ABC design (Effting & Kindt, 2007), and (c) human treatment studies (Mineka, Mystkowski, Hladek, & Rodriguez, 1999; Mystkowski, Craske, & Echiverri, 2002; Mystkowski, Mineka, Vernon, & Zinbarg, 2003; Rodriguez, Craske, Mineka, & Hladek, 1999). In these studies, context can be such stimuli as the external environment (e.g., cage design; Bouton, 1993; Harris, Jones, Bailey, & Westbrook, 2000), internal stimuli (e.g., level of drug modulated arousal; Bouton, Kenney, & Rosengard, 1990), or recent events (e.g., Bouton, Rosengard, Achenbach, Peck, & Brooks, 1993). For example, animal research indicates that internal state (e.g., anxiety/arousal modulation from benzodiazepine administration) is a sufficiently context cue such that extinction (safety) learning may be achieved solely in that context (Bouton, Kenney, & Rosengard, 1990). In an experimental demonstration of a similar effect in humans, Mystkowski et al. observed a significant return of fear among phobic patients who underwent exposure therapy after they had ingested either caffeine (arousal induction) or placebo (no arousal reduction) and then were tested at a follow-up period in the context of either the same or different arousal condition. Regardless of the direction of the change in internal context (caffeine to placebo or placebo to caffeine) those who were tested under a different internal state had more return of fear than those tested under the same arousal condition as during extinction. Indeed, this context (internal state) dependency has been used to explain the apparent loss of CBT efficacy when it is delivered in the context of medication treatment that is later discontinued (Otto, Smits, & Reese, 2005; see Hofmann et al., this volume).

To date, three mechanisms have been proposed for the renewal of fear responses including the following: (a) the Modulatory Mechanism – new extinction information only applies in the extinction context (Bouton, 1988, 2000), (b) the Inhibitory

Mechanism (Lovibond, Davis, & O'Flaherty, 2000) – the extinction context predicts no US and thus protects the CS from extinction (“I not at risk under these conditions”); and (c) Generalization Decrement (Lovibond, Preston, & Mackintosh, 1984) – the extinction stimulus is “wrong” and thus protects the acquisition stimulus from extinction (“I was never afraid of this”). All accounts stress the match between extinction and the contexts that signal that the fear cue is really to be feared. Future research may determine which theory best fits the data.

Return of fear can also occur with the mere passage of time following extinction training (spontaneous recovery: Brooks & Bouton, 1993; Brooks & Bowker, 2001; Brooks, Karamanlian, & Foster, 2001; for a demonstration in humans, see Marescau, Vansteenwegen, Vervliet, & Eelen, unpublished manuscript). Bouton (2002) has argued that “time” can also function as a context (as internal and external contexts change over time). In that sense, testing the extinguished CS at a later moment in time is conceptually equivalent to contextual renewal. Accordingly, a fading of treatment sessions or the use of a booster session months after the last weekly session may have a valuable influence on helping patients know that they can still respond well to phobic cues despite passage of time. These procedures help ensure that the passage of time since extinction training is linked in memory to continued extinction success not to the old fear memories. This procedure also serves as a nice introduction to one of the core feature of our heuristic for the application of exposure (Fig. 1) – exposure should be programmed across relevant contexts to ensure that memories of success will be available at the time of need. Given the extraordinary cognitive abilities of humans, this programming can occur in these realms in addition to the active practice of exposure across contexts. Even these cognitive procedures are consistent with the notion from animal research that contextual cues set up “expectancies” for extinction or fear memories. In the case of time cues, in addition to the use of booster sessions and booster homework assignments (e.g., the programming of exposure homework for intervals after formal therapy ends), the therapist may help the patient understand the nature of the treatment changes to help the patient be prepared on how to approach phobic situations in the future (e.g., “I am different now that I have had treatment, I know how to approach my phobic situations/events differently now.”).

In addition to the external and internal environment, contextual cues also include what the patient does in the situation (e.g., superstitious behaviors that create a context for the patient that signals relative safety). As explicated by all three of the theoretical accounts of context effects, these behaviors could signal trouble for extinction. According to the Modulatory Mechanism, the use of these superstitious behaviors would help insulate extinction learning from true safety should those superstitious behaviors not be used or available. According to the Inhibitory Mechanism no aversive outcome would be expected anyway under these conditions (“I knew I would be OK because I...”); and, similarly, according to the Generalization Decrement “I was never afraid of the exposure stimulus as long as I could...” To broaden this discussion, we will broaden and re-label the concept of superstitious behavior as “safety behaviors.”



**Fig. 1** A heuristic for exposure planning

## Safety Behaviors

Safety behaviors are subtle avoidance behaviors people use to reduce anxiety or prevent a perceived threat that can undermine the efficacy of exposure (Wells et al., 1995). Some examples include carrying a cell phone for fear of needing help, carrying rescue medication (benzodiazepines) to take in the event of a panic attack, using air conditioning to reduce sweating, drinking alcohol to reduce anxiety, gripping the wheel tightly while driving to prevent an accident, or asking questions

excessively to prevent ambiguity. As we see from the list the defining characteristic of a safety behavior is not the form of the behavior but rather the function that is important. If the perceived threat is a potential “true alarm” (i.e., car accident) then the safety behavior may be adaptive (i.e., wearing a seatbelt). However, if the threat is “false alarm” (i.e., panic attack) then using safety behaviors may prevent the learning of unambiguous safety – I’m safe as long as I have my medication in my pocket. Indeed, Powers, Smits, and Telch (2004) showed that participants who had safety behaviors available during exposure treatment reported significantly less improvement compared to participants who did not rely on safety behaviors (i.e., response rates were 45% versus 95%). Interestingly, the deleterious effects of safety behaviors were evident whether patients used them or not – suggesting the mere availability of safety behaviors can interfere with fear reduction. These data are consistent with clinical observations that patients who carry rescue medications (i.e., benzodiazepines) are at greater risk of relapse even if they never take them.

When it comes to medication treatment, one direct measure of the context specificity of exposure learning is the attribution of treatment gains. For example, Basoglu et al. (1994) reported a 60% relapse rate at medication-free follow-up if patients in their combination treatment trial attributed their gains to medication. This is compared to no relapses in the group that attributed their gains to their own efforts. However, this correlational design prevents causal inferences. Powers, Smits, Whitley, Bystritsky, and Telch (2008) conducted a randomized trial with claustrophobic participants who underwent exposure-based treatment in the context of a pill placebo that they were led to believe was a medication that made the exposures easier (sedating side-effect profile) or more difficult (activating side-effect profile). Consistent with prediction, an attribution for pill facilitation led to higher relapse. The return of fear rate at follow-up was nearly 40% in the medication attribution group compared to 0% in the other conditions. These data highlight the importance of monitoring patient attributions for improvement.

Taken together, a central implication of these context studies is that clinicians must plan for the generalization of safety learning from the treatment environment (at the clinic, with the therapist, etc.) to non-treatment contexts and across the presence and absence of safety behaviors. Repeated exposure sessions on different days (when the patient is entering the session in different mood states, after different life events) and when exposure is conducted in different ways (standing, sitting, after induction of dizziness), as well as the use of homework (exposure without the presence of the therapist or clinic context) should help achieve this variability in training. Clinicians should also carefully consider the role of relaxation training in their anxiety treatment. Teaching the patient to relax in the presence of the phobic stimulus may create a unique context for safety learning, a context that may be lost should the patient be unable to relax adequately at a later point in time. In the treatment of research, there is evidence that relaxation training or breathing retraining does not lead to superior outcome (e.g., Schmidt et al., 2000), and accordingly protocols that encourage the experience of anxiety as part of the stimulus context (Eifert & Forsyth, 2005) may lead to more durable extinction.

**Exposure Planning Checklist**

**Attending to the Correct Core Fear:**  
 What is/are the core fear or fears that should be targeted by treatment? Ask the patient: “what is so bad about...” to elucidate the central feared features.

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

**Attending to the Contexts Surrounding the Fear:**  
**Aggravating Contexts**  
 What are the contexts in which this fear is worse? Consider the following:

- Time of day/year (including light dark, certain weather conditions (e.g., hot weather in the case of panic disorder): \_\_\_\_\_
- Presence of others (known or unknown people): \_\_\_\_\_
- Presence of symptoms (e.g., muscle tension, certain worries, etc.): \_\_\_\_\_
- Mental or physical fatigue (also including menstrual cycle): \_\_\_\_\_
- Interpersonal conflict: \_\_\_\_\_
- Other \_\_\_\_\_
- Other \_\_\_\_\_

**Safety Behaviors/Events**  
 What are the behaviors or events that lead the patient to assume relative safety from the feared outcomes?

- Contact with others (e.g., cell phone, presence of safe other, knowledge of availability of safe other): \_\_\_\_\_
- Food or drink (bottle of water, mints, antacids, crackers, fruit): \_\_\_\_\_
- Something to hold (glass)/ position near a wall or door: \_\_\_\_\_
- Medication (often benzodiazepine or beta-blocker use): \_\_\_\_\_
- Cognitive rituals (e.g., affirmations, lucky sayings): \_\_\_\_\_
- Body positions/eye contact (averting one’s eyes while speaking, clasping the hands, leaning against a wall, bracing against a chair): \_\_\_\_\_
- Talking with others: \_\_\_\_\_
- Other: \_\_\_\_\_
- Other: \_\_\_\_\_
- Other: \_\_\_\_\_

Fig. 2 Understanding exposure cues and contexts

Figure 2 is designed to help clinicians develop a plan for the application of exposure to core fears across contexts. It is used to help elucidate the range of contexts

that need to be targeted in treatment. Treatment planning thus involves progression along two types of hierarchies – movement toward the core fears that define the top of a hierarchy of concerns and movement along a continuum of contexts, removing safety behaviors and ensuring that fear at the top of the hierarchy are rehearsed across a range of contexts. An especially relevant context is medication use, and as detailed in Powers et al. (this volume), reinstatement of CBT across medication taper may be crucial for helping patients maintain their treatment gains when CBT was learned originally in the context of medication use

## Considering Exposure Parameters

By considering the core learning that needs to be achieved by the patient from exposure, and evaluating this learning in relation to the contexts under which it is to be learned and applied, a surprising amount of research on exposure parameters can be understood. In the following sections, individual exposure parameters are reviewed and then discussed relative to safety learning and context considerations.

### *Exposure Duration and Spacing*

Both animal and human studies indicate that continuous extinction trials yield better outcomes compared to distributed and interrupted extinction trials (Chaplin & Levine, 1981; Girodo & Henry, 1976; Baum, Andrus, & Jacobs, 1990; Davis, 1970; Mackintosh, 1974; McCutcheon & Adams, 1975; Miller & Levis, 1971). For example, Chaplin and Levine compared a single 50-min exposure with two 25-min exposures among participants with public speaking fear. The continuous 50-min condition outperformed the two 24-min exposures that were separated by a 10-min inter-trial interval. Clinical studies also demonstrate an advantage for continuous exposure (Foa, Jameson, Turner, & Payne, 1980; Grayson, Foa, & Steketee, 1986; Rabavilas, Boulougouris, & Stefanis, 1976; Stern & Marks, 1973). Stern and Marks compared 2-h sessions with 4½-h sessions of in vivo exposure for panic disorder with agoraphobia and found that the longer sessions yielded higher response rates. Foa et al. (1980) conducted a crossover study with agoraphobics and found that massed exposure (10 daily sessions) was superior to spaced exposure (10 weekly sessions), particularly in reducing avoidance. Foa and Kozak (1986) also suggested that a long (90 min) exposure outperformed nine brief exposures (30 s) with long inter-trial intervals (10 min) among participants with OCD. Another consideration is when to terminate an exposure trial. Subjective fear and physiological response during exposure typically follows a curvilinear trajectory (Foa & Chambless, 1978; Stern & Marks). As illustrated in Fig. 1, we can see that if a patient were to terminate treatment during Session 1 after 40 min of exposure they may be more “sensitized” than when they started treatment (Emmelkamp, 1982).

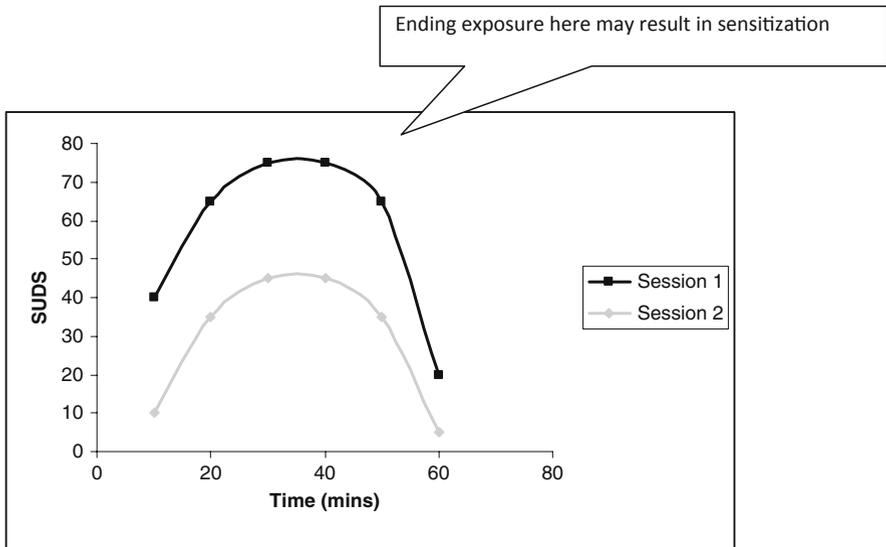


Fig. 3 Mean subjective anxiety ratings during exposure sessions

From the perspective of what is learned from therapy, longer exposures give a greater opportunity for unambiguous learning that the exposure cues can be tolerated and do not lead to negative outcomes. Ending an exposure when anxiety is low allows for the retention of a “comfortable” exposure experience and appears to aid ultimate outcome (Hayes, Hope, & Heimberg, 2008).

### *Distribution of Sessions*

Although massed sessions result in quicker extinction learning, spaced sessions result in more resilient extinction learning at follow-up (see Cain, Blouin, & Barad, 2003). This is consistent with the notion that intensive (massed) practice provides less ambiguous learning of safety, but that greater spaces between sessions allows for the integration of this learning relative to the contextual cue of passage of time, and hence reduces the likelihood of spontaneous recovery due to the passage of time as a context cue for the original fear learning. Craske and colleagues suggest capitalizing on the acute advantage of each of these strengths by massing sessions early in treatment and then gradually expanding intersession intervals to protect against relapse (Lang & Craske, 2000; Rowe & Craske, 1998a; Tsao & Craske, 2000). An example schedule of 12 treatment sessions for panic disorder may include twice-weekly sessions for 3 weeks then once-weekly sessions for 4 weeks and finally once every other week for 4 weeks.

### ***Stimulus Properties (Imaginal, In Vivo, Virtual Reality)***

Learning theory posits that the salience of the feared stimulus is an important factor influencing the speed of learning, both for the acquisition and extinction of conditioned fear (Rescorla & Wagner, 1972). Hence, it is anticipated that response changes occur faster to high versus low salient stimuli. Indeed, direct comparisons show the superiority of in vivo over imaginal exposure (Barlow, Leitenberg, Agras, & Wincze, 1969; Dyckman & Cowan, 1978; Emmelkamp & Wessels, 1975; Litvak, 1969; Sherman, 1972; Stern & Marks, 1973; Watson, Mullett, & Pillay, 1973). Emmelkamp and Wessels (1975) compared: (a) 90 min of flooding in vivo, (b) 90 min of imaginal flooding, (c) 45 min of imaginal flooding followed by 45 min of flooding in vivo. The results clearly showed an advantage for flooding in vivo. However, imaginal exposure can be effective when in vivo methods are not possible (Rentz, Powers, Smits, Cogle, & Telch, 2003; Rothbaum, Astin, & Marsteller, 2005). For example, flying phobics may find it difficult financially to regularly conduct in vivo exposures. Likewise, virtual reality exposure treatment (VRET) can be an option when in vivo exposure is difficult. A recent meta-analysis suggested that virtual reality treatment was more effective than waitlist and placebo treatments and equipotent to exposure in vivo (Powers & Emmelkamp, 2008). The meta-analysis of nine studies where virtual reality exposure therapy was compared to an adequate control group yielded a large combined effect size of  $g = 1.05$  (95% CI: 0.71–1.40). Emerging technology may further improve VR treatment. The new technology known as CAVE (CAVE Automatic Virtual Environment) increases the realism (immersion) resulting in more potent exposure (Cruz-Neira, Sandin, & DeFanti, 1993). Taken together, perceptual proximity (salience) seems an important factor for the efficacy of extinction treatments.

### ***Gradation of Exposure***

After constructing a fear hierarchy the patient and therapist must decide which exposure item should be the first target. Studies show that if patients stay in the exposure session long enough there is little difference in outcome between starting on an easier or more difficult item on the fear hierarchy (Gelder et al., 1973). However, there is some evidence that massed flooding outperforms spaced graded exposure at a 5-year follow-up (Feigenbaum, 1988). Even though flooding may be a viable option more patients may either reject or drop out of this type of therapy (Emmelkamp & Ultee, 1974; Emmelkamp & Wessels, 1975). A method that capitalizes on both options is to choose a moderately difficult item in the context of an expanding-spaced schedule (Foa & Wilson, 2001; Rowe & Craske, 1998a). For example, a therapist may suggest beginning exposure to an item with a SUD of 60 or 70 from the patient-generated fear hierarchy.

## ***Fear Activation***

Foa and Kozak (1986) proposed that successful exposure must include (a) activating the fear structure and (b) disconfirmatory evidence that must be available and processed by the patient (Foa & Kozak). The first assertion led some to hypothesize that with greater fear activation comes greater fear reduction. However, studies have not supported the theory that higher fear during exposure leads to better outcome (Hafner & Marks, 1976; Kirsch, Wolpin, & Knutson, 1975; Marks, Boulougouris, & Marset, 1971; Telch et al., 2004). Possibly, the crucial factor is not the level of fear, but the level of expectancy of negative outcomes. Indeed, associative learning models (e.g., Rescorla & Wagner, 1972) anticipate stronger extinction learning with a larger discrepancy between the expected presence of the aversive outcomes and its actual absence.

## ***Focus of Attention and Behavior During Extinction***

It is widely accepted that attention is a necessary precondition for learning to occur (Rescorla & Wagner, 1972; Mackintosh, 1975). However, although some clinical studies suggest that distraction during exposure leads to poorer outcome (Kamphuis & Telch, 2000; Telch et al., 2004), others do not (Johnstone & Page, 2004). In fact, Johnstone and Page found an advantage when patients engaged in threat-irrelevant conversation during exposure. Upon closer examination, it appears that the *type* of distraction is most important. If the distraction prevents the patient from functional exposure to the stimuli (i.e., cognitive load) then it will interfere with fear reduction (Kamphuis & Telch, 2000). Borkovec and Grayson (1980) stated that, “objective presentation of the stimuli does not guarantee functional exposure to those stimuli” (Borkovec & Grayson, p. 118). However, if the patient is able to “act normal” by having a conversation while still maintaining awareness of the stimuli then outcome may be enhanced (Johnstone & Page). Telch and colleagues propose that acting in a way that is inconsistent with fear sends safety information (Wolitzky & Telch, under review). For example, a patient may be instructed to smile during exposure even though inconsistent with their fear. Although such actions may enhance exposure-based treatment it is not yet certain why. However, research shows that producing a smile (even when unobtrusively) or a fearful expression elicits a consistent emotion (Flack, 2006; Larsen, Kasimatis, & Frey, 1992; Strack, Martin, & Stepper, 1988; Zajonc, Murphy, & Inglehart, 1989). One obvious possibility is that these interfering emotions during exposure are conceptually similar to a counterconditioning procedure. This procedure involves the pairing of the CS with a new US that has contrasting affective value (e.g., an appetitive versus an aversive stimulus), and is known to produce robust extinction of fear responses (e.g., Peck & Bouton, 1990) and even changes the affective value of the feared stimulus (Baeyens, Eelen, Van den Bergh, & Crombez, 1989). This counterconditioning hypothesis is fully consistent with the “new learning” account of extinction, and instead of pairing the CS

with “no aversive outcome” it pairs it with a positive stimulus. Further investigation of this process in human fear conditioning is pending.

## Concluding Comments

Research on the nature of fear extinction and relapse offers a number of valuable heuristics to guide interventions to minimize treatment resistance and relapse. This chapter highlighted a number of factors that may contribute to treatment resistance: (1) Both conditioning and clinical research has demonstrated weakened extinction effects with short and interrupted exposures to the feared stimulus, rather than long and continuous ones. (2) Expanding-spaced sessions seem to capitalize both on a

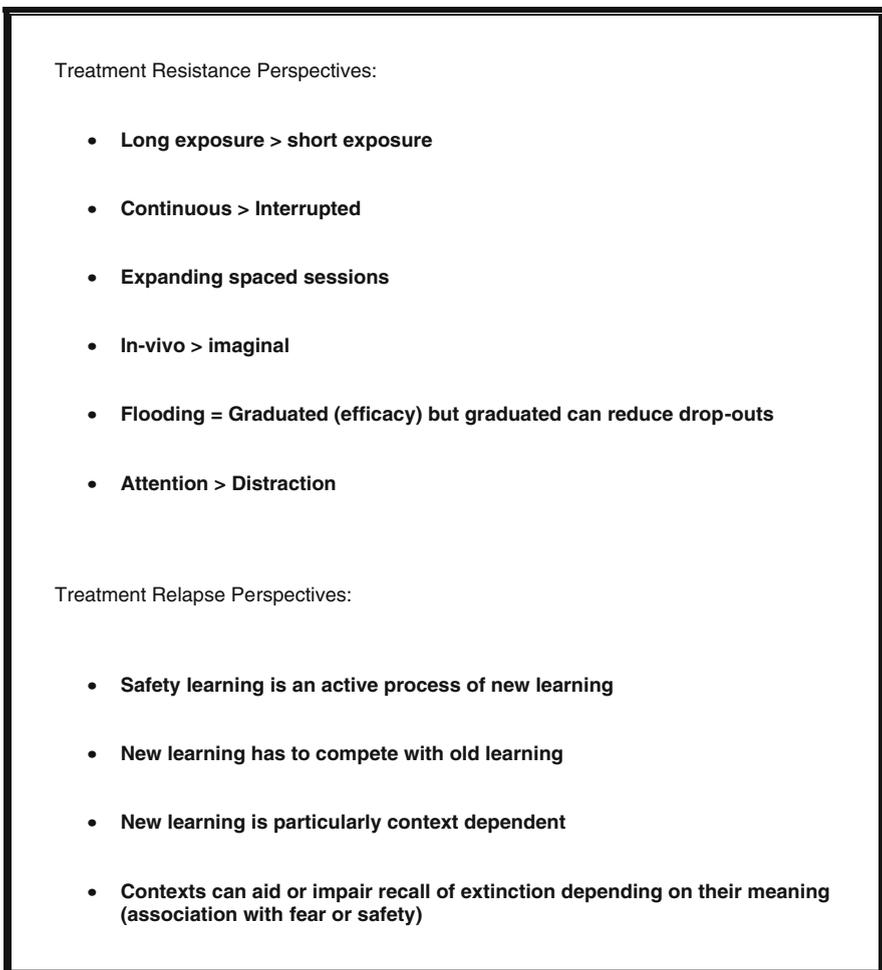


Fig. 4 Summary of learning perspectives on treatment resistance and relapse

fast reduction of anxiety (through initial massed extinction trials) and better retention on the long run (through later spaced extinction trials). (3) Exposure to high salience stimuli seems to produce better results, that is, in vivo exposure is more powerful than imaginal exposure. Virtual reality protocols are promising in this regard. (4) There is no clear picture on the graduated exposure versus flooding, but the former may lead to fewer drop-outs. (5) There appears to be no supporting evidence for the widespread idea that more fear activation during exposure leads to better extinction effects. Possibly, the expectancy of the occurrence of the threatening event (which is disconfirmed in extinction) is more important than the overt fear response. (6) Out-of-clinic practice (self-exposure) clearly improves the outcome of exposure treatment and (7) comorbidity does not seem to be a major problem for successful treatment of anxiety (Fig. 4).

It is now commonly acknowledged that extinction and exposure treatments are active learning processes, and result in the formation of an extinction memory that co-exists and competes with the original fear memory. Attention to the context of treatment and post-treatment application of therapy appears to be crucial for the long-term maintenance of treatment gains. We described a variety of procedures to aid clinicians in targeting the correct core fear and providing unambiguous training in safety learning across relevant contexts.

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# Therapeutic Alliance and Common Factors in Treatment

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Changes in therapy can result from variables unique to a particular treatment package (i.e., specific factors), as a function of variables common to a variety of treatments (i.e., nonspecific/common factors), or a combination of both specific and nonspecific ingredients (Kazdin, 1979). Nonspecific (or common) treatment factors include variables that are common across different treatment modalities, which are thought to influence outcomes in therapy but are often not well linked to a mechanism of change. Due to differences in what is posited to be important to the change process by any one treatment theory, specific factors that are deemed crucial for one theoretical approach might be considered a common factor for another approach (Kazdin, 1979; Wilkins, 1979).

A unified perspective on the role and significance of common factors is made more difficult by the degree to which similar nonspecific factors have been discussed as distinct concepts, with little guidance on the degree of overlap between these concepts. For example, therapeutic alliance, empathy, goal consensus, and therapist–patient collaboration have each been cited as important contributors to therapeutic change (Norcross, 2002), despite the obvious overlap between these concepts. For the organization of this chapter, we will use therapeutic alliance as a central organizing concept for discussing a range of common factors. As such, the focus of this chapter is on those factors distinct from the specific treatment interventions that are most commonly the focus of randomized controlled treatment trials. Particularly when a specific treatment is examined relative to a supportive treatment comparison condition, nonspecific treatment factors, such as entry into a supportive and caring professional relationship that has, as an explicit goal, the healing of a patient in need, are balanced across the two treatment conditions. Specific treatment effects represent the benefits demonstrated beyond these common factors (see Chambless & Hollon, 1998; Chambless & Ollendick, 2001). Specific treatment effects are also the focus of the second section of this volume (Otto & Hofmann, 2009), and hence this chapter provides a complement to the direct attention on empirically supported treatment strategies found in those chapters.

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## The Concept of Therapeutic Alliance

Traditionally associated with psychoanalytic theory, the therapeutic alliance was once conceptualized as a positive form of transference (Freud 1913/1958). Humanistic theorists expanded upon this idea by emphasizing the role of therapist empathy and unconditional positive regard in treatment success (Rogers, 1957). In recent years, the therapeutic alliance has gained recognition across treatment modalities as a common factor correlated with treatment outcome (Constantino, Castonguay, & Schut, 2002; Wolfe & Goldfried, 1988). Due to the early focus on the therapeutic relationship as a primary mechanism of change in psychodynamic therapy, there is sometimes an unfortunate tendency to use this theoretical frame of reference when discussing issues in the therapeutic relationship. Cautioning against this tendency, one of the founders of behavior therapy, Lazarus (2003), aptly stated:

Would any well-schooled CBT practitioner find it farfetched to point out to a client that she appears to react to her therapist in the same way that she views her abusive husband, and then use in-session cues to deal with the problem? Be that as it may, to refer to this as “transference” takes us very wide of the observation that clients may have distorted perceptions of the therapist that seem to rest on reenactments with significant others. . . . To borrow and use the murky term “transference” (on which many vague and complex psychoanalytic tomes have been written) leads to obfuscation. Perhaps it behooves us to find a different term that describes peoples’ penchant to generalize and project onto one another accurate as well as distorted attributes and feelings that stem from past experiences. (p. 380)

Accordingly, common factors in general, and therapeutic alliance and its determinants in particular, need to be discussed in general terms that are free from the theory-specific meanings reflected in their historical roots. Doing so frees clinicians and researchers to consider the range of understandings and interventions that may be relevant to addressing therapy relationship issues and maximizing therapy outcomes.

As the most frequently cited common factor, the alliance is now commonly defined as the emotional and collaborative bond between the therapist and patient (Martin, Garske, & Davis, 2000). According to Bordin (1979), the alliance is composed of three important factors: goals, tasks, and bonds. Goals refer to the desired outcomes that must be endorsed by both parties. Tasks vary based on a therapist’s theoretical orientation and should be tailored to facilitate progress toward a patient’s individual goals. Both therapist and patient should believe that the tasks are necessary for treatment and accept the responsibility to execute them. Lastly, the bond describes the therapist-patient attachment that depends largely upon high levels of trust.

## Empirical Support for the Therapeutic Alliance

Meta-analytic reviews across the years have shown a high degree of reliability in providing estimates of the strength of alliance effects in predicting outcome. For example, in a relatively recent large-scale meta-analysis of 79 studies, alliance was

found to be moderately correlated to outcome ( $r = 0.22$ ; Martin et al., 2000). This analysis replicated a meta-analysis from almost a decade earlier ( $r = 0.21$ ; Horvath & Symonds, 1991), suggesting stability in these effect size estimates over time. Likewise, in a meta-analysis of 23 studies examining treatment of children and adolescents, Shirk and Karver (2003) obtained almost identical results. All of these estimates are in the small range according to Cohen's (1988) standards.

By way of comparison, a meta-analysis of controlled trials of the treatment of anxiety disorders indicates a medium to large effect size (Hedges'  $g = 0.73$ ) according to Cohen's (1988) standards for the advantage of cognitive behavioral therapy (CBT) over psychological or pill-placebo comparison conditions for individuals completing treatment (Hofmann & Smits, 2008). There are a number of interpretations of this larger effect size. First, because alliance with caregivers is likely to be a factor in both the active and comparison conditions reviewed by Hofmann and Smits – the vast majority of control conditions were supportive counseling, followed by select use of pill placebo or other interventions such as relaxation or problem-solving training – these data may suggest that specific treatments offer substantial benefits beyond alliance and other common factor effects alone. Alternatively, alliance effects may differ depending on the treatment being offered, such that stronger alliance effects would be found in the CBT group relative to the comparison conditions, and hence alliance may be a confounded component in the larger effect sizes found for specific treatments.

There is evidence for such confounding between specific treatment and alliance effects. For example, significantly greater alliance effects were found for CBT as compared to interpersonal therapy in a study of the treatment of depression (Raue, Goldfried, & Barkham, 1997). Empirical evidence supports that alliance increases following treatment gains. For example, Tang and DeRubeis (1999) showed that individuals who experience sudden gains in treatment have a subsequent enhancement in therapeutic alliance. Hence, treatments that differ in efficacy may also differ in alliance, leading to a confounding in alliance assessments and variance attributable to what may be the specific effects of treatment. Moreover, there may be a mutually reinforcing cycle between alliance and treatment outcome, where early gains may enhance alliance and alliance may make the patient more receptive to ongoing treatment efforts. In one study, after controlling for prior symptom improvement in a sample of depressed patients, higher alliance scores on the Working Alliance Inventory (WAI; Horvath & Greenberg, 1989) after Week 2 of treatment predicted symptom change between Weeks 3 and 12 (Klein et al., 2003).

One role alliance may play, especially in relation to a confounding between specific treatment and alliance effects, is in enhancing engagement in treatment. In a treatment like CBT, this may simply translate to greater application of in-session or home assignments. An extended example may serve well to illustrate this point. Cloitre, Koenen, Cohen, and Han (2002) examined the pattern of symptom change in a treatment study where skills training in affective and interpersonal regulation were used as a prelude to exposure-based treatment for post-traumatic stress disorder (PTSD) in 58 women who had experienced childhood abuse. This study is of interest for understanding alliance effects in part because the participants – all

who had PTSD due to abuse at the hands of caregivers – were those who would be hypothesized to have alliance issues. First, PTSD-related anger symptoms have been linked to ruptures in the therapeutic relationship, poorer treatment outcome, and dropout (Chemtob, Novaco, Hamada, & Gross, 1997). Second, physical or sexual abuse at the hands of caregivers is associated with feelings of distrust and disconnection in adulthood (Briere, 1988; Cole & Putnam, 1992). Cloitre et al. (2002) assessed therapeutic alliance during sessions 3, 4, and 5 of the first (skill building) stage of treatment and found a link to a reduction in PTSD symptoms, at a level ( $r = .46$ ) that was approximately double the mean in meta-analyses (as reviewed earlier).

Indeed, in a subsequent analysis Cloitre, Stovall-McClough, Miranda, and Chemtob (2004) showed that the degree of improvement in emotional regulation skills mediated the prediction offered by alliance scores, such that the alliance scores offered no prediction when improvements in emotional regulation were considered. Interestingly, it was the change in emotional regulation across exposure that was submitted for analysis; that is, it is not clear whether training in emotional regulation was important for intensifying the alliance or leading to clinical change, as the relevant changes in emotional regulation were assessed across the exposure phase (Phase 2 of treatment) rather than the emotional regulation skill building phase (Phase 1 of treatment). Moreover, alliance ratings did not predict the 29% of patients who failed to complete treatment. What, then, is the meaning of alliance scores during Phase 1 of treatment? It is possible that, as the authors suggest, the degree of alliance during the first phase of treatment determined how well the patients were able to make use of the exposure phase of treatment. It may simply be that patients who agree with the goals and tasks of emotional regulation training are likely to give higher alliance ratings during Phase 1 of treatment and are likely to fully engage in and benefit from subsequent exposure treatment.

In summary, as noted in a review by Loeb et al. (2005), despite the consistency of findings that the therapeutic alliance is moderately correlated with treatment outcome across different types of treatment, the role of the alliance has not been reliably established as a causal mechanism of change. Alliance may result from, as much as cause, successful treatment effects. Nonetheless, there is some evidence for a mutually reinforcing cycle, where alliance may help patients engage in treatment, and likewise successful engagement may lead to desired outcomes and an enhanced alliance. One issue for the field, however, is that the research on alliance provides little guidance on what may be the most operative characteristic for enhancing outcomes. It may be tempting to assume that the more interpersonally focused the therapy, the more alliance issues will be addressed. However, this expectation is not confirmed by research. For example, in comparative treatment trials, CBT has been shown to have higher alliance scores than interpersonal psychotherapy (Raue et al., 1997) or transference-focused psychotherapy (Spinhoven, Giesen-Bloo, van Dyck, & Arntz, 2007). Moreover, if alliance is conceptualized as a concurrence between therapist and patient on the goals, tasks, and bonds in therapy, then efforts to improve alliance may presumably focus on any of these elements. In the following sections we thus consider strategies for enhancing the patient's alliance with the

therapist and the tasks of treatment. Concerning goals of treatment, therapists from a variety of therapeutic orientations assert the importance of actively working to align goals, intervening with the perceptions and hopes of the patient, so that they are in accordance with treatment methods (Conoley, Padula, Payton, & Daniels, 1994). In our discussion of these factors, we will consider the alignment of treatment goals both in terms of (1) motivation for treatment and (2) specific treatment expectancies.

## Motivation for Treatment

Patients enter treatment at varying stages of readiness for change (Maltby & Tolin, 2005), and may differ in their conceptualization of the mechanism of that change. For example, individuals with generalized anxiety disorder (GAD) identify their worry as problematic but may simultaneously view it as distracting and protective (e.g., avoidant worry; Borkovec & Roemer, 1995). In obsessive-compulsive samples, anticipatory fear toward exposure-based therapies is not uncommon. Franklin and Foa (1998) report that about 25% of patients with obsessive-compulsive disorder (OCD) refuse exposure plus response prevention (ERP) therapy. Moreover, the initiation of psychological treatment is often accompanied by ambivalence or opposition to the prospect of change (Miller & Rollnick, 2002).

Motivational interviewing (MI) is an intervention specifically designed to target and resolve feelings of ambivalence that lead to patient resistance (Miller, 1983). Client-centered theory is evident in MI, as the therapist must convey empathy and minimize patient-therapist conflict (Slagle & Gray, 2007). Where MI differs from client-centered approaches, however, is in the directive role of the therapist. The goal of a therapist using MI is to persuade rather than coerce the patient to choose goals that are consistent with his or her own values and motivations and are therefore more meaningful (Rubak, Sandboek, Lauritzen, & Christensen, 2005; Slagle & Gray, 2007).

As a brief intervention for a wide variety of alcohol and drug use problems, MI is a well-validated and useful intervention (Burke, Arkowitz, & Menchola, 2003). In its application to the treatment of anxiety disorders, MI has shown initial promise. Specifically in a pilot study, participants meeting diagnostic criteria for at least one anxiety disorder were randomly assigned to receive either group CBT for anxiety management with MI as a pre-treatment or CBT without pre-treatment (NPT) spanning across a 4-week time period (Westra & Dozois, 2006). Treatment compliance was measured by rates of attrition and homework completion as assessed by both patient and therapist. Administered at baseline and subsequent to the pre-treatment (or post NPT), the Anxiety Change Expectancy Scales (ACES; Dozois & Westra, 2005) was used to gauge an individual's expectancy for change in treatment and, more globally, MI efficacy. According to Miller and Rollnick (2002), proper MI strengthens positive expectancies for change by increasing optimism. High scores on the ACES were significantly correlated with improved symptoms across both groups. Results showed that participants in the MI pre-treatment condition exhibited greater homework compliance, greater numbers of sudden gains, and greater

positive expectancy relative to participants treated only with CBT. At 6-month follow-up, the gains made in managing symptoms of anxiety were maintained in both pre-treatment conditions.

Another study randomly assigned treatment-refusing OCD patients to a four-session readiness intervention (RI; a prelude treatment following the same principles as MI) or waitlist (WL) condition before being given the option to enter into 15 sessions of ERP (Maltby & Tolin, 2005). Subsequent to completing the pre-treatment, a greater proportion of patients in the RI condition accepted ERP compared to WL (86% vs. 20%). However, before beginning the exposure treatment, the two groups did not show any difference in symptom severity, indicating a differential effect of readiness training on treatment cooperation rather than efficacy for those who received the RI.

In addition to motivation for treatment, expectancies about the nature of treatment can have an impact on treatment acceptability and outcome. Some argue that expectancies in therapy are related to the general concept of hope (Frank & Frank, 1991; Snyder, Michael, & Cheavens, 1999). Hope is commonly defined as wishes or desires that are tied to an expectation of achieving desired results (Dew & Bickman, 2005). Snyder and colleagues argue that hope involves both conceptualizing specific goals based on one's own ability to work towards reaching the goals (pathway thinking), and also being able to continue to strive towards the goals (agency thinking). The model of hope put forth by Snyder, Ilardi, Michael, and Cheavens (2000) posits that an individual's perceived ability to begin and sustain movement toward a goal, combined with a belief in the pathways available to reach the goal, leads to positive outcomes in psychotherapy.

As compared to the more global concept of hope, expectancy effects have received more specific attention, with three types of expectancy effects commonly described in the literature: role expectancies, outcome expectancies, and control expectancies.

## **Role Expectancies**

In general, role expectancies refer to the patterns of behavior viewed as appropriate for a person in a specific role (Delsignore & Schnyder, 2007; Dew & Bickman, 2005). In a therapeutic context, this involves the expected roles of the patient and the therapist. Patients may have expectations regarding their role (e.g., expecting that there will be a collaboration in therapy) and the therapist's role (e.g., expecting to be given advice from the therapist) in the therapeutic process (Arnkoff, Glass, & Shapiro, 2002). Helping patients form expectations of an active treatment role for themselves may be particularly important, given the general association between homework adherence and treatment outcome for the anxiety disorders (Westra, Dozois, & Marcus, 2007), although not all studies have supported the association between greater homework compliance and more positive outcomes in individuals being treated with CBT (Woods, Chambless, & Steketee, 2002).

## Outcome Expectancies

Outcome expectancies (also referred to as treatment expectancies or prognostic expectations in the literature) describe how strongly a patient presumes that a given therapy will work (Arnkoff et al., 2002; Delsignore & Schnyder, 2007; Greenberg, Constantino, & Bruce, 2006). Outcome expectancies directly relate to the progress that a patient believes will be made in therapy (Dew & Bickman, 2005; Delsignore & Schnyder, 2007). This form of patient expectation has received the most empirical attention in the literature (Arnkoff et al., 2002; Greenberg et al., 2006).

Outcome expectancies may appear to be similar to other patient variables, such as motivation for treatment and therapy preferences, but these are distinct constructs, as it is possible for a patient to be highly motivated for therapy and still have low prognostic expectations that they will benefit from therapy (Arnkoff et al., 2002; Greenberg et al., 2006). Greenberg et al. differentiate between the types of outcome expectancies found in the literature (e.g., pre-treatment, during-treatment). Pre-treatment outcome expectancies refer to prognostic beliefs that a patient has prior to any contact with the therapist or initiation of treatment. During-treatment outcome expectancies are examined after a patient has already met with the therapist. Treatment credibility, a conceptually similar during-treatment construct, describes how logical, believable, and convincing a particular treatment is (Greenberg et al., 2006; Kazdin, 1979). Treatment credibility and treatment outcome expectancy are related, yet distinct constructs that have predicted therapeutic change across a wide range of studies (Borkovec & Costello, 1993; Chambless, Tran, & Glass, 1997; Devilly & Borkovec, 2000; Dew & Bickman, 2005; Goossens, Vlaeyen, Hidding, Kole-Snijders, & Evers, 2005). It is still unclear exactly how these constructs are related to change; however, burgeoning research strongly suggests a link to psychotherapy outcome.

## Empirical Support for Patient Expectancies

A recent review of 35 studies investigated the relationship between specific patient expectancies (e.g., role expectancies, outcome expectancies, and control expectancies) and outcomes in therapy (Delsignore & Schnyder, 2007). Overall, the authors concluded that there was a modest direct relationship between specific expectancies and improvement in therapy, but there were inconsistent results relating to global expectancies and therapy outcome. Findings varied widely among the studies specifically examining outcome expectancies, but generally reflected modest effects, indicating that patients with higher positive outcome expectancies seemed to benefit more from psychotherapy than patients who lacked confidence in the therapy. Studies investigating role expectancies in therapy produced inconsistent results, with four out of six studies reporting a positive association between a patient's expectancy about their role and therapy outcome across diverse treatment approaches (Delsignore & Schnyder, 2007; see also Arnkoff et al., 2002).

In another review examining both adult and child studies in the expectancy literature, Dew and Bickman (2005) found a positive relationship between outcome expectancies and patient improvement (with 10 out of 13 studies reporting a significant relationship), but the authors did not find a relationship between expectancies and attrition. A complex association between expectancies and attrition was also reported in a study by Nock and Kazdin (2001) who investigated parents' pre-treatment expectancies for their child's psychotherapy in a large sample of children with oppositional, aggressive, and antisocial behavior. Treatment included parent management training as well as cognitive problem-solving skills training directed to the child. Although, as might be expected, lower parental expectancies for treatment predicted higher barriers to treatment, it was the parents with very high or very low expectancies for treatment who attended the most therapy sessions and were least likely to discontinue treatment. The authors explain that individuals with very high expectancies for therapy might be more likely to attribute changes in therapy to the treatment itself. Alternatively, individuals with very low expectancies may be less likely to expect any positive changes resulting from the therapy, and are therefore more likely to increase expectancies when observing any therapeutic gains, leading to continued treatment attendance.

Linehan, Cochran, Mar, Levensky, and Comtios (2000) provide at least one caution to the role of high expectancies. Individuals with borderline personality disorder, a disorder characterized by marked interpersonal challenges, have been shown to be more prone to burnout when therapeutic expectations are high.

However, it is important to note that pre-treatment expectancies may give only part of the picture of the relationship between treatment expectancies, treatment engagement, and improvement. Once therapy has begun, individuals may be more likely to continue in therapy and achieve greater gains if their treatment expectancies match the capabilities of therapy, and they believe that the given treatment is worthwhile (Frank & Frank, 1991; Garfield, 1994). In support of these hypotheses, Westra et al. (2007) tested the assertion that positive expectancy can lead to greater compliance in CBT, and influence subsequent outcomes. Their study examined the relationship between pre-treatment expectancies for anxiety change and early homework compliance, and cognitive symptom change (initial change and total change) throughout treatment. Westra and colleagues found that early homework compliance mediated the relationship between expectancy for anxiety change assessed at baseline and initial change in CBT. Results also indicated that homework compliance was positively correlated to overall treatment change, an effect that was mediated by initial symptom change. In other words, initial cognitive symptom improvement mediated the relationship between homework compliance and post-treatment outcome. The authors suggest that expectancy for change is an important cognitive variable that may serve to bolster the momentum for involvement in therapy, thus contributing to subsequent gains. They also propose that the dynamics among expectancy for change, homework compliance, initial symptom change, and overall treatment outcome might reflect a reciprocal relationship. Although not directly tested in the present study, Westra et al. posit that this (reciprocal) pathway may go through multiple iterations. For example, if expectancy influences homework

compliance, homework compliance contributes to initial symptom change, and early symptom change reinforces expectancies, the process could go on and on.

## **Changing Expectancies**

Expectancies can be altered by adding pre-treatment interventions that provide information about the therapy. In several studies, pre-treatment preparation has been shown to affect both role and outcome expectancies. For example, Bonner and Everett (1982) found that children who listened to an audiotape designed to prepare them for treatment had more positive outcome expectancies and more appropriate role expectations than did the group that was denied pre-treatment information. These findings were later replicated with both children and their parents (Bonner & Everett, 1986). Preparatory videos and instructional brochures have also been shown to affect expectancies (Day & Reznikoff, 1980; Shuman & Shapiro, 2002).

## **Post-treatment Expectancies: Attributions About Treatment Gains**

Treatment expectancies and attributions are also relevant to the post-treatment period. Research indicates that how patients view the outcome of psychotherapy impacts rates of relapse. There is evidence of a higher risk for relapse when a patient makes external attributions (i.e., external locus of control) for success in therapy because they believe that the therapy worked as a result of therapist skills or powerful treatment techniques (for review see Brewin & Antaki, 1982). Likewise, Brewin and Antaki argue that patients who attribute gains to their own efforts are more likely to maintain treatment gains than patients who attribute their improvement to external factors (e.g., a drug's action or a therapist's abilities).

This argument is well supported in the anxiety disorders. For example, in a recent study, Powers, Smits, Whitley, Bystritsky, and Telch (2008) actively manipulated attributional processes concerning medication compliance on return to fear following an exposure-based intervention for individuals with claustrophobia. Participants were randomized to a waitlist control condition, a psychological placebo condition, a single session exposure-based treatment, or a single session exposure-based treatment in conjunction with an inactive pill. Participants in the exposure plus inactive pill group were either led to believe that the pill they were given was a sedating herbal supplement with anxiety-dampening effects, a stimulating herbal supplement with anxiogenic effects, or a placebo pill that did not effect exposure treatment. By manipulating patients' perceptions of the pill's effects, the researchers were able to achieve differential attributions to treatment improvement and return of fear. Results indicated that participants who were led to believe that they took a sedating herb rated the pill as being more helpful than those who were told that they had taken the stimulating herb or a placebo. Additionally, participants who believed that

they had taken an herbal stimulant (that made the exposure more difficult) rated the pill as hindering their treatment more than the other groups did. As predicted, participants who were told after treatment that they had taken a sedating herb that dampened their anxiety had a significantly higher return of fear at a 1-week follow-up. Moreover, at follow-up, participants in the exposure plus sedating herb condition no longer outperformed the waitlist or psychological placebo condition, while the other three exposure conditions (e.g., exposure alone, exposure plus a pill described as a stimulating herb, and exposure plus a pill described as a placebo) maintained improvement over the waitlist and psychological placebo conditions. These findings are consistent with previous correlational studies that found that individuals who attributed their improvement to a medication were at risk of poorer maintenance of treatment gains (Basoglu, Marks, Kilic, Brewin, & Swinson, 1994; Biondi & Picardi, 2003).

In one of these correlational studies, Basoglu et al. (1994) found that attribution of improvement to medication (alprazolam or placebo) predicted relapse in patients with panic disorder with agoraphobia who were also being treated with a psychological intervention (exposure or relaxation). Patients who attributed gains to their own efforts maintained those gains at a 10-month follow-up better than those who attributed their improvements to external factors (medication or the therapist). Patients had more severe withdrawal symptoms during tapering if they had strongly believed that it was the medication that helped them. Patients who were given alprazolam made more external (drug) attributions than those who were given the placebo medication. Moreover, patients who made self-attributions were able to make more outings into phobic situations during tapering than those who attributed improvement to the medication.

In a subsequent study, Biondi and Picardi (2003) supported the relationship between attribution of improvement to medication and risk of relapse in individuals with panic disorder with agoraphobia. Patients from a prior study who were able to achieve remission after medication treatment, in conjunction with or without CBT, became participants in this follow-up study. After patients had reached a satisfactory level of improvement, medication was gradually tapered over 1–2 months, and the follow-up period ranged from 2 to 86 months. Results indicated that patients who attributed their improvement to the medication were at a greater risk of relapse.

In a randomized controlled trial, Livanou et al. (2002) examined post-treatment beliefs, sense of control, and treatment outcome in PTSD. The interventions included exposure (five sessions of imaginal exposure and five sessions of graded live exposure), cognitive restructuring (CR; ten sessions), both in combination (five sessions of CR plus imaginal exposure and five sessions of CR plus live exposure), or a placebo condition (ten sessions of deep muscle relaxation). In line with previous studies that found external attribution of improvement to generalize less at post-treatment (for data on other disorders see Davison, Tsujimoto, & Glaros, 1973; Jeffrey, 1974; Sonne & Janoff, 1979), Livanou and colleagues found that maintenance of PTSD treatment gains at follow-up were predicted by post-treatment sense of control over symptoms and internal attribution of gains. However, baseline beliefs and improvement of beliefs were not predictive of outcome, an unexpected finding

that differed from previous studies on PTSD (Ehlers, Mayou, & Bryant, 1998), agoraphobia (Chambless & Gracely, 1989), and OCD (Basoglu, Lax, Kasvikis, & Marks, 1988), leading the authors to conclude that the divergent results may be due to methodological differences between studies.

The dramatic attribution effects found by Powers et al. (2008), Basoglu et al. (1994), and Biondi and Picardi (2003) have important implications for combined drug-psychosocial treatments. Working with treatment attributions and the role of (drug) context effects in therapy is discussed in more detail in “Combined Cognitive Behavioral and Pharmacologic Treatment Strategies: Current Status and Future Directions” by Smits et al. (this volume).

## **Treatment Adherence**

Treatment adherence, or integrity, is described as the extent to which a set of prescribed intervention principles and techniques are followed by the therapist (Waltz, Addis, Koerner, & Jacobson, 1993). A recent study by Hogue et al. (2008) examined adolescents with behavioral problems receiving CBT, focusing on treatment adherence as a main effect. Results indicated that intermediate levels of adherence were associated with the greatest improvements, above and beyond the effects of rigid and low adherence levels. In another study, participants low in motivation receiving manualized CBT had poorer treatment outcome, while outcome was unrelated to high protocol adherence in participants low in motivation (Huppert, Barlow, Gorman, Shear, & Woods, 2006). One interpretation of these findings is that with high motivation, stricter adherence may better match the level of motivation and expectancies of patients, providing additional circumstantial support for the importance of managing expectancies throughout the therapy.

Efforts to strengthen alliance also appear to offer therapeutic benefit should therapeutic relationship issues arise. Ruptures in therapeutic alliance, marked by difficulty sustaining the relationship and an overall negative shift in quality, have received considerable attention in alliance research (Strauss et al., 2006). Such a problem may occur when a therapist does not attend to signs of relationship strain (Safran, 1990). There is some evidence that alliance ruptures should be perceived as opportunities to facilitate therapeutic change, whereby maladaptive patterns are brought to attention and acknowledged. For example, Strauss and colleagues measured rupture-repair episodes in a sample of avoidant and obsessive-compulsive individuals receiving cognitive therapy. Episodes were quantified by a decrease of 7 or more points on the California Psychotherapy Alliance Scale (CALPAS; Marmar, Weiss, & Gaston, 1989) followed by an increase of at least 7 points. Of the patients that experienced a rupture-repair episode, a large portion evidenced greater symptom reduction across treatment measured by both self-report (Wisconsin Personality Disorder Inventory; Klein et al., 2003) and clinician-rated assessments (Structured Clinical Interview for DSM-III-R – Personality Disorders;

Spitzer, Williams, Gibbons, & First, 1990). In effect, ruptures in alliance may have a therapeutic effect if adequately managed and repaired.

## Summary and Conclusions

The available evidence suggests that common factors in therapy – variables defined as therapeutic alliance, motivation, and expectancies – have fairly reliable albeit limited effects on treatment outcome. As reviewed above, these effects have been shown to be important for enhancing CBT outcome in many cases, but do not account for CBT outcome effects. In this chapter, the degree of alliance, motivation, and expectancies in therapy have been treated as both relevant and modifiable attributes. The available evidence suggests that part of the task of the CBT therapist treating anxiety disorders should be: (1) helping patients clarify goals and values before treatment, (2) establishing a link between these general goals and values and the specific goals and methods of treatment, and (3) monitoring and working to enhance the ongoing congruence of these factors in the therapeutic relationship. Working to enhance these common treatment factors, while offering empirically supported CBT strategies, might provide an especially powerful approach to helping patients in need.

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# Combined Cognitive Behavioral and Pharmacologic Treatment Strategies: Current Status and Future Directions

Jasper A.J. Smits, Hannah E. Reese, Mark B. Powers, and Michael W. Otto

This chapter provides a review of the current status of traditional combined treatment strategies: co-application of cognitive-behavioral therapy (CBT) and pharmacotherapy, most typically, with antidepressant or benzodiazepine medications. The success of these strategies, as judged from the perspective of randomized clinical trials and naturalistic case series, will be reviewed and followed by an account of some of the issues and complexities underlying the limited treatment efficacy of this approach. Guidance is provided to clinicians on some of the contextual factors that may hinder CBT efficacy in the context of co-occurring medication treatment. Furthermore, we discuss the theory and potential for new combination treatment strategies that avoid some of the issues associated with current approaches and which rely much more on maximizing the core therapeutic learning offered by exposure-based CBT approaches.

## The Efficacy of Cognitive-Behavioral and Pharmacological Treatments

A large body of work indicates that cognitive-behavioral and pharmacological treatments are both effective interventions for the acute-phase treatment of anxiety disorders. Cognitive-behavioral therapy (CBT) is a learning-based approach aimed at helping patients reacquire a sense of safety around cues associated with anxiety and panic. To achieve this type of learning, CBT protocols, usually 12–15 weeks in length, emphasize education about anxiety psychopathology as well as repeated exposure to fear-eliciting cues, often in combination with restructuring of false threat appraisals. For example, a patient suffering from panic disorder who fears that panic attacks will cause her to faint (i.e., false threat appraisal) may be provided with education about the physiology of panic to help her understand that dizziness (i.e., fear-eliciting cue) is a natural and harmless response to overbreathing.

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A review of previous personal experiences or experiences of others with dizziness may provide further disconfirmatory evidence (e.g., “the chances of fainting when feeling dizzy are slim;” “I have fainted before, but never did it cause any significant harm”). Finally, repeated interoceptive exposure to activities such as chair spinning or hyperventilation (activities which induced the feared sensations) offers the opportunity for the fear of dizziness to be confuted and ultimately extinguished. From a neurobiological perspective, Gorman, Kent, Sullivan, and Coplan (2000) proposed that CBT deconditions contextual fear at the level of the hippocampus and enhances the ability of the prefrontal cortex to inhibit the amygdala.

A recent meta-analysis of randomized placebo-controlled trials indicated that CBT protocols of this nature are associated with clinically meaningful improvements at the conclusion of acute-phase treatment across the anxiety disorders (Hofmann & Smits, 2008). Although long-term follow-up studies are sparse, the available data suggest that the gains achieved with CBT can be durable over time (Gould, Buckminster, Pollack, Otto, & Yap, 1997; Gould, Otto, & Pollack, 1995; Gould, Otto, Pollack, & Yap, 1997; Otto, Penava, Pollack, & Smoller, 1996). However, many patients do seek further treatment, either because they remain symptomatic or relapse over follow-up periods (e.g., Brown & Barlow, 1995).

In contrast to CBT, pharmacological interventions aim to directly target biochemical pathways underlying the anxiety elicited by disorder-specific cues (e.g., interoceptive cues for panic disorder patients or social scrutiny for social anxiety disorder) by decreasing activity in the amygdala (Gorman et al., 2000). Pharmacological agents that have demonstrated efficacy for at least one anxiety disorder include selective serotonin reuptake inhibitors (SSRIs), monoamine oxidase inhibitors (MAOIs), tricyclic antidepressants, and benzodiazepines (Abramowitz, 1997; Lydiard, Brawman-Mintzer, & Ballenger, 1996; Mitte, Noack, & Hautzinger, 2005; Pollack, 2005; van Etten & Taylor, 1998). Treatment-free follow-up data suggest that pharmacotherapy is a long-term commitment; relapse is common following medication discontinuation (e.g., Mavissakalian & Perel, 1992; Noyes, Garvey, Cook, & Suelzer, 1991; Stein, Versiani, Hair, & Kumar, 2002; Walker et al., 2000).

In response to the desire for stronger acute and longer-term outcomes for anxiety treatments, one natural inclination is to combine the outcomes offered by pharmacological and CBT interventions.

## **The Efficacy of Acute-Phase Combined Treatments**

The efficacy of combined CBT and pharmacotherapy relative to either treatment delivered alone has been studied most extensively in the treatment of panic disorder (PD), and to some extent in obsessive-compulsive disorder (OCD) and social anxiety disorder (SAD). Limited data are available for generalized anxiety disorder (GAD), and at the time of this writing, there was only one published report evaluating sequential combined treatments for post-traumatic stress disorder (PTSD) or acute stress disorder (ASD). Overall, the results of the available studies are mixed and seem to vary somewhat as a function of the target disorder,

the prescribed pharmacological agent, as well as the time of assessment (i.e., acute vs. follow-up outcomes; see for review Foa, Franklin, & Moser, 2002; Otto, Smits, & Reese, 2005).

With respect to acute outcomes (i.e., assessment conducted immediately following acute-phase therapy), studies involving PD samples converge to suggest that combining CBT with medications may offer an advantage over either modality alone. A recent meta-analysis of 21 trials examining the efficacy of combined treatment for panic disorder yielded a slight advantage of combined treatments over CBT (relative risk of response (RR) = 1.16) and pharmacotherapy (RR = 1.24; Furukawa, Watanabe, & Churchill, 2006).

Several studies indicate that combination approaches may also offer advantages in the acute phase over single-modality interventions for the treatment of OCD. Early studies conducted by Marks, Stern, Mawson, Cobb, and McDonald (1980) and Marks et al. (1988) indicated that the combination of exposure and response prevention (ERP) with clomipramine, a tricyclic agent, resulted in greater improvements compared to ERP plus placebo or clomipramine plus relaxation or clomipramine plus anti-exposure instructions. Short-term advantages of combination treatments for OCD were also observed in two studies comparing the single and combined effects of fluvoxamine, an SSRI, and ERP (Cottraux et al., 1990; Hohagen et al., 1998). Of note, these two studies also provided evidence to suggest that the advantage of combined treatment was particularly evident among patients who presented with comorbid depression. Hohagen et al. (1998) suggested that the antidepressant properties of the medication may successfully reduce the depressive symptoms which enable individuals to more effectively engage in and benefit from ERP.

Findings of a recent randomized placebo-controlled trial (Foa et al., 2005) suggest that combining clomipramine with exposure and ritual prevention (EX/RP) in the acute phase may *not* be cost-effective when the EX/RP treatment is delivered more intensely (i.e., 15 2-h sessions over 3 weeks). Although the combination treatment yielded greater response rates than clomipramine alone (i.e., acute-phase response rates 70% vs. 48%), the response rates for EX/RP alone were not significantly different than for the combination treatment (i.e., 62% vs. 70%). Unfortunately, patients with comorbid depression were excluded from this trial, which, in addition to the EX/RP delivery schedule, perhaps limits the generalizability of the study results to all patients. Nonetheless, the Foa et al., (2005) findings in relation to those from Hohagen et al. (1998) support the notion that combined treatment strategies for OCD, relative to CBT alone, may have limited efficacy outside patients' samples with comorbid depression and/or clinical settings where intensive CBT can be offered.

Limited positive effects of combination treatments have been observed for the treatment of SAD. In a randomized placebo-controlled study, Blomhoff et al. (2001) examined the single and combined effects of sertraline, an SSRI, and physician-guided exposure therapy for SAD. Response rates at the end of acute-phase treatment were 45.5%, 40.2%, 33%, and 24% for the sertraline plus exposure, sertraline, exposure plus placebo, and placebo conditions, respectively. These findings suggest that sertraline can augment the effects of exposure treatment, particularly

when exposure treatment is delivered with minimal therapist contact (i.e., eight 20-min sessions involving instructions for homework exposures). However, these benefits of combined treatment, relative to CBT alone, were no longer evident at a 1-year follow-up evaluation (Haug et al., 2003). Moreover, consistent with findings from Foa et al. (2005) for OCD, antidepressant medication treatment does not appear to augment the acute effects of more comprehensive programs of CBT for SAD. Davidson et al. (2004) compared the outcome of five treatment conditions: group CBT, fluoxetine (an SSRI), pill placebo, group CBT plus fluoxetine, and group CBT plus placebo among individuals with generalized SAD. Fluoxetine alone was associated with the strongest initial response at week 4, but the active treatments were all significantly better than placebo and not significantly different from each other at week 14, with response rates of approximately 50% in each of the conditions.

Based on the available data as well as cost/benefit considerations (see Otto, Pollack, & Maki, 2000; McHugh et al., 2007), it appears that there is no strong justification for recommending that combined treatment be adopted as a standard, first-line treatment for optimizing acute outcomes for the anxiety disorders. Whether combination treatments are indicated for certain subgroups of patients with anxiety disorders (e.g., those with comorbid depressive disorders) or in settings where CBT cannot be delivered in its most optimized form (e.g., primary care) are questions that deserve further inquiry.

## **Outcomes After Treatment Discontinuation**

Certainly, a case can be made for approaching the treatment of anxiety disorders as a chronic illness (see Bruce et al., 2005), requiring long-term interventions (i.e., continuation-phase treatment) following acute-phase therapy (Mavissakalian & Prien, 1996). Today's practice of pharmacological and CBT interventions for the anxiety disorders, however, appears to reflect an approach that is more in line with treating anxiety psychopathology much like acute infectious illness. Indeed, CBT is a short-term approach focusing mostly on the acute management of anxiety and related avoidance. Likewise, although a long-term proposition, patients often fail to adhere to pharmacological prescriptions over the long run (Cowley, Ha, & Roy Byrne 1997; Sirey et al., 1999; Weilburg, O'Leary, Meigs, Hennen, & Stafford, 2003). Given this reality, it is important to examine outcomes of combination treatments following treatment discontinuation.

Studies examining long-term outcomes following the discontinuation of acute-phase combination treatments are limited in number. The available evidence suggests that, if at all evident during the acute phase, advantages of combined treatments are lost during treatment-free follow-up among patients with OCD (Cottraux, Mollard, Bouvard, & Marks, 1993; Marks et al., 1988) or SAD (Haug et al., 2003). Findings from two large multi-center trials suggest that combining medication and CBT in the acute-phase treatment of panic disorder may actually be associated with poorer long-term outcomes compared to those conferred by CBT alone. Marks et al. (1993) randomly assigned patients to receive alprazolam plus exposure, alprazolam

plus relaxation, placebo plus exposure, or placebo plus relaxation (double placebo). The authors found that although combined treatment was associated with a modest advantage over monotherapy in the acute phase, it resulted in less improvement relative to exposure alone at treatment-free follow-up. Similarly, Barlow, Gorman, Shear, and Woods (2000) found that a combination of imipramine plus CBT was slightly more efficacious than monotherapy at post-treatment, but after medication discontinuation, the combined treatment was associated with the highest relapse rates. These studies are of particular concern because they suggest that medication treatment may actually weaken the effect of CBT over time after patients discontinue treatment. In the next section, we discuss some possible mechanisms that may account for the deleterious effects of medication on the long-term effects of acute-phase CBT.

### **Possible Mechanisms of Relapse Following Discontinuation of Combined Treatments**

There are several possible reasons for the increased risk of relapse following termination of combined exposure-based and pharmacological treatment (see for review, Powers, Smits, Leyro, & Otto, 2007). Animal work on extinction has offered one explanation (see Bouton, 2002). Specifically, animal studies have shown that extinction learning, which involves procedures similar to exposure-based treatments, is context-specific (Bouton, 2002). That is, extinction of fear that occurs in one context (e.g., room A) may not generalize to a second context (e.g., room B). Accordingly, context shifts such as medication discontinuation (i.e., the drug-state is withdrawn) may account for the loss of gains apparent during acute-phase treatment (i.e., the extinction memory is specific to the state of being on medication). Mystkowski, Mineka, and Vernon (2003) tested this hypothesis using a sample of spider-fearful participants. They randomly allocated these participants to ingest either caffeine or a pill placebo before receiving treatment involving exposure to live spiders. Outcome was assessed by means of a behavioral approach task immediately following treatment completion. To test the hypothesis that extinction learning during CBT is context-specific, and thus would be lost as a result of changing the drug state, the investigators retested participants 1 week following treatment under conditions of either the same or opposite drug context. Consistent with predictions, participants who were randomly allocated to be tested under the incongruent condition (e.g., treated while taking caffeine and later tested while taking placebo) displayed greater return of fear compared to those tested under the congruent condition (e.g., treated while taking caffeine and later tested while taking caffeine).

Self-efficacy theory (Bandura, 1977) offers a possible alternative or complementary mechanism underlying the negative effects of medication on long-term effects of CBT. Self-efficacy theory posits that phobic behavior is a function of one's perceived inability to execute effective coping behavior in response to potential phobic threats (Bandura, 1977). It seems plausible that medication-taking may cause patients to attribute their gains to the medication instead of their own efforts and accomplishments, thereby undermining self-efficacy enhancement. Indeed, patients

with anxiety disorders tend to attribute treatment gains to external factors (Adler & Price, 1985; Anderson & Arnoult, 1985; Broadbeck & Michelson, 1987; Cloitre, Heimberg, Liebowitz, & Gitow, 1992; Emmelkamp & Cohen-Kettenis, 1975; Hoffart & Martinsen, 1990). The influence of patients' attributions of treatment gains to medication on the outcome of combined treatments has been examined in a few studies. Basoglu, Marks, Kilic, Brewin, and Swinson (1994) reported that attributions of improvement to the medication (i.e., alprazolam or placebo) significantly predicted relapse in panic disorder patients treated with exposure in combination with medication (Basoglu et al.). Interestingly, Biondi and Picardi (2003) reported that making external/medication attributions was associated with a 60% relapse rate, whereas making internal attributions was associated with a 0% relapse rate.

Perhaps the strongest evidence for the causal role of external attributions in relapse following the discontinuation of combined treatments comes from a recent analogue study by Powers, Smits, Whitley, Bystritsky, & Telch (2008). Using an experimental design, the investigators first randomly assigned participants displaying marked claustrophobic fear to one of four conditions: (1) waitlist; (2) psychological placebo; (3) exposure-based treatment, (4) exposure-based treatment plus an inactive pill. Following post-treatment assessment that revealed an advantage of exposure over control conditions and no effects of pill taking, they manipulated attributions concerning medication taking by randomly assigning participants in the exposure-based treatment plus pill placebo condition to one of three instructional sets: (1) the pill was described as a sedating herb that likely made exposure treatment easier; (2) the pill was described as a stimulating herb that likely made exposure treatment more difficult; or (3) the pill was described as a placebo that had no effect on exposure treatment. Assessments at follow-up showed a relapse rate of 39% among participants who believed that the pill had a sedating effect, whereas a relapse rate of 0% was observed among participants in the other two conditions involving medication taking. Moreover, reduced self-efficacy accounted for the elevated relapse rates associated with the sedating instructional set.

Collectively, these findings suggest that the practice of acute-phase combined treatments may warrant the utilization of specific relapse prevention strategies, including the assessment and possible modification of patient attributions regarding the improvements achieved with treatment as well the continuance of exposure practice following the termination of medication treatment. In the next section, we discuss the evidence for this approach of offering sequential as opposed to concurrent combined interventions.

## **Starting CBT as a Strategy for Discontinuation of Pharmacotherapy**

There is evidence that when CBT is continued or reinstated during medication discontinuation, it may help patients maintain their treatment gains. For patients who have received CBT concurrently with medication, continuing CBT treatment during and after the taper period may help the individual extend their learning to a

medication-free context. Additionally, CBT has been found effective in helping patients tolerate the discontinuation symptoms of benzodiazepine taper in PD (Hegel, Ravaris, & Ahles, 1994; Otto et al., 1993; Spiegel, Bruce, Gregg, & Nuzzarello, 1994) and GAD (Gosselin, Ladouceur, Morin, Dugas, & Baillaregon, 2006). For example, Gosselin et al. (2006) found that patients who received CBT during benzodiazepine taper were more successful at stopping the medication and also had lower relapse rates relative to those individuals who received a control treatment emphasizing active listening. Similarly promising findings have been reported for the addition of CBT during SSRI discontinuations in panic disorder (Whittal, Otto, & Hong, 2001; Schmidt, Wollaway-Bickel, Trakowsky, Santiago, & Vasey, 2002).

### **Starting CBT when Pharmacotherapy Fails**

CBT has been shown to be an effective treatment for individuals who have failed to respond to medication treatment for PTSD (Otto et al., 2003), OCD (Kampman, Keijsers, Hoogdiun, & Verbraak, 2004), and PD (Otto, Pollack, Penava, & Zucker, 1999; Pollack, Otto, Kaspi, Hamerness, & Rosenbaum, 1994; Heldt et al., 2006). For example, Heldt et al. (2006) offered 12 weekly sessions of group CBT to individuals with panic disorder who had remained symptomatic despite an average of 3 years of pharmacotherapy. At 1-year follow-up, nearly two-thirds of the participants met remission criteria. Given that psychiatrists and primary-care physicians greatly outnumber psychologists trained in CBT, the most readily available treatment for the anxiety disorders is often medication. Thus, the use of CBT as a treatment strategy for individuals who have not benefited from medication may be a practical solution when treatment resources are limited.

### **Starting Pharmacotherapy when CBT Fails**

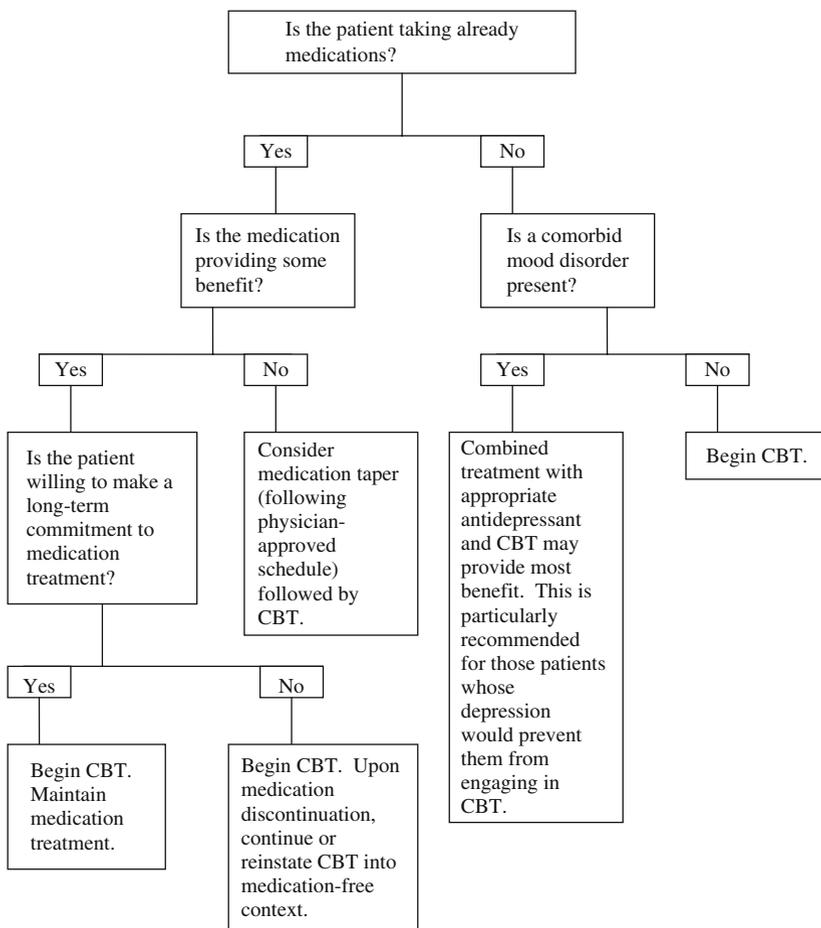
There is also some evidence that pharmacotherapy may be effective when CBT fails. For example, Kampman, Keijsers, Hoogduin, and Hendriks (2002) randomly assigned individuals with panic disorder who remained symptomatic after 15 sessions of CBT to continued CBT plus paroxetine, or CBT plus placebo. Those subjects who received the paroxetine experienced significant additional improvement on measures of avoidance and anxiety whereas those who received the placebo did not.

### **Clinical Considerations**

Our review of studies examining combined treatment strategies for the anxiety disorders suggests that it is too early to draw firm conclusions and to set specific clinical guidelines. Although the evidence to date provides no justification for recommending combined treatments as a first-line intervention for the anxiety disorders, it does not rule out that the combined approach holds great value for many patients suffering

from anxiety disorders. It appears that the utility of combined treatments may vary depending on the combination of a number of factors, including, but not limited to, patient preferences with respect to pharmacotherapeutic approaches, availability of state-of-the-art CBT, and the presence of comorbid conditions at presentation. In this section, we discuss some considerations with respect to the prescription of combined intervention approaches for anxiety disorders (see Fig. 1).

Many patients seeking psychotherapy for anxiety are already taking medication when they arrive for treatment (Roy-Byrne et al., 2002; Taylor et al., 1989). As such, the question of whether or not to provide CBT along with pharmacotherapy is one that is often presented to clinicians working with patients suffering from anxiety disorders. During the initial evaluation, the clinician should obtain



**Fig. 1** Considerations with respect to the prescription of combined interventions for the acute treatment of anxiety disorders

information about the prescribed medications the patient is taking. In addition to the type and dosing of the medication, the clinician should assess the degree to which the patient has received benefits from taking the medication. In this context, it is important to consider the effects of the medication on problems often comorbid with anxiety disorders. Indeed, suboptimal outcomes after CBT (e.g., response, attrition) may be particularly common among adults who present with anxiety disorders and are also diagnosed with unipolar depression (e.g., MDD, dysthymia; Lincoln, Rief, Hahlweg, et al., 2005; Rief, Trenkamp, Auer, & Fichter, 2000; Steketee, Chambless, Tran, 2001). Accordingly, the extent to which ongoing pharmacotherapy effectively manages depression in addition to anxiety is an important factor to consider in deciding to either continue or discontinue medications during acute-phase CBT.

Equally important in this decision is the preference of the patient with respect to continued pharmacological treatment. Irrespective of the benefits of pharmacotherapy, many patients may wish to stop taking medications during the course of CBT. Some patients may indicate that stopping medications is necessary (e.g., women who wish to become pregnant, patients who experience severe side effects); others may indicate that taking medications over longer periods of time is not consistent with personal values (e.g., patients who do not like being “dependent” on medications). When the patient wishes to discontinue pharmacotherapy, the clinician should consult with the patient’s physician and determine a tapering schedule appropriate for the prescribed medication. An important aspect of medication discontinuation is preparing the patient for the potential consequences of stopping pharmacotherapy. In addition to the possible relapse of mood and anxiety episodes that can occur with medication discontinuation, the tapering of medications, particularly with benzodiazepines, is associated with the emergence of a host of physical sensations. These withdrawal symptoms present an additional challenge to patients with elevated levels of anxiety sensitivity (i.e., fear of anxiety and related sensations; Reiss & McNally, 1985), as they are more likely to respond with anxiety or panic when exposed to physical sensations.

In order to assist patients in medication discontinuation, the clinician can offer a combination of informational (e.g., education, cognitive restructuring) and behavioral (e.g., behavioral experiments, (interoceptive) exposure) interventions designed to correct false threat appraisals as well as to enhance tolerance of physical sensations and negative affect (see Fig. 2). Manual-based guidance for both clinicians and patients is available for this task (Otto & Pollack, 2009 a,b). Because extinction learning may be context-specific, it is important to continue these interventions following the completion of the taper. This approach may also be beneficial for the patient who attributes the success of treatment to medication taking. Indeed, patients may be less likely to hold on to external attributions of therapy gains, and are therefore less likely to relapse, if they experience continued improvement with treatment following medication discontinuation.

What if the patient wishes to stay on medication? The appropriate proposed plan of action offered by the clinician depends on the extent to which the patient has received benefits from medication treatment. Clearly, the lack of substantial improvement following an adequate course of pharmacotherapy provides a rationale

- **Prepare patient for discontinuation through providing information (e.g., education, cognitive restructuring)**
- **Discuss physical withdrawal symptoms as interoceptive exposure**
- **Taper off medications slowly**
- **Apply/Reapply CBT during the taper**
- **Assess and modify external/medication attributions for improvement**

**Fig. 2** Strategies for patients wishing to discontinue medication

for discontinuation and subsequent application of CBT. However, clinicians must approach this process with appropriate caution, with sensitivity to the therapeutic relationship established between the patient and their prescribing physician. Education of both the patient and their previous provider about the options available for the next phase of treatment and the potential for CBT to aid with medication discontinuation may be important for successful intervention. The clinician could use a cognitive framework (i.e., with gentle challenging the patient's beliefs about medications and its effects) to present this proposed intervention plan.

If the medication has exerted positive effects, supporting the patient's preference to continue pharmacotherapy during acute-phase CBT is appropriate in many circumstances. For example, for patients with panic disorder or comorbid mood conditions, and in settings where conducting state-of-the-art CBT is not feasible, ongoing pharmacotherapy may assist the psychologist in achieving optimal outcomes with acute-phase CBT. To help patients attribute additional gains to their efforts in CBT, we recommend initiating CBT when a stable dose of medication over weeks has been achieved and combining CBT with close monitoring of symptoms. Therapy can then include a review of the link between the patients efforts in CBT and changes in symptom levels and the attainment of well being and enhanced role functioning. Also, clinicians need to recognize that many patients fail to adhere to long-term antidepressant treatments. Accordingly, the monitoring of medication adherence is critical and the clinician should be prepared to prolong or re-initiate therapy to allow the patient to extend their learning to a medication-free context.

## **Novel Combination Strategies**

We end this chapter discussing a recent development in the strategy of combining pharmacotherapy and CBT that may hold particular promise for the future. An accumulating body of work initiated by Davis and colleagues on D-cycloserine (DCS), a partial agonist at the glutaminergic *N*-methyl-D-aspartate (NMDA) receptor, suggests that DCS may be an effective augmentation strategy for exposure-based

CBT. Prompted by animal studies implicating a critical role for NMDA receptors in extinction learning (Falls, Miserendino, & Davis, 1992), Davis and others investigated the effects of DCS on extinction learning in rats (cf. Davis, Ressler, Rothbaum, & Richardson, 2006). The results of these studies converge to suggest that DCS facilitates extinction consolidation in animals (Davis et al., 2006). Following this work, initial studies with humans indicate that the administration of DCS prior to exposure therapy sessions enhances treatment outcome among patients with height phobia (Ressler et al., 2004, social phobia (Hofmann et al., 2006), and obsessive-compulsive disorder (Kushner, et al., 2007; Wilhelm, et al., 2008; for review of effect sizes see Norberg, Krystal, & Tolin, 2008). These exciting findings await replication and extension to other anxiety disorders, but they do suggest that a new approach to combined treatment – where the pharmacotherapy element is targeted to memory enhancement rather than affect management – may offer a new perspective on strategies for enhancing CBT efficacy. Research on other mechanisms for enhancing therapeutic learning from exposure-based strategies are also underway (Cain, Blouin, & Barad, 2003; Powers, Smits, Otto, Sanders, & Emmelkamp, 2009), and together these approaches represent a particular achievement of translational research, and a promising new approach for extending the efficiency or efficacy of CBT for the anxiety disorders (Anderson & Insel, 2006).

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# Cultural Considerations and Treatment Complications

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## Introduction

As ethnic and racial minority groups continue to grow (United States Bureau of the Census, 2005), the field of clinical psychology must meet mental health needs in a culturally appropriate manner. Despite the acknowledgement of the need for culturally relevant responses, the Surgeon General reports that ethnic minority members: (1) have less access and availability to mental health services, (2) are less likely to receive needed mental health intervention, (3) often receive poorer quality of care, (4) experience stigma associated with seeking treatment, and (5) are not always able to be matched with a practitioner that shares their language (U.S. Department of Health and Human Services [USDHHS], 2001). Unfortunately, data also suggest that ethnic and racial minority members experience higher poverty rates and greater incidence of related social stressors that negatively impact psychological functioning than their Caucasian counterparts (Mays & Albee, 1992; USDHHS). In addition, explicit conflicts between the cultural values of ethnic and racial minority clients and the mainstream values often exemplified in traditional psychotherapies introduce several treatment complications. In short, we are not adequately meeting the needs of our underrepresented populations.

Since psychotherapy is a cultural phenomenon, it follows that culture should play a substantial role in treatment (Bernal & Saez-Santiago, 2006; Bernal & Scharron-del-Rio, 2001). Historically, clinical research with cognitive-behavioral therapy (CBT) has largely focused on individuals of European-American descent (Bernal & Scharron-del-Rio, 2001) with relatively little attention given to the application of CBT techniques within various ethnic minority cultural groups (Bhugra & Bhui, 1998). As a result, there is a noticeable lack of concrete and organized intervention strategies and techniques that cognitive-behavioral clinicians can utilize when aiming to reduce treatment complications for their clients. At the same time, the tenets of CBT do implicitly accentuate the unique circumstances and experiences of the

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individual client (Hays & Iwamasa, 2006). As such, the basic techniques of CBT serve as an excellent foundation from which therapeutic approaches within clinical psychology can continue to be made more culturally responsive.

The purpose of this chapter is to provide evidence for the need and importance of considering culture in the conceptualization, diagnosis, and treatment of anxiety disorders among ethnic and racial minority clients. These factors will be reviewed through a discussion of some of the cultural issues specific to individuals of ethnic and racial minority groups, and also through an examination of how these issues might relate to culture-specific treatment complications and treatment resistance. This review is approached from the perspective that learning more about how the issues faced by this portion of our population might complicate clinical case conceptualization, diagnosis, and treatment and with a view that increasing our knowledge about these potential complications will, in turn, increase our ability to meet the psychological needs of members of these groups. Accordingly, the empirical research literature will be briefly reviewed to highlight the importance of these topics as they relate to the psychological well-being of ethnic and racial minority members and specific recommendations will be provided to assist clinicians working with clients from diverse populations.

## **Prevalence of Psychological Disorders Among Ethnic and Racial Minorities**

Inequities in the availability and accessibility of psychological assessment and treatment services combined with other factors, such as a general paucity of research relating to rates of psychological disorders among ethnic and racial minorities (USDHHS, 1999, 2001), make the task of efficiently and accurately examining prevalence rates of psychological disorders among various racial and ethnic groups complex. Some of this complexity relates to diagnosis, as members of some ethnic and racial minority groups are more likely to be over-diagnosed with particular types of psychological disorders. For instance, African-Americans tend to be over-diagnosed with schizophrenia (e.g., USDHHS, 1999). Similarly, among Latino/a groups, depressive disorders with psychotic features are also often misdiagnosed as schizophrenia (Lawson, 2003). On the other hand, members of some racial and ethnic minority groups might be subject to underdiagnosis of certain disorders. Indeed, among African-Americans, depression may be underdiagnosed (Skaer, Selar, Robison, & Galin, 2000).

In addition to problems related to misdiagnosis, members of some minority groups also appear more likely to present with a greater incidence of certain symptomology. For example, a significant proportion of ethnic and racial minority group members experience an elevated incidence of substance-related disorders (Thomason, 2000). Indeed, alcoholism among Native-American populations occurs at nearly twice the rate of that noted among any other population group (USDHHS, 1999). Incidence of suicide among this group is also staggering; with a rate that is

consistently 50% higher than the national average (USDHHS, 2001). Moreover, use and abuse of illicit substances is higher among African-American groups than rates found within any other racial group (USDHHS, 2000). Specific examples related to anxiety disorders are highlighted by Zhang and Snowden (1999), who found that African-Americans were significantly more likely than Caucasians to have a phobic disorder, and Glover, Pumariega, Holzer, Wise, and Rodriguez (1999), who found that anxiety disorders were more prevalent in some Latino/a populations than among other groups.

Another issue related to the prevalence of psychological disorders among many ethnic and racial minority groups is socioeconomic status. In the U.S., ethnic and racial minority members are three times as likely as Caucasians to live below the federal poverty line (USDHHS, 2001). The most important implications of this fact are that people who live in poverty experience poorer overall psychological health (Miranda, Nakumura, & Bernal, 2003) and are up to three times as likely as those in higher socioeconomic strata to experience significant psychological problems (Muntaner, Eaton, Diala, Kessler, & Sorlie, 1998; USDHHS, 2001). What is more, people who are poverty-stricken also often reside in destitute neighborhoods and communities that present a multitude of risk factors for increased psychopathology (Miranda et al.).

Although the process of understanding prevalence rates of psychological disorders across racial and ethnic groups is convoluted, what is clear is that ethnic and racial minority group members experience psychological problems at a similar or higher rate as Caucasians (e.g., DHHS, 2001). Yet, there still exist notable disparities in utilization patterns among ethnic and racial groups. For instance, Latino/as underutilize all psychological services (O'Sullivan, Peterson, Cox, & Kirkeby, 1989; Sue, 1991). African-Americans also underutilize outpatient psychiatric services, but overutilize inpatient services (Snowden & Cheung, 1990). Furthermore, ethnic and racial minority populations are more likely than Caucasians to seek treatment in primary care settings than from agencies specializing in psychological health services and frequently access psychological treatment through hospital emergency rooms (USDHHS, 2001). African-Americans are also more likely than other racial groups to prematurely terminate treatment (Diala et al., 2000; Sue, Ivey, & Pederson, 1996), with up to 50% of African-American clientele dropping out after just one session (Council of National Psychological Associations for the Advancement of Ethnic Minority Interests, 2003). Finally, Asian-Americans and Native-Americans appear to underutilize services even more than African-Americans and Latinos/as (USDHHS, 1999).

In brief, it is evident that quality social and psychological services are as necessary for ethnic minority members, but are also less available and accessible to these groups than they are to Caucasians. Thus, for the purposes of this chapter, it is important for clinicians to note that the source of some anxiety-related symptomatology among many ethnic and racial minority clients could be a direct effect of their environment and life circumstances, rather than only being attributable to intra-individual characteristics.

## Culture and Treatment Complications

### *Culture and Treatment Resistance*

An important issue related to work with ethnic and racial minority members is what many clinicians might view as treatment resistance. One of the most significant correlates of apparent resistance to engaging psychological treatment is social stigma (USDHHS, 2001), which could impede treatment. Although stigmatization is ubiquitous in psychological services and exacerbates the difficulties of anxiety disorders in general, certain ethnic minority populations might hold beliefs that compound the perception of stigma related to those services. Stigmatization related to treatment seeking may be attributable to social situations (i.e., overt discrimination), cognitive appraisals (i.e., perception and fear of discrimination), and/or schema (i.e., internalization of socially introduced stigma; Green, Hayes, Dickinson, Whittaker, & Gilheany, 2003). Social stigma related to psychological problems might be so prevalent among some ethnic minority groups that members fail to seek and engage in any sort of psychological treatment. For instance, in a sample of both African-American and Caucasian clients, Copper-Patrick et al. (1997) found that African-Americans were more likely than their Caucasian counterparts to say that they were concerned with the social stigma that might be associated with receiving psychological treatment. Likewise, Thompson, Bazile, and Akbar (2004) found that common perceptions of psychological services among African-American populations involved high levels of perceived stigma and embarrassment associated with engaging services that were specifically related to psychological health. In other words, while many Americans likely perceive the need for and choice to seek psychological intervention as an embarrassing and stigmatizing prospect, this might be especially relevant for members of ethnic and racial minority groups.

Another important factor associated with perceived treatment resistance relates to the apparent mistrust that some ethnic and racial minority group members feel toward potential psychological service providers. Indeed, the USDHHS (1999) reported that members of some ethnic and racial groups cite fear of the treatment and/or treatment provider as the foremost reason for failing to seek and engage needed psychological intervention. Obviously, interpersonal trust or mistrust will have some impact on whether any individual will present for treatment, but again this association appears especially relevant among ethnic and racial minority populations. In addition, people with psychological problems are often the victims of degradation, rejection, and prejudice because they are considered sick, dangerous, worthless, and insincere (Biernat & Dovidio, 2000). This prejudice has been described as being similar to what many ethnic minority groups in the United States have endured for years thus compounding the problem of psychological problems in underserved populations (Biernat & Dovidio). For instance, in a study of African-American students, Nickerson, Helms, and Terrell (1994) found that those who endorsed high rates of cultural mistrust of Caucasians were significantly less likely to seek psychological services than those who endorsed lower rates of cultural mistrust. Likewise, Boyd-Franklin (2003) reported that some

African-Americans appear to cope with the realities of racism and oppression by refusing to extend interpersonal trust to individuals who differ from them in color or economic class. Thompson et al. (2004) also reported that a significant proportion of African-Americans who were engaged in discussion of whether they can trust psychological treatment providers asserted their belief that most clinicians are elitist and biased against African-American concerns. This brief sampling of empirical findings highlights the fact that even in areas where psychological services and treatment facilities are available, misunderstanding or fear about services and service providers probably impede their usage among some ethnic and racial minority groups (USDHHS, 1999).

It is also clear that family ties are especially strong among many ethnic and racial minority communities and that the family system is often one of the primary or sole sources of support (e.g., USDHHS, 1999; Wilson & Stith, 1991). These ties and notions of inter-familial reliance might be so strong, in fact, that some ethnic minorities are likely to strive to overcome psychological problems with family support and guidance in lieu of seeking formalized psychological intervention (Snowden, 1996). Related to both stigma and family ties, African-Americans might be more likely to experience the stigma of treatment because relying on non-familiar third parties to solve personal problems goes directly against a common cultural belief of inter-familial and self-reliance (Snowden, 1996).

Moreover, while many of our traditional psychotherapy models emphasize the treatment of the individual, many ethnic and racial minority group members live with extended family (e.g., grandparents, cousins) and might place greater emphasis on the family than on the individual and expect the family to be involved in the treatment process (USDHHS, 1999) and, thus, be less likely engage in treatment if these family members are not included. In short, ethnic and racial minority clients' expectations about the format in which psychological treatments are provided might account for a portion of perceived treatment resistance among some members of these groups.

Much like the importance of the family system, religion and spirituality also serve important intrapersonal and interpersonal roles among many ethnic and racial minority populations (e.g., Bernal & Saez-Santiago, 2006; Kelly, 2006). Accordingly, some ethnic and racial minority group members appear more likely to seek guidance from religious figures, rather than psychological health professionals, when faced with personal difficulties (USDHHS, 1999). Concisely, some ethnic and racial minority clients have been found to endorse more traditional methods of intervention (e.g., use of folk healers) as a means of treating psychological problems and might be less likely to seek treatment if they believe that traditional methods will not be integrated into the treatment process (Thomason, 2000).

As discussed above, it is evident that ethnic minority members are also uniquely subject to significant inequalities in psychological service availability and accessibility (USDHHS, 2001). In a word, access to psychological services is almost always lower for poorer communities than more affluent ones (Gresenz, Stockdale, & Wells, 2000; Zane, Hall, Sue, Young, & Nunez, 2004). Also, ethnic and racial minority members are also less likely than Caucasians to have private health

insurance resources to pay for necessary psychological treatment (USDHHS, 1999). Thus, cost of services has also been noted as one of the primary barriers to treatment for many ethnic and racial minorities (Thompson et al., 2004; USDHHS, 2001). Therefore, the cost associated with psychological treatment probably also necessitates and explains some ethnic and racial minority members' increased tendency to engage in alternative means of treatment. For instance, data from a series of focus group discussions with a large group of African-American participants revealed that excessive fees and preference for using low-cost, self-help methods such as prayer and consultation with religious leaders were the most commonly cited reasons why many clients did not engage in needed psychological services (Thompson et al., 2004). Thus, another significant cultural issue which complicates psychological treatment for many ethnic and racial minority group members has to do with cost and affordability of necessary treatment, as opposed to characteristics of the specific treatment or treatment provider. Meaning, some members of these groups might want to engage our services, but are precluded from doing so due to the price associated with a given treatment.

### *Culture and Language*

Language is one of the primary means by which cultural information is conveyed; so it follows that to be most effective, psychological services should be provided in the language each client prefers. Yet the majority of psychological health professionals are monolingual, English-speakers (Zane et al., 2004). This means that the majority of ethnic and racial minority members who do present for treatment will see a clinician who only speaks English and to whom they might not be as able to accurately convey their thoughts, feelings, and experiences. Obviously, this reality means that the misinterpretation of verbal expression probably affects the manner in which clinicians conceptualize a given problem and its appropriate method of treatment (Barona & Santos de Barona, 2003; Bernal & Saez-Santiago, 2006), posing another significant treatment complication. More specifically, when working with immigrant populations (e.g., Chinese or Mexican) language can present a major complication for treatment. The effort expended in communication by bilingual clients interviewed in a non-native language may produce greater vigilance and control over emotion than is desirable for proper assessment of distress (Malagady & Zayas, 2001).

Language complications in treatment are not limited to English versus non-English languages. Cultural values and language customs from any particular group may impede progress in treatment because cultural idioms of distress may not be understood. Indeed, many nuances of communication are contained in the cultural expressions of thoughts, feelings, and emotions (Ayonrinde, 2003). For example, an English-speaking Chinese-American may express the anxiety of living in a new country in terms of how the honor of the family is affected rather than in terms of personal worry or fear. Language and behavior used to express distress in ethnic minority populations may lead to misdiagnosis in assessment and further treatment

complications due to misunderstanding of both verbal and non-verbal expression (Malgady & Zayas, 2001). Throughout the relevant literature, these and other types of variations in culture-specific ways of talking about and relating distress are often termed *cultural idioms of distress* (e.g., Minhas & Nizami, 2006).

### ***Culture and Idioms of Distress***

One of the most prevalent idioms of distress among many ethnic minority groups is somatic presentation of psychological symptoms, particularly in relation to anxiety disorders (USDHHS, 1999). For instance, *Ataque de Nervios*, which is noted among some Latino/a populations, is a cultural idiom that is so common that it has been termed a *culture-bound syndrome* (APA, 2000). *Ataque de Nervios* is the manifestation of anxiety in which the individual might engage in uncontrollable shouting, verbal or physical aggression, crying, and/or trembling (e.g., Liebowitz et al., 1994). These symptoms might therefore lead to a misdiagnosis of a cluster B personality disorder when, in reality, the individual is expressing culturally specific symptoms of anxiety. Obviously, because these types of conditions might sometimes obscure or explain psychopathology, clinicians and researchers must account for cultural influences of behavior to realize the most accurate understanding, measurement, and diagnosis of psychological problems (Vega & Rumbaut, 1991).

Linguistic misinterpretations of expressions of distress by ethnic minority populations may also be attributable to the stress particular to acculturation. Acculturation involves the sociocultural process by which an individual from one culture comes into contact with and acquires a new culture (Berry, 2003). Individuals experiencing this process will experience group (termed cultural) and individual (termed psychological) levels of change. After contact with a host culture, individuals are likely to experience behavioral shifts first as they learn new behaviors associated with their new roles at work or school (Sue, 2003). Contact with a new culture may be accompanied by acculturative stress, or stress associated with behavioral shifts in learning new customs, language, and cultural practices. In many instances, acculturative stress is a direct result of prejudice and discrimination from the larger society (Berry, 1998). Although prejudice and discrimination are not as overt as in the past, it continues to be a significant problem presented in more subtle ways (Nelson, 2006). For example, when ethnic minority groups attempt to access adequate housing, medical care, or political rights, they may be denied these otherwise desirable features of a society because of implicit policies designed to exclude acculturating groups from full participation in society (Berry, 1998). Consequently, acculturating groups may be marginalized by the larger society leading to increases in stress, anxiety, and general malaise that can complicate treatment or prevent treatment seeking (Kanel, 2002).

Although conceptualized as necessary for an individual's sociocultural adaptation (Berry, 2003), *acculturative stress* has been linked to various psychological outcomes. For example, Latino/a adults have been found to experience more psychological problems (e.g., anxiety and substance use) as they increase in level of

acculturation (Ortega, Rosenheck, Alegria, & Desai, 2000). In addition, acculturative stress in Mexican-American adolescents has been shown to be significantly associated with increased depressive symptoms, lower self-esteem (Romero & Roberts, 2003), increased suicidal ideation, lower family functioning, and increased anxiety about the future (Hovey & King, 1996). Thus, acculturative stress can further complicate treatment and must be addressed in a culturally appropriate manner.

Even if these issues are addressed and ethnic and racial minority individuals present for treatment, it is important for clinicians to be aware of the differences in manifestation of symptoms linked to religious beliefs, values, and the understanding of one's place in society (Varela et al., 2004). Specific to anxiety disorders, *anxiety sensitivity* has been identified as a potentially culturally-relevant phenomenon among underserved ethnic minority populations. Anxiety sensitivity is the degree to which anxiety-related somatic/physiological symptoms are viewed as distressing or aversive (Peterson & Heilbronner, 1987). This is distinct from trait anxiety (a majority Anglo/Caucasian cultural construct) for ethnic minority youth (Silverman, Fleisig, Rabian, & Peterson, 1991) and adults (Peterson & Heilbronner) such that somatic signs of anxiety receive more attention and concern than cognitive and behavioral indicators. However, at this point it is essential to remember that the practice of drawing overarching conclusions about any individual based solely on group membership must be avoided. This caution will help us to remember that there is always a risk that cumulative suppositions about what are assumed to be cultural norms will be overgeneralized, misleading, and possibly stereotypical in describing treatment-seeking behaviors among ethnic and racial minority populations (Donohue et al., 2006; Zane et al., 2004).

As an example, when the first author worked with a 30-year-old Mexican-American woman diagnosed with generalized anxiety disorder, it was quickly discovered in assessment that cognitions related to fears and worry were not as distressing to her as her heart palpitations, shortness of breath, and dizziness. Specifically, when describing a particularly anxious evening reviewing her finances, she rated the severity of her distress related to feeling her heart "beat like it would break [her] ribs" and her stomach "being rung out like a wet towel" at a 9 out of 10 while she rated her worrisome thoughts of being evicted over late rent at a 4 out of 10 in severity of distress. In contrast, a 25-year-old Caucasian college student presented to the same clinic with descriptions of anxiety focused on her distressing thoughts that new acquaintances may think she is strange or "disturbed" by simple small talk. In assessing her cognitive and physiological symptoms, she reported the severity of detailed anxious thoughts such as "they're expecting me to say something but I'll sound stupid" and "I wish I hadn't said anything" at 8 out of 10 in distress. However, when asked, the Caucasian woman rated vague descriptions of her physiological status, such as "butterflies in [her] stomach," during such troublesome thoughts at 3 out of 10 in severity. Thus, when working with underserved ethnic minority populations, it may be important to include sensitivity to physiological symptoms as part of the behavioral conceptualization of anxiety rather than emphasizing cognitive aspects. This has the potential of assisting clinicians to

focus treatment techniques on that which can help reduce or manage the physiological aspects of anxiety (e.g., shortness of breath, racing heart, sweating, etc.) and therefore reduce overall distress.

## **Resolving Treatment Complications**

The above discussion of potential treatment complications related to work with some ethnic and racial minority group members highlights the necessity to examine and appropriately modify our standards of practice to meet the needs of these populations. As previously noted, the basic techniques of CBT serve as an excellent foundation from which therapeutic approaches within clinical psychology can continue to be made more culturally responsive. However, this is best accomplished by first accentuating the unique circumstances and experiences of the individual client in assessment (Hays & Iwamasa, 2006). In that way, culturally sensitive treatment adaptations will be based on individual cultural explanations of distress rather than broad sweeping generalizations that generally lead to prescribed treatment models that fail to recognize the within-group variability of various cultural groups.

### ***Culturally Appropriate Psychological Assessment***

In a review of cognitive-behavioral theory and techniques in assessment, Okazaki and Tanaka-Matsumi (2006) highlighted seven culturally responsive assessment paradigms that might have clinical utility in the cognitive-behavioral assessment of anxiety disorders across diverse cultural groups. The seven paradigms reviewed by Okazaki and Tanaka-Matsumi include (1) functional analysis, (2) the Culturally Informed Functional Assessment Interview (CIFA), (3) the DSM-IV-TR Outline for Cultural Formulation, (4) the Multicultural Assessment Procedure (MAP), (5) the ADDRESSING Framework, (6) the Explanatory Model Interview Catalogue, and (7) the Bicultural Evaluation. However, for the purposes of this chapter the Bicultural Evaluation will be discussed in further detail. Interested readers are referred to Okazaki and Tanaka-Matsumi for an excellent resource for a review of assessment techniques currently available with utility in culturally responsive assessment, case conceptualization, and treatment modifications.

As an extension of the functional analysis, the *Bicultural Evaluation* method proposed by Evans and Paewai (1999) is an example of culturally responsive assessment. This cognitive-behavioral model of case conceptualization is intended to build rapport, ensure cultural fairness, and use multiple sources of data similar to the functional analysis approach. In extending the functional analysis, Evans and Paewai created a checklist of 15 points of quality indicators of cultural responsive assessment. The checklist includes assessment of the cultural identity of the client, idioms of distress used in that client's cultural group, and the culturally relevant social support available to the client. In so doing, one may also assess the degree to

which conflicting demands in the social environment causes, maintains, or exacerbates distress that may be present due to acculturative stress (Berry, 1998; Evans & Paewai, 1999). Although this checklist of culturally responsive functional analysis was developed for the Maori people of New Zealand, it is easy to see how such a cognitive-behavioral assessment could be applied to various cultural groups.

It has also been suggested that an important general first step in culturally sensitive psychological assessment is comprised of working toward the development of a *cultural schema* (Hays & Iwamasa, 2006). In exercising this cultural schema in their work with culturally different clients, clinicians should gather cultural information about the population within which their interventions will be applied, obtain information from their clients regarding their specific personal experiences within their culture, and consult with culturally diverse colleagues (Hays & Iwamasa, 2006). Specific to anxiety disorders, another crucial aspect of moving toward more culturally relevant treatment involves the inclusion of broader aspects of the client's culture in case conceptualization and treatment delivery. This can be accomplished by employing some previously mentioned assessment paradigms that have been empirically supported to be sensitive to the idiographic nature of psychotherapy. For instance, in the assessment of anxiety, demographic characteristics are accepted as important considerations (e.g., Cooley & Boyce, 2004). However, failure to respond to the treatment of anxiety disorders has been conceptualized as a problem in the function of anxiety (Davies, Dubovsky, Gabbert, & Chapman, 2000).

Thus, proper assessment of the function of anxiety for a client can lead to more appropriate treatment options. For example, a thorough assessment may reveal that anxiety is a reaction to a comorbid psychological problem (e.g., schizophrenia) rather than being the primary disorder of concern. Davies and associates (2000) provide a fairly extensive list of 11 possible functions of anxiety that may be assessed from a medical/psychiatric standpoint. Some examples include questions such as "is anxiety caused by a medication?" and "is the family exacerbating the patient's anxiety?" (pp. A25 and A26, respectively). Although this list of questions is not a complete examination of the functions of anxiety, it may serve as the catalyst for further exploration of the client's particular situation which will invariably include their cultural beliefs and practices.

### ***Culturally Appropriate Treatment Adaptations***

Appropriate culturally sensitive assessment will assist clinicians in arriving at a specific case conceptualization and treatment planning similar to widely accepted methods of assessment and case conceptualization (e.g., Funnel Approach of Behavioral Assessment; Haynes & O'Brien, 2000). Marin (1993) suggested that a culturally appropriate intervention is one wherein the treatment strategies are expressly based on the cultural values of the group being served and are reflective of the subjective characteristics of the members of the group (e.g., attitudes, expectancies about treatment). More generally, cultural adaptations are characterized as adjustments that are applied to established treatments to better accommodate

the specific beliefs, values, and practices of the person being treated (Whaley & Davis, 2007). Although we do not have a well-studied and extensive understanding of the factors that will aid in the development of appropriate cultural responses to members of many ethnic and racial minority groups (e.g., Thompson et al., 2004), there are some considerations that might help begin to resolve treatment complications and enhance the psychological services and interventions we provide. Since we know that applied cultural adaptations appear to improve the therapeutic alliance and increase professional credibility (Ancis, 2004; Wampold, 2001) and that clients who were engaged in treatment programs that are specifically tailored for their specific ethnic minority group are significantly less likely to drop out of treatment than ethnic minority clients participating in more mainstream treatment programs (e.g., Takeuchi, Sue, & Yeh, 1995) it is important for all clinicians who work with members of diverse ethnic and racial groups to give ongoing consideration to these and other recommendations in the services they provide.

In terms of beginning to acknowledge and resolve some of the stigma that some ethnic and racial minority groups members might associate with psychological treatment, Hays (1996) offered that as part of her initial engagement of culturally different clients she explains the process as *counseling* rather than *therapy*. This recommendation could help to communicate the notion that clinician and client are partners in the treatment process, rather than subordinates and insubordinates. Hays also acknowledged that she seldom uses the terms *cognitive-behavioral therapy* in an effort to avoid conveying a disregard for the social, emotional, and spiritual aspects of the human experience. Concisely, one useful step in making CBT less stigmatizing and more culturally relevant might be as simple as changing the manner in which it is presented and discussed.

A recent meta-analytic review revealed that one of the most frequently acknowledged cultural adaptations involved explicitly acknowledging and discussing varying cultural values and experiences (Griner & Smith, 2006). This group of recommendations can serve several purposes, including the diminishment of client fear and mistrust toward the clinician. For instance, Gim, Atkinson, and Kim (1991) found that clinicians who openly acknowledged the importance of understanding facets of the client's ethnicity and culture were rated as more credible than clinicians who did not acknowledge these factors. Similarly, Thompson, Worthington, and Atkinson (1994) conducted a study in which clinicians discussed issues of being African-American with some of their clients and not with others. Results revealed that African-American clients in the group that was engaged in discussion of their racial background were subsequently more willing to disclose personal information to the clinician than those who were not engaged in such discussion. Moreover, in a sample of Asian international students, Zhang and Dixon (2001) found that clinicians' interest and regard for other cultures, including that of the client, was positively associated with student ratings of clinician trustworthiness and expertise. Thompson et al. (2004) also reported that African-American clients were more likely to report feeling interpersonal comfort and rapport with clinicians who were willing to openly acknowledge their interest in the client's background and ethnic experiences. Data such as these support the point that an important cultural

consideration that can be applied to any therapeutic paradigm is open and deliberate discussion of the background and experiences of the individual client.

In terms of making CBT more accessible and appropriate for culturally different clients, the above recommendation might be realized through working to validate the client's individual experiences with racism and discrimination and assessing how these might influence the problem and means for resolving it (Kelly, 2006). Inclusion of a candid dialog about the meanings of race and ethnicity to the individual client and discussion of his or her experiences as a member of a minority group might help reiterate the clinician's role as both an expert and a trustworthy confidant. In addition, when clinicians strive to express a genuine curiosity about the diverse experiences of their ethnic or culturally different clients, they might also facilitate beneficial treatment impacts like reduction of interpersonal mistrust and compensatory increases in therapeutic rapport. Also, ensuring we engage these types of discussion might also help us to remember that these experiences themselves place our clients at greater risk for anxiety-related disorders (USDHHS, 2001). Lastly, it is constructive to emphasize clients' ethnic and racial differences as assets rather than inadequacies (Quintana & Bernal, 1995). Therefore, clinicians should consider talking with ethnically and racially diverse clients about the facets of their heritage that bring them joy, satisfaction, and pride.

Given the above discussion of the importance of family ties among some ethnic and racial minority populations, some members of these groups might be more likely to seek professional intervention if their extended family system is also directly involved in the treatment process (USDHHS, 1999). Thus, clinicians should assess for these preferences and consider involving client's families to increase the likelihood that they will seek needed psychological services. Similarly, clinicians should also consider that they might be better able to retain ethnic or cultural minority clients in treatment if they contemplate ways in which they might integrate alternative methods into their already established scientifically-based interventions (e.g., encouraging prayer as a form of counter cognitions for anxiety).

Griner and Smith (2006) found that an often-mentioned cultural adaptation is to strive for an ethnic-match between client and therapist. For instance, Russell, Fujino, Sue, Cheung, and Snowden (1996) found that African-American adult clients who were matched with an African-American therapist were judged to have made more psychological improvements following treatment than those who were not matched. Likewise, in a sample of African-American adolescents, Yeh, Eastman, and Cheung (1994) found that clients who were matched with an African-American therapist were significantly less likely to drop out of treatment than those who were not matched with a therapist of the same race. Similarly, Sue, Fujino, Hu, Takeuchi, and Zane (1991) found that client-therapist ethnic match was related to lower treatment drop-out and increased use of psychological services for Asian-American and Latino/a clients. However, given the abovementioned percentage of clinicians who are Caucasian and presumably Anglo in culture, it would be difficult to realize this adaptation. Therefore, it is essential that the clinician acknowledges ethnic, racial, or cultural similarities and differences, while also understanding how

clients' cultural values foster specific expectations of the therapeutic process and the clinician's role in it (Bernal & Saez-Santiago, 2006). This could mean that the clinician engages the client in discussion of his or her ethical and professional limitations while attempting to clarify the client's expectations about the treatment.

Sue (2006) suggested that some culturally different clients might not even know what therapy is, what it can do, and what to expect from the process. Thus, another recommendation involves the engagement of the client in discussion of the purposes of the therapeutic process before treatment even begins. These types of pre-therapy interventions or client-orientation programs typically familiarize clients to psychotherapy by explaining the process, typical roles of clinicians and clients, what can be expected from treatment, and issues of confidentiality (Sue, 2006). In a study of the overall effectiveness of a pre-therapy intervention program with a sample of African-American clients, Acosta, Yamamoto, Evans, and Stillbeck (1983) found that engagement in a client-orientation program significantly increased general client knowledge about and favorable attitudes toward engaging in psychotherapy. Likewise, in a sample of Asian-American clients, Lambert and Lambert (1984) found that clients who were engaged in pre-therapy intervention reported being more satisfied with treatment and dropped out of treatment less often than those who did not receive pre-therapy intervention. In light of these data, it is important for clinicians to consider the potential benefits of taking extra time at the outset of the therapy process to ensure that their clients are aware of what will take place over the course of assessment and treatment.

Although we might not be able to impart significant change in the overarching costs associated with our expertise and services, we can apply specific adaptations to the treatment itself that might make the intervention more cost effective. The key, it seems, is to think of creative ways that might make our treatment suggestions more attainable for clients who do not have the financial resources that are more often noted among the majority population. For instance, one treatment for anxiety-related disorders is to include social activities as part of behavioral activation (Hays, 1996). A CBT therapist sensitive to the economic difficulties of the client might emphasize social activity lists that include enjoyable endeavors which do not require money.

Regarding culture and language, Sue (1998) found that clients matched to therapist with the same native-language were significantly less likely to prematurely terminate treatment and more likely to report satisfaction with interventions provided, as compared to non-matched clients. Therefore, whenever possible, it is essential that clients be matched with a clinician who is fluent in their preferred language. This could not only help the client to feel more comfortable and able to accurately reflect his or her experiences, but might also increase his or her willingness to remain in treatment. Of course, it will not always be possible for clients to be matched with a clinician who speaks their preferred language. However, we must still strive to make the language we do use more culturally syntonetic. Bernal and Saez-Santiago (2006) discussed how the language used in interventions should always be culturally appropriate for the person receiving the treatment.

They suggested that this will require more than just broad translation of a given intervention, but also the engagement of client with culturally syntonic language that gives reference to existing differences in regional and subcultural group dialects and preferences. Obviously, it would also prove beneficial to recruit and employ a larger number of bilingual clinicians.

## Conclusion

Although the topics discussed in this chapter are not exhaustive, those that are discussed here are considered most important by the authors to the understanding of cultural treatment complications and resolutions. The general issues of culture as it relates to treatment resistance, language, and idioms of distress have been examined. However, it was the direct intention of the authors to provide broad recommendations for culturally appropriate assessment and treatment. It is hoped that clinicians recognize that while some examples of possible assessment and treatment techniques that lend to culturally relevant assessment and treatment were discussed, it remains the responsibility of mental health providers to utilize sensitive clinical judgment that recognizes the importance of the individual and within-group variability. Thus, as the ethnic and racial minority populations of the United States continue to grow rapidly (U.S. Bureau of the Census, 2005) the field of clinical psychology will be more prepared to respond to the current and future mental health needs of underserved populations.

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**Part II**  
**Avoiding Treatment Failures:**  
**Disorder-Specific Perspectives**

# Avoiding Treatment Failures in Panic Disorder

Heather W. Murray, R. Kathryn McHugh, and Michael W. Otto

Panic disorder (PD) is characterized by recurrent panic attacks accompanied by worry about future attacks, worry about the consequences of the attacks (e.g., having a heart attack), or substantial behavioral changes in response to the attacks (American Psychiatric Association, 1994). Panic attacks themselves are distinct periods of intense fear accompanied by four or more physical symptoms which begin suddenly and reach a peak of intensity within 10 min. Panic attacks are ubiquitous to a wide range of anxiety disorders, but in PD the focus of the phobic concern is on the anxiety symptoms themselves and the feared consequences of these symptoms (Barlow, 2002).

Initial panic attacks are predicted by stress as well as the interaction between stress and the vulnerability factor, anxiety sensitivity. Anxiety sensitivity is the fear of anxiety-related somatic sensations based on the presumed catastrophic consequences of these sensations (McNally, 2002), and is an effective predictor of individuals who panic in response to the biological provocation of these symptoms (e.g., Rapee & Medoro, 1994; Schmidt & Mallott, 2006). Whereas stress may be a prospective predictor of first panic attacks (Watanabe, Nakao, Tokuyama, & Takeda, 2005), those with higher anxiety sensitivity appear to be particularly at risk for panic when confronted by stress (Schmidt, Lerew, & Jackson, 1997; Zvolensky, Kotov, Antipova, Leen-Feldner, & Schmidt, 2005).

Although panic attacks can occur in nonclinical populations and across the spectrum of anxiety and mood disorders, few individuals proceed to develop PD. For example, whereas approximately 10% of people experience a panic attack that meets full criteria each year (Norton, Cox, & Malan, 1992), the estimated lifetime prevalence of PD in the U.S. is approximately 3.5–5% (Grant et al., 2006; Kessler et al., 1994, 2006). The lifetime prevalence of PD with agoraphobia (PDA) is approximately 1%, and both PD and agoraphobia occur in isolation, with prevalence rates of 3.5–4% and 0.2–0.8%, respectively (Grant et al.; Kessler et al.). Women are more likely to develop PD than men, with higher numbers of women characterizing those with more extreme agoraphobia (Yonkers et al., 1998).

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From a cognitive-behavioral perspective, PD is conceptualized as the learned fear of autonomic arousal and its associated physical sensations. Panic attacks consist of the sympathetic nervous system “fight-or-flight response” activated outside of the context of an identifiable threat. The development and maintenance of PD following the presence of one or several unexpected panic attacks can be facilitated by the aversive nature of the physical symptoms themselves and catastrophic interpretations of the symptoms. Frequently, panic attacks are misinterpreted as signs of impending death (e.g., a heart attack or stroke), disability (e.g., fainting or going crazy), or loss of control (e.g., inability to escape). Although fears of humiliation may serve to intensify the catastrophic interpretation of some of these sensations (“others will see I am losing control,” “they will think I am an anxious fool”), panic is differentiated from social anxiety disorder by the fear of these sensations independent of social evaluation. Fears and catastrophic misinterpretations of symptoms along with subsequent hypervigilance for somatic sensations help create a self-perpetuating pattern of anticipatory anxiety, vigilance for symptoms, memories of past attacks, and fears of future attacks (Barlow, 2002; Clark, 1996). Future panic attacks can be cued by minor psychological and physical changes, which become associated with danger and the resultant panic cycle (Bouton, Mineka, & Barlow, 2001). Furthermore, anxiety and tension resulting from hypervigilance can mimic the symptoms of panic and further contribute to this pattern. This cycle of panic maintains and may exacerbate the core fear of panic-like physical sensations.

Similar cycles of fear and vigilance characterize a number of other disorders. For example, hypochondriasis has been characterized as a feed-forward cycle of misinterpretation of bodily symptoms, anxious apprehension and increased vigilance to these symptoms accompanied by checking behaviors and reassurance seeking (Warwick & Salkovskis, 1990). Indeed, generalized anxiety, worries about somatic symptoms, and tendencies toward avoidance appear to characterize the premorbid state of many patients with PD. For example, 18 of a series of 20 patients with PDA reported a history of generalized anxiety, hypochondriacal fears and beliefs, and/or agoraphobic avoidance before the first panic attack (Fava, Grandi, & Canestrari, 1988; see also Fava, Grandi, Rafanelli, & Canestrari, 1992). For example, Fava, Grandi, Saviotti, and Conti (1990, p.352) describe the following case:

Mrs E. was a 50-year-old blue-collar worker with a 35-year history of disease phobia (consistently focused on the heart) and hypochondriasis. Eight months prior to the assessment, the patient’s sister, to whom she felt very close, had surgery for gastric carcinoma. Shortly afterwards, Mrs. E. ’s preoccupations with having a heart attack increased, and panic attacks ensued.

According to the cognitive-behavioral model of PD, once these panic attacks occur, and are catastrophically misinterpreted, then a fear-of-fear cycle can ensue with recurrent panic attacks accompanied by vigilance to symptoms and avoidance of events and situations associated with panic attacks. This model is summarized pictorially below (Fig. 1). The value of the model is in providing an overview of the primary patterns in PD, and in identifying elements of the panic cycle for intervention.

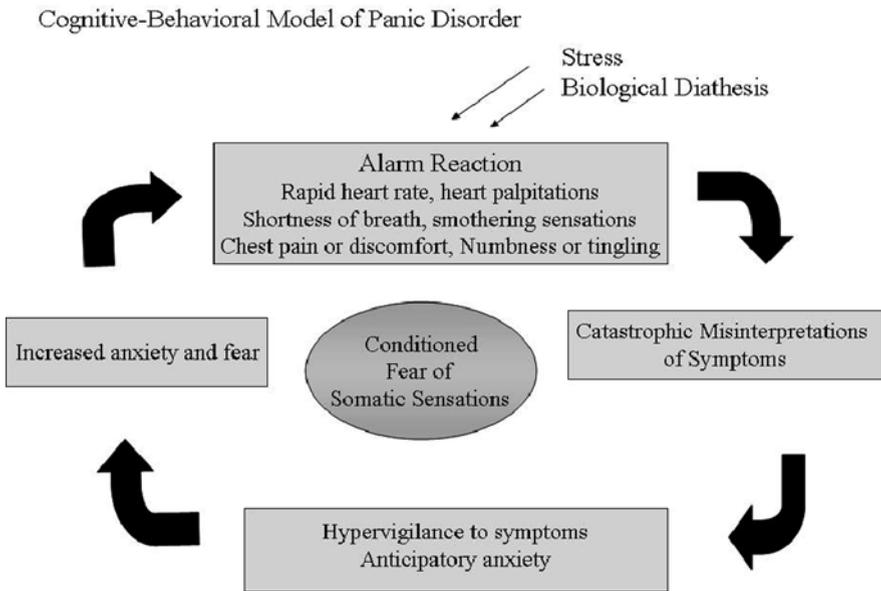


Fig. 1 Cognitive-behavioral model of panic disorder. Adapted from Otto and Gould (1996)

### Core Elements of Treatment

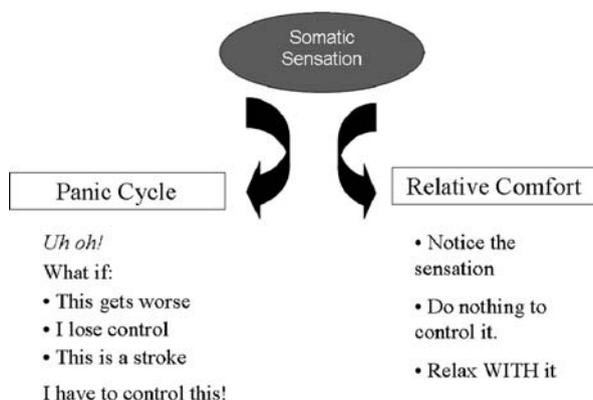
Core interventions for PD include psychoeducation, cognitive interventions, and, particularly, interoceptive (internal) and in vivo exposure (Fig. 1). Psychoeducation includes the nature and source of panic symptoms, the nature and style of catastrophic misinterpretations of these symptoms, associated anxious apprehension and vigilance, and the role of avoidance behaviors. This education is complemented by prospective monitoring of symptoms to help to build awareness of panic patterns within the new framework provided by the model of the disorder.

Cognitive interventions are aimed at eliminating catastrophic misinterpretations of symptoms and establishing more adaptive thinking patterns. Of particular importance to cognitive restructuring efforts for PD are the cognitive errors of: (1) overestimating the probability of negative outcomes, and (2) assuming the consequences will be unmanageable (i.e., catastrophizing). Reviewing these maladaptive cognitive patterns and teaching the patient to challenge these thoughts can reduce their salience, and shift interpretation of these thoughts from “fact” to “guess.” These strategies include evaluating evidence for the thought and evaluating the cost of the feared outcome (e.g., could you cope with fainting in public?). Subsequent exposure interventions provide patients with direct opportunities to examine negative predictions about the consequences of anxiety and panic symptoms and to learn directly from experience.

In current treatment protocols, exposure is initially targeted to feared internal sensations (Craske & Barlow, 2007; Otto & Pollack, in press). A range of physical

exercises are used to induce these sensations. For example, head rolling can be used to induce dizziness; hyperventilation can be used to induce dizziness, shortness of breath, dry mouth, hot flushes, and numbness and tingling; stair running can be used to induce a racing heart, shortness of breath, and sensations of heavy legs; breathing through a straw can be used to induce shortness of breath; and spinning in a chair can be used to induce dizziness and nausea.

During exposure patients are to attend to these sensations without engaging in defensive behaviors (e.g., tensing, moving, escaping the situation). By learning to do nothing in response to these sensations, patients have the opportunity to examine the degree to which the sensations are tolerable and manageable. As fears of the sensations decline, these sensations lose the ability to provoke panic. Figure 2 summarizes the use of interoceptive exposure to help train an alternate response to symptoms.



**Fig. 2** Training Alternative Responses to Feared Somatic Sensations; From Smits and Otto (2009) (Reprinted with permission from Oxford University Press)

The progression of exposure, like in other exposure treatments, should be graded. Therapists work to provide more intense interoceptive exposure procedures while also fading out safety cues (e.g., practicing independently of the therapist, practicing away from home). Likewise, as therapists transition to treating the fear of situations, a hierarchy of agoraphobic situations should be identified. For each exposure, patients should expect initial anxiety, and apply skills learned during interoceptive exposure (note the anxiety but do not try to control it) while waiting for the anxiety to dissipate and completing goal-directed activities (e.g., shopping during exposure to a mall).

A critical component to the success of exposures is the accurate identification and fading of safety cues or behaviors. Individuals often have developed behaviors to reduce the anxiety experienced and/or to feel an increased sense of safety while remaining in the feared situation. These behaviors are referred to as “safety behaviors”, and contribute to the cycle of PD in that the individual “learns” that he/she needs to engage in these behaviors to cope with the situation. Safety behaviors may

be difficult for the patient to identify, as the patient may have been engaging in such behaviors for years without fully recognizing the function of their behavior. Also, individuals with PD engage in cognitive strategies to reduce anxiety. An example of a cognitive safety strategy is praying before entering an elevator. These safety behaviors are often very subtle; therefore, taking the time to identify such behaviors or cognitive strategies is important before initiating the exposure to order for the patient to experience the anxiety fully when completing the exposure task. These can be important to the grading of exposures in allowing for the adjustment of difficulty level as exposures progress. For example, the patient may first go to the mall with a friend (i.e., a “safe” person) and carry her cell phone and a bottle of water. Subsequent exposures can begin to strip away these safety cues in order to increase the difficulty of the exposure. It is of particular importance to identify and eventually remove any signs of safety to ensure that learning has generalized and is not limited by conditional thought (e.g., “I was only OK because I had my cell phone.”).

**Table 1** Key elements of exposure

- 
- Review rationale in the context of the cognitive-behavioral model of panic, including emphasizing the importance of re-learning safety to innocuous sensations and situations.
  - Evaluate feared physical sensations and situations.
  - Emphasize the importance of remaining in the exposure despite anxiety and not utilizing management strategies (observe for any subtle strategies).
  - Instruct the patient how to induce sensations and what sensations to expect from each induction. Model inductions for the patient to ensure proper use.
  - Begin with exposures of moderate difficulty to provide a model for more difficult exposures and to build patient efficacy in tolerating feared sensations.
  - Instruct the patient to evaluate the nature, intensity, and similarity of sensations to panic.
  - Use exposures as an opportunity to test beliefs about panic (e.g., “I will faint if I feel dizzy.”)
  - Conduct interoceptive exposures first in-session, ensuring adequate duration of induction and repeating each exposure at least twice.
  - Change the context of interoceptive exposures to increase difficulty and facilitate generalization of learning (i.e., conduct exposures outside of the session, alone, etc.).
  - Introduce in vivo exposures in a graded fashion beginning again with moderate difficulty. Combine in vivo and interoceptive exposures to increase difficulty.
  - Evaluate and remove safety cues and behaviors with continued in vivo exposures.
  - Consider additional context changes (e.g., time of day, with caffeine or after a meal, with various people).
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## Non-essential Elements of Treatment

Research evidence suggests that some commonly used components are not necessary for achieving positive outcomes (see Schmidt et al., 2000). Some cognitive-behavioral treatments for PD include components of training in skills for reducing anxiogenic responses to bodily sensations, such as diaphragmatic breathing and progressive muscle relaxation. These techniques are introduced to reduce levels of anxiety and to assist in the preparation for exposures. Although an excellent strategy for reducing tension and arousal, particularly for generalized anxiety

(see Behar & Borkovec, this volume), the use of these strategies for PD may be deleterious to treatment outcomes if they are adopted as avoidance or safety behaviors (Schmidt et al., 2000).

## Format of Treatment

Individual CBT for panic typically consists of 12–15 weekly, 1-hour sessions (Craske & Barlow, 2007). Briefer versions have also demonstrated efficacy, including four-session (Craske, Maidenberg, & Bystritsky, 1995) and five-session treatments (Clark et al., 1999). Brief CBT has also been applied in a primary care setting. Roy-Byrne et al. (2005) found that patients randomized to six sessions of CBT in combination with a patient manual (“Mastery of your Anxiety and Panic”, 3rd edition; Barlow & Craske, 2000) compared to TAU, sustained gradually increased improvements relative to TAU on both PD symptoms, overall mental health functioning, and disability scales. Furthermore, the CBT was delivered by mid-level behavioral health specialist with little or no CBT experience prior to training for this study, exemplifying that CBT can be disseminated in this manner. CBT can also be administered in a group format while maintaining efficacy (e.g., Telch et al., 1993) and may offer a particularly cost-effective option.

## Treatment Example – Interoceptive Exposure

David is a 35-year-old male presenting with PDA. Onset of panic symptoms occurred approximately 8 years ago following the death of his grandmother. His panic attacks were characterized by heart palpitations, shortness of breath, choking sensations, dizziness, sweating and fear of having a heart attack or insanity. Since his initial panic attack, he has had several panic attacks while driving in traffic and subsequently avoided driving during rush hour or on main roads.

Initial sessions were focused on psychoeducation providing treatment rationale and information on the nature and maintenance of panic. Several sessions were dedicated to cognitive restructuring. David was able to identify his catastrophic thinking patterns and to replace his cognitive distortions with more adaptive alternatives. The rationale for interoceptive exposures was explained and the following dialog highlights sticking points in this intervention.

Therapist (T): The first exercise we will try is hyperventilation. This exercise will lead to feelings of dizziness, lightheaded, and occasionally numbness and tingling in the arms or around the mouth or scalp. Also, because your eyes may dilate from hyperventilation, you may notice that things look bright after we finish. I will demonstrate the exercise to do and then I will have you perform the exercise. We will hyperventilate for a minute, and then I will tell you to stop (*therapist briefly demonstrates hyperventilation*).

- Patient (P): (*patient discontinues exercise before therapist does*) I don't know if I can do this.
- T: I understand this is hard for you. You do have concerns about the symptoms we are inducing. Why is this telling you that you cannot do this exercise?
- P: I don't understand why this is important.
- T: I can understand why you would not want to bring on these distressing sensations. You've been really good at avoiding these sensations, and you have been doing so for years. In what way has this avoidance been a problem for you?
- P: Well, I have had to rearrange my life to avoid the panic. I can't go on vacations with my family, I leave for work early, I can't take business trips.
- T: Right, avoiding these sensations is not working in the long run. These exercises give you the opportunity to confront these feared sensations to learn to tolerate them. Let's try it again. And this time, while doing the exercise, attend to the sensations that you are feeling, so that you can describe them to me. And as you notice these feelings, I would like you to do nothing to manage them. Just let the feelings happen, notice them, and do nothing to control them. (*patient completes exercise*)
- T: Great. What physical sensations did you notice during the exercise?
- P: I felt dizzy. It felt like I wasn't getting enough air.
- T: What else did you notice?
- P: I also felt numb around my face.
- T: OK, we knew that might happen. How did you do at not managing the sensations.
- P: Well I really wanted them to stop.
- T: Why did that seem important?
- P: I did not know what was going to happen. I kept thinking, "what if they get worse."
- T: OK, so you started focusing on the future, and what could happen. But how were the sensations that you actually felt.
- P: They were weird. I was dizzy.
- T: Yes, they are weird, but how did you do with the weird sensations when you just let yourself feel them – in the present.
- P: I guess they were OK. I did not like them, but they were OK.
- T: OK, I would like you to try the exposure again, and let yourself really focus on the present. Even though you may have thoughts about the future ("what may happen next") I want you to really focus on the sensations you are experiencing right now and see how comfortable you can be in just letting the sensations occur.
- P: OK. (Patient completes a second exposure.)
- T: How do you feel?
- P: Dizzy but OK.
- T: Are you staying in the moment, or are you focusing on the future.
- P: A little of both.

- T: OK, let me get a sense of how strong the dizziness was, from 0 to 100, with 0 representing “not at all” and 100 representing “the most intense dizziness you can imagine.”
- P: It was probably a 40.
- T: How similar was this dizziness to what you feel during a panic attack, again from 0 to 100?
- P: Not that similar. Maybe a 50.
- T: And how anxious did those sensations make you feel?
- P: I felt pretty anxious the first time, but the second time was around a 50.
- T: OK, it looks like we have a nice procedure to help you get comfortable with the sensations linked to your panic. With some practice feeling the sensations and doing nothing in response to the sensations, I think we can help you feel much more comfortable with dizziness.

Several sessions later, David has successfully continued his interoceptive exposures and developed a fear and avoidance hierarchy. At this point, the focus shifts to situational exposures (or if needed, imaginal exposures). Graded exposures were planned, starting low on David’s hierarchy. In an early exposure, in which David stayed in an enclosed space with the therapist, he was not experiencing sufficient anxiety.

- T: How anxious do you feel now, from 0 to 100?
- P: Not very, maybe a 20.
- T: What sensations are you feeling? And how intense are they?
- P: I’m not really experiencing any physical sensations.
- T: Let’s make this more challenging. What would make this more difficult for you?
- P: I don’t know.
- T: Do you think if you were to do the hyperventilation exercise while staying in this situation, that would increase your anxiety?
- P: Yeah, it would. (*patient completes hyperventilation*)
- T: How anxious do you feel now?
- P: A little higher, maybe a 40.
- T: Remember we talked about safety behaviors. Do you think you might be engaging in one of those behaviors?
- P: I don’t know. I don’t think so.
- T: We talked about cognitive safety behaviors, what thoughts were you having during the exposure?
- P: I told myself that I just have to get through it because it will be over soon.
- T: That sounds like a safety behavior to me. Let’s try this again, this time, without those thoughts; instead just let yourself feel the sensations and see what it is like when you don’t manage them.

After removing safety behaviors, David was able to engage in the exposure and to experience moderate levels of anxiety. Later in treatment, David experienced

difficulty as exposures became more difficult. An exposure higher on his hierarchy involved riding in an elevator. Prior to the exposure, the therapist reviewed the rationale for exposure and the patient chose to participate in the in-session exposure. After several minutes in the elevator with the therapist, David reported that his anxiety remained high.

P: My anxiety isn't going down. I want to get off now.

T: Ah, and tell me what makes your anxiety seem so dangerous. You have been learning that you can have anxiety sensations and still be OK. Just ride it out and it will decrease on its own. This is good practice to see that the anxiety isn't dangerous.

P: But if I can't leave, I am not sure if I can stand my anxiety. What if I really panic?

T: There is one of those "what if" thoughts. And you are focusing on a need to escape and you seem to be actively *trying* to make your anxiety go down. I am sure that isn't helping. I want to give you a chance to experience anxiety and notice what happens if you let yourself stay in the elevator, to know you are in the elevator, and to not focus on the door or leaving. Will you try it? (*therapist is hesitant to engage in too much discussion as this may serve as a cognition distraction*)

P: I guess I can try it.

David continued in the exposure until his anxiety decreased. Following completion of the exposure, the therapist reinforced his efforts and discussed his experience. Homework assignments reflected in-session exposure and continued interoceptive exposures.

## **Efficacy of CBT Treatment**

Of the available treatments, CBT for PD has demonstrated excellent treatment efficacy that equals or surpasses that for pharmacologic treatments (e.g., Barlow, Gorman, Shear, & Woods, 2000; Furukawa, Watanabe, & Churchill, 2006; Gould, Otto, & Pollack, 1995). Treatment for PD is well-tolerated by patients (Barlow et al., 2000; Hofmann, et al., 1998), associated with quality of life improvement, including improvement in co-occurring disorders (Tsao, Mystkowski, Zucker, & Craske, 2002; Telch, Schmidt, Jaimez, Jacquin, & Harrington, 1995), and durability of gains after treatment discontinuation (Margraf, Barlow, Clark, & Telch, 1993; Gould et al., 1995; Barlow et al., 2000; Craske, Brown, & Barlow, 1991). CBT also improves response to pharmacologic interventions (e.g., Marks et al., 1993; Mavissakalian, 1990; Schmidt, Wollaway-Bickel, Trakowski, Santiago, & Vasey, 2002) and is efficacious as a next-step strategy for patients who did not respond adequately to pharmacotherapy (Heldt et al., 2003, 2006; Otto, Pollack, Penava, & Zucker, 1999; Pollack, Otto, Kaspi, Hamerness, & Rosenbaum, 1994). Furthermore, CBT aids in the discontinuation of anxiolytic (e.g., benzodiazepine, antidepressant) medications

without loss of treatment efficacy (e.g., Otto, Hong, & Safren, 2002; Whittal, Otto, & Hong, 2001).

The transportability of CBT outside of research settings is supported by its strong cost-efficacy (McHugh et al., 2007; Otto, Pollack, & Maki, 2000), and effectiveness in clinical practice (Addis et al., 2004; Stuart, Treat, & Wade, 2000; Wade, Treat, & Stuart, 1998) and primary care settings (Roy-Byrne et al., 2005). For example, in a study of the clinical effectiveness of CBT relative to treatment as usual, Addis and colleagues (2004) found that 49% of patients receiving CBT exhibited significant symptom change over the course of treatment. Although decreases in symptoms were also noted in the treatment as usual condition, less than 20% of patients demonstrated significant change from pre- to post-treatment.

In summary, there is ample good news about the efficacy of CBT. In some trials, CBT has helped patients achieve panic-free rates in the range of 74–85% (e.g., Barlow et al., 2000). Moreover, maintenance of treatment gains during follow-up periods of 1–2 years have shown equally promising panic-free rates ranging from 81% to 87% (DeRubeis & Crits-Christoph, 1998). However, it is also clear that panic-free rates does not mean freedom from panic-related disability (Margraf et al., 1993; Clark et al., 1994), nor are patients who are panic free at one assessment necessarily those who are panic free at the next assessment, and some patients also seek additional treatment after the initial treatment episode (Brown & Barlow, 1995). Also, in other investigations, lower response rates have been reported. For example, in a large, multi-site investigation of the relative efficacy of individual and combined pharmacologic and cognitive-behavioral treatments for PD, cognitive-behavior therapy was associated with a 51% rate of non-responders at treatment termination, which grew to approximately 67% at 6-months post-treatment (Barlow et al., 2000). Consideration of predictors of non-response can provide a perspective on these variables outcomes.

## **Depression Comorbidity and Treatment Resistance**

The most common finding in the PD treatment literature is that CBT is often resilient to the effects of comorbid depression (for review see Deveney & Otto, this volume). This conclusion is based on studies where depression was evaluated by symptom scores as well as diagnosis in individuals where the PD was the primary target of treatment (e.g., Basoglu et al., 1994; Black, Wesner, Gabel, Bowers, & Monahan, 1994; Barlow et al., 2000; Kampman, Keijsers, Hoogduin, & Hendriks, 2002; Laberge, Gauthier, Cote, Plamondon, & Cormier, 1993; McLean, Woody, Taylor, & Koch, 1998; Wade et al., 1998). However, these findings are not without inconsistencies, and there is evidence that at times depression does assert a negative effect on CBT outcomes for PD (Basoglu et al., 1994; Maddock & Blacker, 1991; Skeketee, Chambless, & Tran, 2001), as well as being associated with greater clinical impairment pretreatment (e.g., Joormann, Hertel, Brozovich, & Gotlib, 2005). When it does have a negative impact, comorbid depression may assert its negative effects on any of a number of factors including motivation, the accuracy and nature of cognition, problem solving, and the evaluation of treatment and progress.

Interventions for these challenges are reviewed in Chapter 12 (Otto & Deveney, this volume) and are not further reviewed here.

## Comorbid Anxiety Disorders and Treatment Resistance

Patients with PD often have comorbid anxiety disorders; studies indicate 20–50% of outpatients have comorbid social phobia (Stein, Shea, & Uhde, 1989), and between 3.5% and 5% of individuals have lifetime prevalence rates of both a PD and GAD diagnosis (Kessler et al., 1994; Wittchen, Zhao, Kessler, & Eaton, 1994) and high rates (27–32%) of panic symptoms with PTSD (Leskin & Sheikh, 2002). Comorbid anxiety conditions may interfere with treatment and attenuate treatment response, though the research supporting this is mixed (Tsao, Lewin, & Craske, 1998; Brown et al., 1995; Tsao et al., 2002). CBT treatment strategies are specific to type of anxiety disorder, though the skill of cognitive restructuring included in CBT for PD can be helpful in intervening for a variety of cognitions. In addition, tolerance to the physiological distress, in particular the distress related to panic attacks, can generalize to similar distress associated with other anxiety disorders, particularly those with similar physiological arousal such as social phobia and specific phobia. We recommend vigorous treatment of the primary presenting disorder, and then subsequent evaluation of whether core fears underlying secondary disorders have been adequately addressed. Therapists should consider re-designing exposures to also take into account additional elements (e.g., fears of being embarrassed by panic symptoms in individuals with comorbid social phobia) that may be inadequately addressed by standard disorder-specific exposure interventions. Likewise, use of “worry-free zone” techniques (see Behar & Borkovec, this volume) to help patients gain control over runaway worry processes may be necessary for individuals with comorbid generalized anxiety disorder. A case example follows that illustrates the shifting of exposure assignments to incorporate anxiogenic concerns that were inadequately treated by core procedures for PD.

John is a 41-year-old African-American male presenting to the clinic with a primary diagnosis of PD and a secondary diagnosis of social anxiety disorder. John had a long-standing history of panic attacks that made it difficult for him to take public transportation and travel far from home. He reported fears of having a panic attack when traveling which would either embarrass him in front of others or prevent him from being able to escape. John avoided all travel for a period of time; however, this became impractical given his job and he disliked the way that panic limited his ability to engage in activities farther away from his home. John began to research his illness and successfully reduced his avoidance by forcing himself to go places and “white knuckled” through the anxiety. Yet, John’s panic persisted and he presented to treatment with the goals of reducing panic attacks and facilitating his ability to travel farther from home.

After initial psychoeducation on the nature of PD, John fully embraced the fear-of-fear model. He was able to accept the logic of his cognitive restructuring, but had difficulty accepting his anxiogenic thoughts at an emotional level. For example, John was able to develop rational responses to his panic fears (e.g., “I have never fainted;

therefore, it is unlikely that I will faint this time”); yet, each time he experienced panic-like physical sensations, the original catastrophic thoughts re-emerged. John and his therapist utilized interoceptive exposure exercises as a way of retraining his reactions to physical symptoms of panic. His therapist described the technique of “doing nothing” in response to the sensations - simply observing the sensations without reacting to them in a catastrophic manner. John participated in several interoceptive exposures, but had difficulty using this technique without safety behaviors - he gripped the sides of his chair during hyperventilation and dizziness exercises until the induction ceased. This “white knuckling” appears to be the same way he has handled previous experiences with anxiety.

John began to understand how to react differently to his sensations when he was able to link the physical sensations of panic with those that he experiences, and tolerates, during physical exercise. It appeared as if the thought, “Huh, this is similar to what I experience when working out”, allowed John to react differently to the physical sensations which, in turn, resulted in a reduction of the sensations, without him having to “white knuckle” through them.

Although this experience was a helpful breakthrough, John continued to report significant anticipatory anxiety. He had difficulty using his interoceptive exposure experience to challenge some of his panic thoughts, and he continued to fear that the next bout of physical symptoms would result in marked embarrassment and prolonged panic.

In light of these fears and John’s comorbid social anxiety disorder, his therapist combined an exposure for PD (riding on public transportation) with a social anxiety exposure to address his inflated estimates of social cost (e.g., doing something embarrassing while riding on public transportation). Once John’s anxiety around riding public transportation had decreased somewhat, the therapist asked John to identify the feared consequences of doing something embarrassing (e.g., sitting in a strange way) while on the bus. John and his therapist then sat in an unusual way on the bus and observed John’s anxiety as well as whether his feared consequences came true. John’s anxiety peaked and then gradually declined, as he observed that there was no social cost to this behavior. Hence, the inclusion of this social cost exposure, more traditionally associated with social anxiety disorder, allowed John’s fears of panic attacks to decline dramatically. During the following session, John reported a marked reduction in anxiety and number of panic episodes. He also reported more success discounting his catastrophic thoughts about panic, realizing that not only was he unlikely to faint, but that others were unaware of his anxiety. Marked progress followed this additional exposure.

## **Alcohol and Substance Use and Treatment Resistance**

Alcohol dependence frequently co-occurs with PD (12-month prevalence rate of 11%) and PDA (12% prevalence rate) as indicated in a large study (Grant et al., 2004). The 12-month prevalence rates for substance use disorders for individuals

with PD and PDA is 17% and 24%, respectively, and the rates of substance dependence is 13% and 15%, respectively (Grant et al., 2004). Substance use and withdrawal can mimic panic symptoms and impacts the quality of session retention as well as overall treatment adherence rates. Interventions for these challenges with comorbid alcohol and substance abuse and dependence are reviewed in Chapter 14 (Lejuez et al., this volume) and are not further reviewed here.

## Personality Disorders and Treatment Resistance

Prevalence rates for comorbid personality disorders, most commonly avoidant, dependent, histrionic, and borderline personality disorders, with PD is common within the range of 40–70% of patients meeting criteria for both (Diaferia et al., 1993, Mavissakalian & Hamann, 1988). A diagnosis of an Axis II disorder often but does not necessarily predict poorer treatment outcome has been debated (Reich & Vasile, 1993, Chambless, Renneberg, Goldstein, & Gracely, 1992; Mennin & Heimberg, 2000; Reich & Green, 1991). Patients with comorbid anxiety disorders and personality disorders often have higher severity of Axis I pathology at follow-up, though the severity is better accounted for by elevated severity at baseline (Van Den Hout, Brouwers, & Oomen, 2006). For example, Van Den Hout et al. (2006) large treatment outcome study found that 72% of the participants with an Axis II disorder made significant clinical improvements with the CBT compared to 78% of the participants without the Axis II disorder, though those patients with personality disorders reported higher symptoms severity at baseline and follow-up.

Given that treatments targeting PD have shown reductions in personality pathology (Mavissakalian, 1990) as well as defensive style (Heldt et al., 2007), we recommend initial targeting of the PD to see how many apparent personality symptoms change as part of the overall change in panic-related distress. Moreover, the enhanced tolerance of emotion brought by interoceptive exposure procedures may offer general benefits for symptoms well outside the domain of PD (Otto, in press). However, when personality pathology appears to interfere with the completion of treatment procedures, consideration of core concepts of dialectical behavior therapy (DBT; Linehan, 1993) may be of use. For example, a therapist may emphasize communication styles that convey acceptance of their patient while focusing on teaching their patient specific skills to change target (frequently avoidant) behaviors. In addition, highlighting behavioral action (exposure) as a choice and using problem-solving techniques in deciding to complete the exposure exercises will empower the patient and enhance the collaborative therapeutic relationship.

Incorporating DBT treatment strategies with panic interventions in the same session may be challenging with more severely impaired patients, particularly when self-injurious behaviors and suicide ideation are endorsed. In these situations, a patient's distress of the week may overshadow the regular practice needed to help patients progress with panic treatment. Under these conditions, it may be helpful to schedule double weekly sessions, with clear demarcation of the focus of each,

reserving one session for weekly distress and the other for skill building to undo panic cycles.

## **Medical Factors**

Certain medical illnesses and side effects from medication may present as PD and may complicate assessment and treatment of panic. Hyperthyroidism, hyperparathyroidism, vestibular dysfunctions, seizure disorders, and cardiac conditions are a few examples of medical conditions that should be evaluated and the impact of anxiety determined before a diagnosis PD is assigned. Medications and medication discontinuation can also cause anxiety. In addition, prevalence rates of patients with medical conditions developing panic attacks is high for patients with respiratory disorders (such as asthma) and cardiac conditions (coronary artery disease), in part because the sensations associated with the anxiety are believed to be related to the medical condition. A comprehensive assessment will further identify medical conditions and medication that may be contributing to panic attacks, and addressing the underlining factors may improve therapeutic interventions and increase efficacy of treatment.

## **Life Stressors**

The onset of life stressors, unrelated to PD, is not uncommon during the course of treatment and may exacerbate symptoms of panic. Assessing life issues that are preventing attention to treatment goals and adjusting the treatment plan to address the life issues may reduce overall anxieties and increase long-term adherence to treatment. Life stressors, including relationship conflicts, may arise due to a decrease in panic symptoms and increased functioning. For example, a wife may become resentful of her husband's treatment since he, as a result of experiencing a reduction in symptoms, is less willing to watch their children every evening when his wife wants to go out and spend time with her friends. An informational couples session is a strategy frequently employed to address changes in relationship roles as changes in symptoms occur (e.g., Otto & Pollack, in press). Highlighting the short-term and long-term benefits of treatment may help the patient continue to persist with the treatment despite short-term consequences that may be challenging to relationship roles. Also, more involved inclusion of couples in treatment has shown beneficial effects (Barlow, O'Brien, & Last, 1984; Cerny, Barlow, Craske, & Himadi, 1987).

## **Addressing Inadequate Treatment Response**

Given the overall success of CBT for PD, and evidence that the model of the disorder is not only useful in treatment but also in predicting and preventing the onset of PD (Gardenswartz & Craske, 2001), we believe that the first step in addressing treatment

resistance lies in a re-evaluation of core fears and better honed efforts at providing the patient with the therapeutic learning most needed to address these fears. As such, in the sections below, we consider the motivational and avoidance-based issues that prevent patients from having a chance to learn from their own experiences, as well as modifications that can be made in treatments to better provide patients with useful learning once they do engage in exposure.

Considering avoidance of treatment interventions, a patient may be hesitant to engage in the interoceptive and in vivo exposures when the rationale for the exposure is unclear or if the patient believes that engaging in the feared situation is not helpful based on previous experiences. If this is the case, reviewing the principles of exposure by utilizing metaphors and examples may facilitate a shift in sense of control. In particular, a patient may begin to challenge himself or herself in facing his or her fears as opposed to feeling forced to engage in the activity based on the therapist's instructions. For patients that demonstrate rigid thinking patterns, worksheets can also be helpful in making the connections. In addition, having your patient explain why exposure reduces anxiety in the long run will help you determine the level of understanding your patient has for the concepts behind exposure. When patients believe that their previous attempt to face their fears is evidence that the exposure won't work, help the patient to identify factors that contributed to their experience. Often, patients will report engaging in safety behaviors, terminating the exposure prematurely, and evaluating their experience based on their emotions rather than their behaviors. Reviewing the rationale of exposures and incorporating the role of safety behaviors, duration of the exposure and setting up clear goals and expectations, based on definable behaviors, will help ensure their future exposure will be a success.

Motivation impacts treatment in many ways and can have many presentations. Homework noncompliance, canceling sessions, refusing to complete in-session assignments may be related to motivation, though a seasoned therapist will assess the function of noncompliance to determine the role of avoidance in the noncompliance behavior. In addition, signs of noncompliance should lead to an evaluation of barriers to treatment and is an opportunity to teach and practice problem-solving strategies and time management skills as they apply to treatment. Restating expectations and verbalizing the framework of the treatment may also be appropriate. Sometimes patients come to treatment believing that their therapist will "fix them" rather than providing a model and training in the change process. Helping patients play an active role in therapy, in part by discussing therapy as action based rather than talk based, may help change these expectations.

Also, a lack of symptom reduction despite progressing through the treatment phases may be linked to targeting the wrong core fears, prescribing exposure exercises that are too specific, or compromising exposures with the use of safety behaviors. Reconfirming the primary diagnosis and assessing comorbid conditions is an excellent start to further examining the nature of current core fears affecting the patient. Also, we recommend attention to reassessing core fears through Socratic questioning, and then re-examination between these fears and the exposures arranged to date. The goal is to assess whether the exposures are truly targeting

the core concerns and fears and attempt to generalize the learning that occurs during the exposures to other feared stimuli. In addition, treatment planning in the face of non-response should include re-evaluating the role and use of safety behaviors and planning to minimize safety behaviors during exposure practice. Table 2 provides a summary of some of these targets for addressing treatment resistance. Also, review

**Table 2** Troubleshooting non-response to CBT for panic disorder

If you believe that treatment resistance is related to:	Then address the issue by trying:
1. Comorbid Axis I disorder	1. Often the CBT strategies generalize to symptoms other than panic, though specific interventions addressing anxiety may increase the response to the panic treatment. Example: for depression, assign behavioral activation for GAD, assign scheduled “worry time”.
2. Comorbid Axis II disorder	2. Since CBT treatment for panic disorder decreases personality pathology, targeting panic disorder is a good initial strategy unless the personality pathology is interfering with the treatment goals. Strategies to address personality pathology include: a. Discussing general tolerance to uncomfortable physiological responses (related to panic) and intense emotions. b. Incorporate DBT techniques in treatment approach. c. Scheduling split or double sessions to address both the panic symptoms and personality pathology separately.
3. Medical Factors	3. Refer out for medical treatment to address underlining medical conditions contributing to the panic symptoms.
4. Life Stressors Example: relationship conflict due to decreased panic symptoms	4. Assess stressors and the impact the stressors have on attaining the treatment goals. Incorporate strategies to reduce life stressors. Example: Conduct an informative couple’s session describing the short- and long-term benefits of symptom reduction.
5. Patient believes the treatment approach will not work	5. Review the rationale for the treatment approach, utilizing metaphors and examples. Assess contributing factors to your patient’s beliefs. Example: your patient believes that he has already completed the exposures on his own and they didn’t work and concludes that the treatment won’t work either. Strategies to try: elicit specifics about the attempted exposures, identify safety behaviors, duration of experience, and measure of success of experience. Provide psychoeducation including principles of exposures.
6. Barriers to treatment	6. Assess and address barriers to treatment compliance utilizing problem solving and time management skills.
7. Targeting the wrong disorder	7. Complete thorough diagnostic assessment and adjust the treatment plan accordingly.
8. Targeting the wrong core fears	8. Assess the patient’s core fears and adjust cognitive restructuring and exposure strategies to target fears.
9. Proscribing exposure exercises that are too specific	9. To increase generalization, assign homework exposures in various contexts.

of exposure parameters as summarized by Powers, Smits, and Otto (this volume) can help ensure that the right context is being addressed by exposures of sufficient duration to optimize change.

## **Other Psychosocial Treatments**

Relative to the wealth of evidence for CBT for PD (e.g., Gould et al., 1995), there is little evidence to guide the application of alternative psychosocial treatments. A multicenter trial of emotion-focused psychotherapy emphasizing empathic listening and supportive strategies for managing painful emotions and stressors did not support its efficacy for PD (Shear, Houck, Greeno, & Masters, 2001). In contrast, a recent trial of a manualized psychoanalytical psychotherapy showed significantly greater benefit than applied relaxation alone, showing a 73% response rate relative to the 39% found for the relaxation condition. This response rate is promising, but was achieved across 24 sessions of treatment – roughly double that offered for many CBT protocols. Also, it is unclear what elements of treatment were of importance to the manualized psychodynamic therapy. It shares a number of elements with emotion-focused approaches, including exploration of feelings surrounding panic onset and the meanings of panic symptoms. Additional elements of treatment were focused on investigating transference and working through recurrent conflicts. In addition, response in this trial appeared to be moderated by the presence of Cluster C personality symptoms according to an exploratory analysis (Milrod, Leon, Barber, Markowitz, & Graf, 2007), raising questions whether an additional focus on recurrent conflicts (e.g., with problem-solving focused treatment) may be of value with this subgroup of patients. Additional research is needed to clarify the reliability and strength of these effects and the elements of treatment most associated with differential responding.

## **Medication Treatments**

Issues of combined treatment are covered in detail in “Combined Cognitive Behavioral and Pharmacologic Treatment Strategies: Current Status and Future Directions” Smits et al. (this volume), but it is also important to note here that any assessment of treatment resistance should consider the nature and timing of medication use. Medications taken on an as needed (PRN) basis almost surely function as a safety cue and can lead patients to attribute gains to factors other than their own efforts. Also, if patients fail to respond to CBT, even after adequate troubleshooting of factors that may be interfering with therapeutic learning, medication treatment is the dominant empirically supported alternative to CBT, and should be considered in collaboration with the patient, as an option for addressing treatment non-response should continued CBT no longer appear to offer clear benefit.

## Conclusions

CBT for PD is a powerful intervention that offers some of the best outcomes in the empirical treatment literature. Nonetheless, partial and non-response to these interventions continues to be a problem. In this chapter we introduce a number of strategies for honing exposure interventions toward the core fears underlying the patient's disorder. In many ways, the art of therapy for the cognitive-behavior therapist is successfully getting patients to use these interventions, providing the therapeutic experiences that can undo the self-perpetuating cycles underlying PD. With careful problem solving, and close collaboration with the patient, therapists have a wide range of options for altering the timing, targets, context, and difficulty of exposure interventions to provide the patient with this needed therapeutic learning.

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# Avoiding Treatment Failures in Obsessive Compulsive Disorder

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## The Case of Maria: “Only a Horrible Mother Would Have These Thoughts”

Maria is a 29-year-old, married, Caucasian female who was prompted by her husband Bob to seek treatment for Obsessive-Compulsive Disorder (OCD) following the birth of their first child. At intake, Maria noted that she had a nursing degree but had decided to stay home after her child was born. During the initial therapy sessions, Maria was upset and reluctant to disclose her obsessions. She stated that she has had some obsessive thoughts and behaviors since she was about 13 years old, but in the past she had been able to work around them. She checked the locks of her house excessively, cleaned the bathroom daily because of fear of germs, and was very perfectionistic in her work as a nurse. Maria noted that when she became more depressed, her OCD symptoms increased, which in turn made it more difficult for her to manage at home and at work. In addition to being diagnosed with OCD at intake, Maria was also given comorbid diagnoses of Major Depressive Disorder, Recurrent (MDD) and Generalized Anxiety Disorder (GAD). She did not meet criteria for any personality disorders. Despite this level of interference and distress, Maria never sought psychotherapy for OCD until she gave birth to her daughter Sophia. Maria had seen a psychiatrist once for her OCD and MDD symptoms, at which time the doctor had recommended that Maria consider taking a selective serotonin reuptake inhibitor (SSRI), but she had refused it due to her desire to get pregnant.

Since the delivery, Maria experienced horrifying, vivid, intrusive thoughts about harming her daughter. Maria felt so guilty and ashamed of these obsessions that she was initially afraid to disclose them to the therapist. Maria feared that if the therapist became aware of the thoughts, she would judge Maria as a bad person who deserved to be in prison.

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After psychoeducation and some normalization, Maria was able to discuss her current obsessions in detail. She reported thoughts about accidentally dropping Sophia or putting her in the microwave, which she interpreted as evidence that she wanted to harm her daughter. In addition, Maria reported intrusive sexual obsessions when changing her daughter's diaper. Again, Maria interpreted these thoughts as evidence that she might want to molest her daughter, and in consequence was terrified. To avoid the associated anxiety, Maria stopped changing Sophia's diapers, which created a great deal of stress in her marriage. When Sophia cried because she had not been changed in hours, Maria would call her husband Bob at work and demand he come home to change the baby. At first, Bob was sympathetic to her fears and tried to reason with her, explaining that these were merely thoughts and that she did not have a history of hurting children. Bob even came home a few times because he was concerned for Maria's well-being, as she sounded very upset on the phone. However, as time passed, Maria got progressively worse, which led Bob to become less understanding and more upset. Finally, Bob demanded that Maria seek treatment.

Maria's case provides an example of OCD symptoms that were significantly exacerbated by the birth of a child. In Maria's case, she had a long-standing history of OCD and some depressive symptoms, which she had previously found ways to manage. When Maria sought therapy, her OCD symptoms were in the severe range and she was on the verge of divorce.

## Overview of Obsessive-Compulsive Disorder

Obsessions are defined as intrusive, recurrent, distressing thoughts, images, or impulses that the person attempts to suppress or ignore (APA, 2000). Compulsions are repetitive behaviors or mental rituals that the individual performs in an attempt to minimize the anxiety generated by the obsessions (APA). Although individuals suffering from OCD might be able to postpone their compulsive rituals, they often cannot stop them. Performing compulsions can, in turn, trigger further obsessions or doubts about whether the compulsions were done correctly, which leads to the need to perform even more compulsions. As a result, untreated OCD symptoms often maintain themselves or worsen over time.

According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV TR; APA, 2000), to be diagnosed with OCD, the individual must at some point have insight that the disorder is unreasonable or excessive; the symptoms must be time consuming (i.e., more than an hour a day); and the symptoms must lead to interference and/or distress (APA). Patients with OCD vary in their level of insight with regard to the senselessness of their symptoms (Foa & Kozak, 1993). Low insight often occurs as a consequence of heightened discomfort/anxiety generated by intrusive thoughts (Kozak & Foa, 1994). Some obsessions may border on delusions when the patient believes that the intrusive thoughts accurately reflect reality.

OCD tends to be a chronic disorder. Symptoms may wax and wane over time, but often do not remit without treatment. Lifetime prevalence for OCD has been estimated at approximately 2% (Weissman et al., 1994). Most often OCD begins during early adolescence, with fewer cases being diagnosed after the age of 15, although some cases do onset in early childhood (Rachman & Hodgson, 1980; Rasmussen & Tsuang, 1986). On average, boys tend to develop symptoms younger than girls, but the prevalence tends to be slightly higher for women than for men (Bellodi, Scuito, Diaferia, Ronchi, & Smeraldi, 1992; Rasmussen & Eisen, 1990). Although onset of OCD symptoms has been associated with stressful life events (Kolada, Bland, & Newman, 1994; Rachman, 1997) and traumatic experiences (de Silva, Marks, de Silva, & Marks, 1999; Rheaume, Freeston, Leger, & Ladouceur, 1998), at least 40% of patients with OCD cannot pinpoint stressors or traumas that preceded onset. As described in our case vignette, pregnancy and childbirth are stressors that have been linked to onset of OCD for new mothers and fathers who are plagued by obsessions regarding the harm of their newborn (Wisner, Peindl, Gigliotti, & Hanusa, 1999).

In contrast to other anxiety disorders in which there is a great deal of symptom homogeneity, OCD symptoms can vary greatly in clinical presentation. For example, patients might engage in ordering and arranging rituals, excessive washing rituals, reassurance seeking, counting, praying to neutralize religious obscenities, etc. Many researchers have attempted to create classification systems reflecting OCD symptom sub-types (Abramowitz, Franklin, Schwartz, & Furr, 2003; Baer, 1994; Calamari, Wiegartz, & Janeck, 1999; Leckman et al., 1997; Mataix-Cols, Rauch, Manzo, Jenike, & Baer, 1999; Summerfeldt, Richter, Antony, & Swinson, 1999). Research suggests that OCD symptoms tend to cluster into the following sub-types: (1) Harming, religious, and/or sexual obsessions with mental or checking rituals; (2) contamination obsessions with cleaning or washing rituals; (3) obsessions about certainty/symmetry and ordering/arranging rituals; and (4) hoarding. However, in clinical practice a patient's symptoms may not fit neatly into these categories. Most patients with OCD report several types of obsessions and compulsions, some of which may not fall within the proposed OCD sub-types. In addition, even though most patients report obsessive thoughts, images, and impulses, some patients cannot identify a clear obsessive thought that triggers their repetitive rituals. Often these individuals report a feeling that they must engage in their compulsive behaviors until it feels "just right."

### ***Comorbidities***

The majority of patients with OCD will report symptoms of additional psychiatric disorders, most commonly another anxiety and/or mood disorder (Lucey, Butcher, Clare, & Dinan, 1994; Rasmussen & Eisen, 1990; Yaryura-Tobias et al., 2000). Patients with OCD are at a high risk for developing another anxiety disorder. For example, research suggests that 25–50% of patients with OCD experience social anxiety disorder, GAD, and panic disorder (Eisen et al., 1999). In addition,

some patients will report a comorbid diagnosis of post-traumatic stress disorder (PTSD; Helzer, Robins, & McEvoy, 1987), which might pose an additional challenge for treatment of OCD. If a patient meets criteria for PTSD, this might warrant treatment prior to addressing the OCD as some of the treatments for OCD (i.e., exposure exercises to feared stimuli) can trigger trauma symptoms (e.g., if the person is obsessed with cleanliness due to a past history of rape). Thus, it is crucial for clinicians to perform comprehensive diagnostic assessments or a behavioral analysis to ascertain which condition would require immediate treatment and if there would be any reasons for addressing the PTSD prior to the OCD or vice-versa.

Mood disorders are highly comorbid with OCD. Indeed, some research suggests that up to 75% of individuals with OCD report depressed mood (Rasmussen & Eisen, 1992). Approximately 25–30% of patients with OCD will meet criteria for MDD and approximately 10% will meet criteria for dysthymia (Steketee, Henninger, & Pollard, 2000). Research suggests that mood disorders are often secondary to OCD (Yaryura-Tobias et al., 2000; Rasmussen & Eisen, 1992; Welner, Reich, Robins, Fishman, & VanDoren, 1976) and may contribute to the poor functioning that is frequently associated with OCD.

Another common diagnostic challenge is distinguishing between OCD and Obsessive-Compulsive Personality Disorder (OCPD), a personality disorder characterized by perfectionism, rigidity, and conformity to rules. OCPD is often associated with OCD, yet their clinical relationship is still unclear. The majority of individuals with OCD (75%) do not have OCPD and the majority of patients with OCPD (80%) do not have OCD (Mancebo, Eisen, Grant, & Rasmussen, 2005). OCD patients were more likely to meet diagnostic criteria for OCPD than healthy controls, but OCPD was not more prevalent in OCD patients than those with Panic Disorder (Albert, Maina, Forner, & Bogetto, 2004). However, there may be a genetic link between the two disorders: first-degree relatives of OCD probands were twice as likely to have OCPD than relatives of healthy controls (Samuels et al., 2000). OCD patients with comorbid OCPD had significantly earlier onset of obsessive-compulsive symptoms and were significantly more likely to have symmetry and hoarding obsessions as well as cleaning, repeating, and ordering compulsions than OCD patients without comorbid OCPD (Coles, Pinto, Mancebo, Rasmussen, & Eisen, 2006). This suggests that OCPD may influence OCD symptom presentation, onset, and severity.

### ***Diagnostic Challenges***

It can be challenging for clinicians to accurately diagnose OCD, given that some OCD symptoms appear similar to symptoms of other anxiety disorders (e.g., recurrent worries in individuals with GAD) and OC-spectrum disorders (e.g., checking mirrors in individuals with Body Dysmorphic Disorder). A comprehensive clinical evaluation that relies on empirically supported screening instruments for OCD such

as the Yale-Brown Obsessive Compulsive Scale (YBOCS; Goodman et al., 1989) can aid clinicians in making the appropriate diagnosis.

Patients who report sexual and harming obsessions (e.g., a man who reports obsessions about molesting his newborn baby and related avoidance of young children) may be a source of uneasiness for clinicians trying to differentiate among diagnoses of paraphilias, sexual cravings, and OCD. In these cases, the clinician should inquire whether the thoughts provoke fear/anxiety (consistent with OCD) or generate pleasure (consistent with paraphilias). In addition, clinicians should obtain the patients' sexual history and inquire about past history of sexual abuse/assault. Patients with OCD will often report tremendous shame, anxiety, and avoidance behavior associated with these thoughts, which is not as typical of patients with paraphilias. Furthermore, patients with postpartum psychotic symptoms might also note intrusive thoughts that could mimic OCD symptoms (e.g., harm to others), which are inherently different from intrusive obsessive thoughts. Clinicians are also advised to consider Antisocial Personality Disorder as a differential diagnosis (Wilhelm & Steketee, 2006).

Another commonly discussed diagnostic challenge is differentiating between the worries characteristic of GAD and obsessions indicative of OCD. Like obsessions, worries tend to be intrusive, repetitive, and often uncontrollable. In addition, GAD-related worries might be associated with repetitive checking rituals (Brown, Moras, Zinbarg, & Barlow, 1993; Schut, Castonguay, & Borkovec, 2001) that are related to safety concerns, but might be mistaken for compulsive checking that is characteristic of OCD. Although worries and obsessions are similar in that they increase the individuals' anxiety and tend to have a repetitive, intrusive nature, worries differ from obsessions in that worries usually involve realistic life concerns like health, finances, or work performance and their content is not usually considered unacceptable. This is in contrast to obsessions, which are more likely to be considered senseless, unacceptable, and ego-dystonic (Turner, Beidel, & Stanley, 1992). In addition, worries often present as linguistic representations whereas obsessions present as images, thoughts, and impulses (Turner et al., 1992).

PTSD also shares symptoms with OCD. Some researchers have postulated that the association between OCD and PTSD might be weaker than previously thought due to the symptom overlap between these disorders and major depression, which has several symptoms that overlap with PTSD and OCD (Huppert et al., 2005). To tease apart these disorders clinically, it is helpful to inquire about the focus/nature of the repetitive thought. For patients with PTSD, intrusive thoughts tend to be recollections of actual traumatic events, and the ensuing anxiety is not typically followed by repetitive or ritualized behaviors common in OCD.

OCD is often comorbid with several disorders that collectively have been referred to as OC- or OCD-spectrum disorders. These are disorders that share a similar clinical presentation or tend to co-occur frequently with OCD. For example, research suggests a symptom overlap between OCD and tic disorders such as Tourette's disorder (Mansueto, Keuler, Mansueto, & Keuler, 2005). One way to differentiate between tics and compulsions is to keep in mind that the former are involuntary movements, the performance of which is unrelated to anxious thoughts or

feared consequences. This is in contrast to compulsions, which are often purposely performed to decrease anxiety generated by the obsession. As mentioned above, sometimes patients with OCD do not report a specific obsession that triggers a compulsion, but rather they feel compelled to perform a behavior until it feels “just right.” In such instances, it becomes challenging to differentiate between OCD and tic disorders. Researchers have labeled this phenomenon “Tourettic OCD,” suggesting that the association between these disorders might be better accounted for by a diagnosis encompassing both disorders (Mansueto et al., 2005).

Impulse control disorders such as hair pulling (Trichotillomania), skin picking, and nail biting are also frequently comorbid with OCD. Often behaviors characteristic of these conditions can also be differentiated from compulsions, as the former, like tics, are not performed to prevent feared consequences. However, there are instances in which behaviors that are often characteristic of impulse control disorders may be best conceptualized as symptoms of OCD or other OC spectrum disorders. For example, a patient presenting with repetitive hair pulling may be best diagnosed with trichotillomania. However, if this patient reports she engages in hair pulling in an attempt to make things symmetrical, e.g., to make sure that her hairline is perfectly even, then the behavior is likely a manifestation of OCD. If the behavior is done to improve one’s appearance, a diagnosis of Body Dysmorphic Disorder (a preoccupation with an imagined or slight defect in appearance) should be considered. Indeed, patients suffering from Body Dysmorphic Disorder often perform compulsive behaviors; however, they are usually triggered by thoughts of being ugly or deformed, and are done to hide, improve, or check on their appearance concerns, which is distinctively different from a symmetry obsession in OCD (Wilhelm, 2006).

## **Treatment for OCD**

### ***Overview of CBT Strategies***

Empirically supported treatment strategies for OCD include behavior and cognitive-behavioral therapies (CBTs), such as exposure and response prevention (ERP), and pharmacological treatments. However, it is important to note that serotonergic reuptake inhibitors (SRIs) such as clomipramine and SSRIs such as fluoxetine have also been demonstrated to decrease OCD symptoms (e.g., Kobak, Greist, Jefferson, Katzelnick, & Henk, 1998; O’Connor et al., 2006; O’Connor, Todorov, Robillard, Borgeat, & Brault, 1999; van Blakom et al., 1994). Although these drugs are helpful, data suggest that they do not perform better than ERP (Kozak, Liebowitz, & Foa, 2000), and that the combination of drugs and CBT is not more effective than either treatment (Cottraux et al., 1990; Kozak et al.; van Balkom et al., 1998). Although early psychotherapy outcome literature focused primarily on behavioral treatments (e.g., ERP), recent data suggest that cognitive therapy, which focuses

on understanding and correcting OCD-related beliefs (e.g., inflated responsibility, need for certainty, perfectionism), is also effective in treating OCD (Wilhelm et al., 2005). For the purposes of this chapter, we will focus primarily on a combination of cognitive and behavioral strategies for treating OCD.

Research suggests that intrusive thoughts are a common occurrence in the general population, with approximately 80% of the population reporting such thoughts (Rachman & de Silva, 1978; Salkovskis & Harrison, 1984). Cognitive models of OCD postulate that OCD results not from the presence of intrusive thoughts per se, but rather from the patients' interpretations of intrusive thoughts as either harmful or significant (e.g., as evidence that they might be responsible for something going horribly wrong, such as accidentally dropping a child; Salkovskis, Shafran, Rachman, & Freeston, 1999; Salkovskis, 1985, 1989; Salkovskis, Campbell, Salkovskis, & Campbell, 1994; Salkovskis & Harrison, 1984). Such interpretations, in turn, lead patients to compulsively try to neutralize thoughts or avoid stimuli that might trigger the thoughts (e.g., Maria not changing her daughter's diapers). Unfortunately, such compulsions and avoidance behaviors reinforce OCD symptoms in the long run.

Behavior therapy (BT) for OCD is based on research showing that compulsions decrease the anxiety generated by obsessions (Rachman & Hodgson, 1980). In the short term, patients are relieved after a successful compulsion. However, the negative reinforcement (elimination of anxiety) increases the chance that the next obsession will be followed by a compulsion, so in the long term doing compulsions worsens the OCD. BT focuses primarily on prolonged *exposures* to feared stimuli and *response prevention* of mental or behavioral rituals. Patients learn that in the absence of ritualizing, anxiety will eventually decrease on its own. Through successive, repeated prolonged exposures, patients' emotional discomfort to feared stimuli decreases as habituation occurs.

In contrast, cognitive therapy (CT) from a Beckian tradition (Beck, 1976) uses Socratic questioning to identify and challenge beliefs that are hypothesized to support OCD behaviors (Wilhelm & Steketee, 2006). Obsessive anxiety experienced by OCD patients is thought to originate from dysfunctional core beliefs described by the Obsessive Compulsive Cognitions Working Group (1997; see Table 1). In addition to the beliefs, a recently published cognitive manual for CT for OCD adds two additional cognitive domains that might be important for clinicians to address: (1) Consequences of Anxiety and (2) Fear of Positive Experiences (see Table 1; Wilhelm & Steketee, 2006). The goal of cognitive therapy is to assist in improving patients' insight into the irrationality of their beliefs about intrusions and help patients to learn cognitive strategies to decrease their anxiety. For further description of CT for OCD, see Wilhelm & Steketee, 2006.

In summary, CBT for OCD relies on cognitive and behavioral principles that suggest the importance of breaking the association between obsessions and compulsions and modifying maladaptive core beliefs that contribute to obsessional fears and behaviors. For a complete guide to treating OCD patients, please refer to Steketee (1996) and Wilhelm & Steketee (2006).

**Table 1** OCD-related beliefs

Type of belief	Definition and clinical examples
Overimportance of thoughts <sup>1</sup>	<ul style="list-style-type: none"> <li>• Belief that thoughts are important and require attention, including the belief that thoughts can generate actions (i.e., thought–action fusion; Rachman, 1993) “If I have a thought about molesting my daughter, it means that I must want to molest her”<sup>3</sup></li> </ul>
Control of thoughts <sup>1</sup>	<ul style="list-style-type: none"> <li>• Belief and desire to control one’s own intrusive thoughts, which result in rebound effects. Often accompanied by mental rituals “Only child molesters have this type of images; I must be out of control if I cannot control these thoughts”</li> </ul>
Overestimation of danger <sup>1</sup>	<ul style="list-style-type: none"> <li>• Belief that things are dangerous unless proven otherwise. Patients often overestimate the likelihood of danger based on faulty data “I am much more likely to be punished than others.” “There was one crime in my area last year, so I should watch out to make sure my doors are locked because there is a high chance I will be robbed”</li> </ul>
Desire for certainty <sup>1</sup>	<ul style="list-style-type: none"> <li>• Based on the hypothesis that patient’s desire for certainty is associated with their inability to cope with ambiguity (Sookman, Pinard, &amp; Beck, 2001). Often, desire for certainty motivated reassurance seeking and re-doing ritual (i.e., checking, re-reading, etc.) “If I am not 100% sure of all possible options, I am bound to make a mistake”</li> </ul>
Responsibility <sup>1</sup>	<ul style="list-style-type: none"> <li>• Belief that the person has the power to prevent or cause unwanted outcomes, often related to moral concerns “If I don’t pick up the nail in the road, there will be a car accident because of me”</li> </ul>
Perfectionism <sup>1</sup>	<ul style="list-style-type: none"> <li>• Based upon the belief that it is possible and desirable to find an exact solution for every problem. There is some evidence suggesting that early experiences with rigid teachings can create a vulnerability to perfectionist standards and fear of failure (Frost &amp; Steketee, 1997; McFall &amp; Wollersheim, 1979) “If I don’t do my work perfectly, people will not respect me”</li> </ul>
Consequences of anxiety <sup>2</sup>	<ul style="list-style-type: none"> <li>• Irrational belief about one’s ability to tolerate anxiety or a fear that one might “go crazy” if the anxiety feels too intense, despite a lack of evidence “If I get too anxious, I will fall apart”</li> </ul>
Fear of positive expectations <sup>2</sup>	<ul style="list-style-type: none"> <li>• As patients progress in therapy, they might express concerns about being able to maintain their gains or that they are not worthy of getting better. Sometimes these doubts might have a superstitious flavor or have a moral tone, where people believe they do not deserve to get better “Good events will be inevitably followed by bad ones”</li> </ul>

<sup>1</sup>Core belief domains proposed by the Obsessive Compulsive Cognitions Working Group (1997).

<sup>2</sup>Core belief domains proposed by Wilhelm and Steketee (2006).

<sup>3</sup>Examples were designed for this chapter, based on several OCD patients seen by the authors.

## ***Treatment Response***

Over the past several decades, many studies have documented the treatment efficacy of behavioral, cognitive behavioral, and, most recently, cognitive treatments for individuals with OCD (Cottraux et al., 2001; Emmelkamp & Beens, 1991; Emmelkamp,

Visser, & Hoekstra, 1988; Van Oppen, de Haan, Van Balkom, & Spinhoven, 1995; Whittal, Thordarson, & McLean, 2005; Wilhelm et al., 2005). Many of these studies focused on individual therapy, but a few have examined group therapy (Anderson & Rees, 2007; Cordoli et al., 2003, 2002; Krone, Himle, & Nesse, 1991) or comparing the efficacy of individual versus group therapy (Fals-Stewart, Marks, & Schafer, 1993). This section will review what type of responses clinicians might expect when treating patients with OCD, followed by a discussion of factors that might predict treatment non-response.

Research has shown that ERP is highly effective. Meta-analytic reviews have demonstrated the superiority of ERP to placebo ERP (van Blakom et al., 1994). Approximately 75% who enroll in BT and do not drop out are likely to benefit from treatment (Steketee & Frost, 1998). On average, patients treated with ERP tend to drop from a severe range (YBOCS>25) prior to treatment to a mild/moderate range (YBOCS between 10 and 17) over a course of approximately 15–20 sessions of ERP (Steketee & Frost, 1998). A meta-analysis by Abramowitz (1997) suggested that more hours of therapist-guided exposure exercises predicted better outcome (Abramowitz, 1997). Although impressive, these results suggest there still is room for improvement in current treatments. Future research should include the addition of relapse prevention strategies as patients who learned relapse prevention were more likely to remain improved at follow-up compared to those that did not (Hiss, Foa, & Kozak, 1994).

Researchers have also examined the benefits of cognitive therapy for OCD (Emmelkamp et al., 1988). Results suggested that cognitive therapy was as effective as exposure-based treatment, although it is arguably difficult to distinguish among these therapeutic interventions. In a subsequent study, these researchers demonstrated that cognitive therapy in conjunction with ERP was not more effective than either treatment alone (Emmelkamp & Beens, 1991).

More recently, researchers began examining the efficacy of Beckian cognitive therapy (Beck, 1976) for OCD, with the first study suggesting that Beckian cognitive therapy was as effective as self-controlled ERP (Van Oppen et al., 1995). Other investigators also conducted similar studies and have reported similar findings (Cottraux et al., 1990; Freeston et al., 1997; Whittal et al., 2005; Wilhelm et al., 2005). Thus, cognitive therapy seems as effective as ERP in reducing symptoms of OCD.

### ***Predictors of Treatment Non-response***

Despite the effectiveness of BT and CT, they are not effective for all patients. Researchers have begun to examine predictors of non-response (e.g., Abramowitz, 1997; Castle et al., 1994; De Araujo, Ito, & Marks, 1996; Kohls, Bents, & Pietrowsky, 2002; Mataix-Cols, Marks, Greist, Kobak, & Baer, 2002; Steketee, Chambless, & Tran, 2001). However, one of the challenges of integrating this literature is the absence of a standardized definition of treatment response across clinical trials.

Treatment outcome is typically quantified as decreases in YBOCS scores from pre- to post-treatment. However, there is no consensus as to the magnitude of YBOCS drop required before a patient can be labeled a “responder.” For example, some studies have classified a patient as being treatment respondent if his/her YBOCS score dropped by 20% (Pigott et al., 1990) versus other trials where patients’ YBOCS would need to decrease by 50% in order for patients to be classified as responders (Kozak et al., 2000). To shed light on this dilemma, Tolin, Abramowitz, and Diefenbach (2005) conducted signal detection analyses of YBOCS in an attempt to identify a percentage drop in YBOCS consistent with adequate sensitivity and specificity. These researchers suggest that patients should be labeled “treatment responders” if their YBOCS scores decrease by 30% or more after treatment (Tolin et al.). As highlighted by these researchers, this drop in YBOCS is not equivalent to symptom remission, as most patients who met this criterion still had a post-treatment YBOCS score above 16, which is the conventional cut-off score to warrant a diagnosis of OCD. Thus, it is likely that these patients whose OC symptoms have decreased by at least 30% have responded to treatment but their symptoms severity might still remain within the mild-to-moderate severity range.

Notwithstanding these discrepancies, researchers have identified some variables that seem to bode poorly for CBT. Despite early speculation that overvalued ideation might be associated with poor treatment outcome (Foa, 1979), more recent research suggested that although poor insight might be associated with more severe symptoms (Eisen, Rasmussen, Eisen, & Rasmussen, 1993; Minichiello, Baer, & Jenike, 1987), it has not always been a predictor of poor treatment outcome (Basoglu, Lax, Kasvikis, & Marks, 1988; Foa, 1979; Kozak & Foa, 1994). Still some maintain that patients who hold strong beliefs about feared consequences might be more difficult to treat (Foa, Abramowitz, Franklin, & Kozak, 1999), and that research examining poor insight has been limited by the lack of empirically validated measures of insight. However, new measures of insight such as the Brown Assessment of Beliefs Scale (BABS; Eisen et al., 1998) appear promising and might aid researchers in clarifying the association between lack of insight and treatment outcome. In fact, recent data suggest that patients in India with low insight had poor outcome to drug treatment for OCD (Kishore, Samar, Reddy, Chandrasekhar, & Thennarasu, 2004). This is an area of research that warrants further investigation.

There are some data to suggest that comorbid anxiety and/or mood symptoms might be associated with treatment non-response. Specifically, some studies have suggested that severe depressive symptoms negatively impact treatment outcome (Foa et al., 1983) and some researchers have hypothesized that OCD patients with many depressive symptoms might have slower habituation curves (Foa et al.). However, other treatment outcome studies have failed to reveal differences between OCD patients who were high versus low in depressive symptoms (Foa, Kozak, Steketee, & McCarthy, 1992; Steketee & Shapiro, 1995). Although the data on depressive symptoms are mixed, there is strong evidence suggesting that a comorbid diagnosis of MDD has a deleterious impact on treatment outcome (Eisen et al., 1999; Steketee et al., 2001). When working with depressed OCD patients,

it is advisable for clinicians to design case formulations that address depressive symptoms in addition to OCD symptoms.

PTSD may also be associated with treatment non-response for patients with OCD. Results of small studies and case reports to date suggest that patients who have comorbid OCD and PTSD may respond poorly to CBT (Gershuny, Baer, Jenike, Minichiello, & Wilhelm, 2002; Gershuny, Baer, Radosky, Wilson, & Jenike, 2003; Sasson et al., 2005).

Data also suggest that some personality disorders might predict poorer treatment outcome for patients with OCD. Some studies have documented a negative effect of borderline personality disorder (Hermesh, Shahar, & Munitz, 1987) and schizotypal personality disorder (Minichiello et al., 1987). However, attempts to replicate these findings have not been successful (Steketee et al., 2001). Similarly, it is unclear if comorbid OCPD has a negative treatment outcome, given that data have been contradictory (Mancebo et al., 2005).

Although more research is needed before definitive conclusions can be drawn about the predictive utility of specific comorbid disorders, it seems reasonable at the present time to suggest that clinicians consider the potential influence of comorbid conditions when designing treatment plans.

### ***Challenges in Treatment Delivery for OCD***

It is not uncommon for OCD patients to exhibit treatment-interfering behavior that substantially hinders treatment progress. OCD patients are often ambivalent about treatment. They may wish to decrease the distress and interference of the disorder, but they may be quite reluctant or fearful to challenge long-standing beliefs and refrain from compulsions. For example, patients who report that they are motivated to seek treatment might still frequently cancel sessions. Therapists should address cancellations immediately. Setting clear goals for treatment or creating a treatment contract that includes length and frequency of sessions may help prevent these problems (Wilhelm & Steketee 2006). However, if cancellations are frequent, therapists should address non-compliance by explaining the relationship between treatment compliance and improvement. Therapists should consider using cognitive strategies to help patients to explore dysfunctional beliefs that might be associated with treatment-interfering behaviors. Cognitive strategies such as downward arrows (Greenberger & Padesky, 1995) can help the patient to explore these beliefs, which might lead to better treatment compliance.

If treatment compliance becomes an issue later in therapy, then the patient might have irrational beliefs about getting better. As illustrated in Table 1, some patients have a fear of positive expectations or believe that they do not deserve to get better. They might therefore begin avoiding sessions when they start to improve. A therapist might want to consider using the downward arrow and Socratic questioning together to help the patient explore his/her thoughts about treatment non-compliance (for a description of these techniques, see Beck, 1995). For example, in the

aforementioned case, Maria initially suggested that she was afraid that she would never get better, which in turn would mean that she was doomed to be a bad mother. After several sessions marked by improvement, Maria still had a difficult time acknowledging that she was getting better. In fact, Maria began to fear that, even if she were to continue improving, she would inevitably relapse.

Cognitive restructuring was initially used to help Maria identify cognitive distortions related to her thought that improvement in treatment had a direct relationship to her role as a mother (e.g., overgeneralization, fortune-telling) and to collect evidence for and against these distortions. As therapy progressed, the downward arrow strategy was used to help Maria identify her core belief related to fear of failure in her role as a mother. Below is an example of this interchange.

T: Maria, I have been so impressed by your progress. You were able to hold your baby near the microwave several times in the past week. Yet, you mentioned that you are afraid that you will relapse.

P: Yes, I have been thinking that the progress has been too good to be true. In fact, I am sure I will relapse in the next few weeks.

T: Okay, I can see how that thought would make you anxious. How about we spend a few minutes exploring the meaning of this thought for you? What would that mean to you if you relapsed?

P: It would mean that I failed treatment.

T: And if you have a thought such as "I failed treatment," what would that mean?

P: That I might never be able to get better.

T: And what would that mean for you if you would never get better?

P: It would mean that I will continue to be sick and not be able to take care of the baby.

T: What would it mean for you if you were not able to take care of your baby?

P: It would mean that I am a horrible mother that cannot even take care of her baby.

T: What would it mean about you, if you were a bad mother?

P: It would mean that I am a failure.

T: Okay, Maria this is an important point. It looks like you are afraid to improve in treatment because you are afraid that if you were to relapse, it would mean that you failed, which in turn would mean that you are a bad mother. Is that right?

P: Yes!

T: I can understand why this thought makes you so anxious. Why don't we spend a few minutes examining how this core belief of being a failure is getting in the way of our work together?

The therapist continued this dialogue by using Socratic questioning to help Maria examine the evidence for and against her core belief and to find alternative ways to think about her progress in therapy. Furthermore, Maria and the therapist

collaboratively designed a behavioral experiment that challenged Maria's core belief by helping her collect actual evidence against this belief.

Another treatment-interfering behavior is session tardiness. Patients often get stuck ritualizing at home or in the bathroom at the therapist's office and thus are late for session. Patients might also cancel at the last minute because they feel overwhelmed about coming to session or are stuck in a ritual. Therapists should address this behavior right away. For example, therapists might discuss alternative hypotheses for reasons why they are not coming to session on time. If the patient suggests that they have a difficult time managing their time, then time management skills (such as breaking down the steps needed prior to coming to session) can help the patient improve attendance. Alternatively, if the patient is stuck ritualizing before session, then the therapist might want to create contingencies that might help the patient come in on time such as telling patients that the appointment is scheduled for an hour earlier to increase the likelihood that they will be on time for session.

Homework compliance is a key predictor of treatment outcome for patients with OCD. However, given that exposure-based homework can be unpleasant, it is not uncommon for patients to avoid it. One way to address homework non-compliance is to engage the patient in problem solving by conducting a pros-cons analysis of completing homework. It is also helpful to engage patients in a discussion about their short-term versus long-term goals. If the patient has set specific goals for treatment, the therapist can also use these goals to encourage the patient to complete homework. Metaphors can also be helpful. For example, the therapist could suggest to the patient that completing homework is like riding a bike, or learning to swim or drive. At first, such new activities can be somewhat anxiety-provoking. However, with practice, it is likely that they will become "second nature" and less anxiety-provoking. Regardless of the approach, it is very important that therapists validate patients' attempts to complete any homework. It is fine to be empathetic about the challenges involved with homework, but it is essential for homework non-compliance to be addressed before moving on. Below is an example of ways a therapist might address homework non-compliance with OCD patients

T: Maria, what do you think got in the way of you doing the homework this week?

P: I had a hectic week, so I just didn't have any time.

T: Do you think that this will happen regularly, that your life is so busy that it will be difficult to fit in the homework necessary for this therapy?

P: It is difficult for me to find extra time. I am always tied up either at work or with the kids, who always need my attention. By the evening, I just feel exhausted.

T: I can understand how difficult it can be to find time. But I think it is important for us to figure this out before moving on. Perhaps, Maria, this is not the time for you to be working on the OCD problems.

P: Oh no, I have to work on the OCD! I am concerned about the time I spend on my rituals and that my kids will learn some of them from me. It broke my

heart when my daughter told me that she didn't want to play with her toys because it took me so long to put them away "the right way."

T: Okay, so it sounds important to you and your family as well. So, let's try to brainstorm ways that you might find some time. If you think back to this past week, can you think of any way you might have found time to do this assignment?

P: I guess I could have asked my husband to watch the kids for a while in the evening.

T: Do you think he would be willing to do that?

P: Well, I guess he might not like it but he also hates the OCD. So, I think if I tell him I need time to complete my assignment to get better, he might be willing to help me out.

T: Great idea, Maria. How confident are you that you will be able to complete the assignment for the next week?

P: 90%.

T: Okay. I think these are great odds. I am also very confident that you will be able to complete the assignment. Let's write that down as the first item in your homework this week: discuss child care with husband tonight, and identify when he would be willing to watch the children so you can do your OCD homework.

This kind of dialogue conveys several messages to patients. It teaches them that homework is important and that it is their responsibility to complete it. In addition, it suggests to patients that you understand their difficulties, while at the same time conveying the notion that you take homework seriously. Finally, it models problem-solving skills.

At times, patients will change the topic to less anxiety-provoking subjects as a way to avoid discussing the OCD. One way to make sure to stay on topic is to set a collaborative agenda in the beginning of the session. The therapist can also ask the patient for permission to interrupt and redirect to the agenda, especially for patients who get distracted easily. If despite these efforts patients continue to derail, then the therapist and patient must assess if the OCD is the primary concern for the patient. If not, the patient might need other treatments prior to addressing the OCD.

A very common challenge in working with patients with OCD is perfectionistic standards held by the patient, which can interfere with their interpretations of progress in treatment. Patients might say or believe, "I still have a long way to go; a little progress means nothing; I will never be able to get better." Such statements might be representative of core beliefs about being a failure, or reflect past unsuccessful therapy experiences. One way to address such concerns in the beginning of therapy is to demystify progress in treatment by directly discussing patients' expectations for progress versus what progress might realistically be like. It is important to highlight to patients that there will be ups and downs in treatment but that they would end up overall doing much better than if they were not in treatment. The therapist can suggest that a monitoring form will be used weekly to monitor change over time. This can be very important for perfectionistic patients, as data might help

them acknowledge progress. In addition, the therapist should include a discussion of lapses in treatment as normal and expected, and perhaps suggest that these are times in therapy when fine-tuning can be productive. If the therapist believes that the patient's core belief of being a failure is getting in the way of treatment, then cognitive strategies such as downward arrow or behavioral experiments might help the patient identify dysfunctional thoughts and challenge the core beliefs.

T: Last session we discussed your difficulties at work. You mentioned that you are getting behind at work because you keep checking and rechecking your memos, letters, and e-mails because you want to make sure that they are perfect. During our session, you came to the conclusion that this behavior was excessive and that the consequences of keeping this up was worse than perhaps making the mistake. So, I was thinking it might be helpful for you to conduct an experiment to test this belief. Are you willing to try this?

P: Does that mean I will have to do something different? I am always so scared to do something different.

T: It makes sense that you would fear doing something different. So, perhaps we should start small and see what happens. For example, how would it feel to send a memo to your close co-workers where you purposefully misspell something?

P: I guess I could do that. I am always fixing prepositions, and making sure I get them correct. I guess I could use an 'in' when I know I should use 'on'.

T: Great idea! Let's begin there. This would mean that you write the e-mail and leave a wrong preposition. I want to remind you that this also means that you would not reread it more than once, and you would leave the mistake there, right?

P: That will be hard to do, but I am willing to try it.

In summary, regardless of patient's initial motivation for treatment, CBT for OCD will challenge the patient's dysfunctional beliefs and ask him/her to behaviorally confront anxiety-provoking situations. Thus, treatment itself is likely to be difficult for patients, which might generate several of the therapy-interfering behaviors described above. It is important for clinicians to be mindful of what they are asking patients to do and to find ways to collaboratively engage OCD patients in the challenge of designing and implementing the strategies that can help them improve, at a level that is appropriate to the patients' abilities at the time.

### ***Future Developments***

OCD is a chronic disabling psychiatric disorder that has received increased research attention in the past few decades. Despite the plethora of data, there is still more research that needs to be done. An active area of research is treatment response as described above. There is still no widely accepted definition in the literature of

what constitutes treatment non-response or partial response. As discussed by Tolin et al. (2005), there is little consistency in the way different researchers categorize treatment response, with some studies using stringent YBOCS cut-off scores and others using looser values. This makes it difficult to compare findings across different research studies. Similarly, leading OCD experts have begun to investigate how to best define treatment-resistant OCD (Sookman et al., 2005; Wilhelm et al., 2006). When several experts were asked about this, there was a wide variability in responses. Thus, the Obsessive Compulsive Cognitions Working Group is in the process of trying to create a uniform definition of treatment-resistant OCD to explore it more consistently across sites

Finally, although the treatment efficacy for OCD has come a long way, there are still many patients who do not benefit sufficiently from treatment. In an attempt to improve treatment efficacy, researchers have begun to explore the role of D-cycloserine (DCS) in augmenting behavioral treatments (Hoffman et al., 2006; Ressler et al., 2004). DCS is an antibiotic that has shown to be effective in enhancing learning in animal trials. Researchers have hypothesized that it might aid in exposure-based treatment for OCD (Wilhelm et al., work in progress). Although results are still preliminary, DCS augmentation of BT appears to be a promising avenue for improving treatment outcome (Wilhelm et al., work in progress)

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# Avoiding Treatment Failures in PTSD

Claudia Zayfert and Jason C. DeViva

*Tom is a 27-year-old married but separated Caucasian male U.S. Army veteran who presented for treatment because he is tired all the time and wants help with his sleep. He feels sleepy during the day, often nodding during breaks at work, though it often takes him several hours to fall asleep at night. He stated that when he does sleep he has nightmares about his service in Iraq. He went overseas with the initial invasion force and participated in combat and house-to-house raids. He also provided aid to injured civilians on several occasions. He keeps to himself at work and in his daily life and as a result has minimal social support. He thinks others will judge him negatively because of his war experiences, and he also gets irritated by the questions civilians tend to ask (e.g., “Did you kill anyone?”). He stated that he would never use “drugs” but disclosed drinking heavily two or three nights per week in order to get thoughts about Iraq out of his head. He smokes marijuana occasionally, usually to stop unwanted thoughts or to calm himself when he is in social situations.*

*His wife separated from him 2 years after he returned from Iraq because “she didn’t know who I was anymore.” Their 4-year-old son lives with her, and Tom is sometimes glad for that because he is scared of the possibility that he might physically abuse his son the way he was abused as a child. However, Tom does think the abuse was good for him in at least one way – it taught him to “be strong” and not give in to his feelings. Most days he wakes up feeling exhausted and would like to stay in bed, but he manages to summon energy to face the day. He often has trouble focusing at work and has to put a lot of effort into suppressing thoughts of his experiences in Iraq. On a few occasions this month, he left work early because he was suddenly reminded of his best buddy from the army who died during the first month of their deployment when his vehicle hit an improvised explosive device alongside a road. Tom still feels responsible because he believes he should have known there would be a danger on that road. His early departures from work lead to conflicts with his supervisor and he has been put on probation at his job as a manager in an office-supply company because he has not accomplished his duties.*

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*Tom presents as a well-dressed, athletically built male appearing about his stated age. He is physically healthy except for minor lower back pain from a fall he sustained in Iraq where he landed awkwardly on the pack he wore on his back. He feels guilty about feeling this pain because so many others were injured while fighting, and because his pain comes from a fall, he doesn't "deserve" to feel it. He is leery about any treatments more intensive than sleep medication because he's not "crazy" or "weak" and doesn't need "any of that other stuff."*

## Introduction

Posttraumatic stress disorder (PTSD) is a highly prevalent anxiety disorder that often follows exposure to traumatic events. There is a pressing need to make effective treatments available to individuals suffering from this disabling condition and the various problems that frequently accompany it. Cognitive-behavioral therapy (CBT) is currently the treatment of choice for PTSD. Several meta-analyses (Bisson et al., 2007; Bradley, Greene, Russ, Dutra, & Westen, 2005) have concluded that there is a solid empirical basis for the use of CBT to treat PTSD, and clinical practice guidelines recommend use of cognitive-behavioral strategies such as prolonged exposure therapy or cognitive therapy (Foa, Keane, Friedman, & Cohen, 2008; Management of post-traumatic stress working group, 2004).

Despite the accumulating evidence of its efficacy, much work remains to achieve the goal of widespread clinical use of CBT for PTSD. For example, Rosen et al. (2004) surveyed VA health care providers and found that few PTSD specialists routinely provided exposure therapy or cognitive therapy, and the rates of PTSD specialists providing these treatments actually declined after the publication of clinical practice guidelines advocating their use. Similarly, a survey of practicing psychologists in the United States found that only a small proportion of providers use exposure therapy in the treatment of PTSD, and this was true even among providers with a cognitive-behavioral orientation (Becker, Zayfert, & Anderson, 2004).

The various challenges that clinicians face when implementing CBT for PTSD may contribute to reluctance to use CBT, particularly with complicated cases, which may be more prone to treatment failure. Our aim in this chapter is to provide guidance to clinicians who wish to implement CBT with the range of PTSD patients they encounter in clinical practice. Successful treatment often depends on the clinician's ability to integrate knowledge of empirical findings with analysis of the individual case to meet the unique challenges that patients present. We will (1) review the core components of CBT, (2) consider challenges clinicians may face when implementing CBT, including available data on risk factors for poor outcome, (3) explain how to use a case formulation approach to guide treatment with complicated cases to optimize treatment results, and (4) discuss several key strategies and tools for troubleshooting when treatment derails from the intended plan.

## Core Components of PTSD Treatment

Understanding of the cognitive-behavioral models that support CBT is essential for the clinician to determine whether treatment is proceeding in a manner consistent with a successful outcome and, if necessary, to make adjustments to avert dropout or failure. We will focus on prolonged exposure and cognitive restructuring as first-line treatments because these are the most widely studied elements of CBT for PTSD. Some CBT treatment programs also include relaxation and/or stress management components (Blanchard et al., 2003; Foa, Rothbaum, Riggs, & Murdock, 1991), but research has not shown that adding these components enhances efficacy (Foa et al., 1999).

Exposure therapy is based on cognitive-behavioral models of PTSD (Foa & Hearst-Ikeda, 1996), which hypothesize that cognitive fear networks (programs for escaping danger) develop following exposure to life-threatening events. These theories propose that PTSD develops due to inaccurate fear networks, whereby the individual erroneously associates benign stimuli with danger. Maladaptive interpretations result when trauma survivors interrupt processing of trauma-related stimuli before they are able to incorporate corrective information that would lead to more accurate conclusions. Thus, the goal of exposure therapy is to activate the fear network (i.e., experience fear elicited by trauma cues) and integrate corrective information about safety to promote new learning.

Exposure therapy for PTSD typically includes both imaginal and in vivo components. Imaginal exposure entails recalling a memory of a traumatic event and evoking the associated emotions for a sustained period of time. Typically, this is accomplished by having the individual relate the memory (usually verbally, though sometimes in writing) in as much detail as possible. The memory is repeated in session for a sustained period, typically 30–60 min, and audio recordings or written accounts are used to guide repeated exposure practice between sessions. In vivo exposure involves exposing individuals to external stimuli that trigger excessive or inappropriate anxiety as a result of their association with the traumatic event. For example, Tom avoids public places with crowds of people, because he associated those places with danger while in Iraq. At first, he might enter grocery stores on weeknights with a friend who he thinks is a safe person. Then he might enter the store on weeknights on his own, then during crowded weekend hours with his friend, and finally on the weekends on his own, until he is able to remain in the store without significant discomfort. Each time Tom practices in vivo exposure, he records his initial, maximum, and ending anxiety levels. This information is used to guide further steps in treatment.

Research has demonstrated the prominent role of maladaptive cognitions in the development and maintenance of PTSD, and these cognitions are featured in most cognitive-behavioral models of PTSD (Ehlers & Clark, 2000). An important goal of CBT is to correct misinterpretations that are learned as a result of the traumatic event. Cognitive restructuring (Beck, Emery, & Greenberg, 1985; Resick & Schnicke, 1993) is a method of learning to identify and modify misinterpretations about the meaning of traumatic events with regard to oneself and the world.

This entails teaching the patient to identify habitual patterns of thinking related to the traumatic experiences and to examine these thoughts to determine the extent to which they are realistic and helpful. Patients are further taught to respond to unhelpful thoughts with more accurate or adaptive self-talk. Some PTSD protocols focus patients' efforts on specific content areas, such as safety, trust, esteem, power, control, and intimacy (Resick & Schnicke, 1993a).

Numerous studies have provided support for the use of prolonged imaginal and in vivo exposure and cognitive therapy for treatment of PTSD (Bryant, Moulds, Guthrie, Dang, & Nixon, 2003; Foa et al., 2005; Marks, Lovell, Noshirvani, Livanou, & Thrasher, 1998; Resick, Nishith, Weaver, Astin, & Feuer, 2002; Schnurr et al., 2007; Tarrrier et al., 1999). Research has been less clear, however, regarding the benefits of combining exposure and cognitive interventions. Several studies have found that a combination of exposure with cognitive therapy produced comparable outcomes to either treatment alone (Foa et al.; Marks et al., 1998; Paunovic & Ost, 2001). In contrast, however, Bryant et al. found that a combination of imaginal exposure and cognitive restructuring was more effective than imaginal exposure alone. Subsequently, Bryant et al. (2008) compared a combination of imaginal and in vivo exposure plus cognitive restructuring to imaginal and in vivo exposure combined and to each delivered alone. They found that the combined treatment produced the best outcome.

Exposure therapy and cognitive therapy for PTSD share common elements and differ primarily in the degree of emphasis placed on exposure to trauma memories versus directly challenging erroneous cognitions related to the trauma. Most forms of trauma-focused CBT involve some element of cognitive restructuring, although various approaches differ in the degree of emphasis given to systematic restructuring of thoughts. For example, protocols for imaginal exposure typically include instructions for the therapist to follow the exposure exercise with discussion aimed at processing thoughts and reactions to the trauma memory (Riggs, Cahill, & Foa, 2006). Likewise, cognitive therapy is rarely devoid of exposure; cognitive processing therapy (Resick & Schnicke, 1993) includes writing about the trauma memory primarily to facilitate formal cognitive restructuring, deemphasizing extinction via repeated and prolonged exposure to the trauma memory. There is some evidence that an approach that favors cognitive restructuring may be more effective in ameliorating symptoms of guilt and depression (Bryant et al., 2008). Due to the simplicity of the underlying model, exposure-based treatment may be the most easily disseminated treatment. For the individual therapist, however, there is reason to consider implementing cognitive restructuring along with exposure therapy (Riggs et al.) for many cases of PTSD, particularly when guilt, shame, and depression are prominent.

## **Challenges Clinicians May Encounter when Implementing CBT for PTSD**

CBT for PTSD, particularly exposure, is unusual in that clinicians tend to have concerns about implementing it. Respondents to the Becker et al. (2004) survey endorsed multiple contraindications for the use of exposure therapy, such as

comorbid psychotic or dissociative identity disorder, severe suicidality, dissociation, current victimization or perpetration of violence, homicidality, low social support, severe numbing, and history of treatment adherence problems or non-response. Respondents perceived various complications as likely to occur with exposure, including increased symptom severity, dissociation, substance abuse/dependence, overwhelming anxiety, numbing, self-harm or suicidality, homicidality, increased desire to drop out of treatment, relationship problems, aggression, and damage to the therapeutic alliance. Cook, Schnurr, and Foa (2004) listed patient barriers and clinician “concerns and misconceptions” (p. 381) that may interfere with the use of exposure therapy specifically and research-based treatments more generally. Cook et al. cited additional patient barriers discussed in the literature, including reluctance to engage with trauma memories, poor ability to image, ongoing crises, excessive shame or guilt, belief that treatment will increase symptoms, extreme anger, emotional numbing, over-engagement with trauma memories, and history of perpetration. They also cited additional clinician barriers, such as low affect tolerance, past failure experiences with exposure, and beliefs that treatment will re-victimize, lead to decompensation, not be effective with patients with multiple traumas, take too long, move patients too quickly, or damage the therapeutic relationship.

Some concerns expressed by clinicians are not supported by research. For example, 83% of the clinicians surveyed by Becker et al. (2004) believed that participation in exposure therapy was “somewhat” or “very” likely to result in worsening of re-experiencing symptoms, 87% believed hyperarousal would worsen, and nearly 60% believed it would increase the likelihood of dropping out of therapy. The actual rates of worsening of PTSD symptoms and dropout observed among participants in randomized trials are substantially lower than clinicians expect, and worsening of PTSD symptoms during therapy has not been associated with dropout (Foa, Zoellner, Feeny, Hembree, & Alvarez-Conrad, 2002; Hembree et al., 2003). In contrast to clinician concerns, research has not identified clear contraindications of CBT for PTSD. Among many variables studied, only higher level of PTSD symptoms (particularly avoidance) (Bryant et al., 2003; Marks et al., 1998), depression (Bryant et al.; McDonagh et al., 2005), anxiety (McDonagh et al.; van Minnen, Arntz, & Keijsers, 2002), male gender, and alcohol use (van Minnen et al.) have predicted dropout. Likewise, only a few variables have been linked to poor treatment outcome: trauma in childhood or multiple previous traumas (Hembree, Street, Riggs, & Foa, 2004), prescription sedative use (van Minnen et al.), male gender, high suicidality, living alone, inconsistent therapy attendance, generalized anxiety disorder, higher levels of PTSD symptoms (van Minnen et al.), greater anger (Foa, Riggs, Massie, & Yarczower, 1995), and greater physical pain (Koch & Haring, 2008). Though some predictors of dropout or poor outcome, such as trauma history, cannot be changed, many can be modified by treatment. Also, there are efficacious therapies for major depressive disorder, generalized anxiety disorder, alcohol use, and chronic pain that may enhance outcomes if delivered prior to or simultaneously with CBT for PTSD.

Despite the demonstrated efficacy of CBT for PTSD, not all patients have a positive outcome, whether treatment is delivered as part of a randomized trial or in a clinic. Dropout rates from cognitive restructuring and exposure therapies for PTSD

tend to be comparable, but usually are significantly higher than for non-trauma-focused, supportive therapies (Hembree et al., 2003). In addition, there is some evidence that dropout rates reported in randomized controlled trials are lower than dropout rates in real-world clinical settings (Zayfert et al., 2005). Even strong advocates of CBT for PTSD note that some patient variables may make either exposure or cognitive restructuring less appropriate for the treatment of PTSD (Cook et al., 2004) (Moore, Zoellner, & Bittinger, 2004). Clinicians who are well-trained and prepared to implement CBT will nonetheless face a variety of obstacles that can hinder treatment success. The problem list may include factors that impede the initiation of CBT, such as co-occurring life problems (work stressors, relationship discord, illness of self or family members) or shame-related beliefs about having symptoms or seeking treatment. Other obstacles may interfere with implementation of CBT or with maintaining the treatment focus on PTSD. Individuals with PTSD may have difficulty trusting their therapists, be reluctant to engage in trauma-focused treatment, or present with comorbid anxiety, mood, substance abuse, and eating disorders that can contribute significantly to impairment in daily functioning and interact with PTSD. Behavioral obstacles, such as urges for self-harm or binge-eating, can emerge during treatment and distract the focus from trauma-focused interventions. In addition, some individuals engaged in trauma-focused interventions may show over-engagement with traumatic emotions (as evidenced by “flashing back” or exceedingly slow rates of extinction), activation of intense anger or shame during treatment, or, conversely, difficulty engaging with the emotions associated with the events. Indeed, the patient may not identify PTSD symptoms as the main treatment priority, or even as needing treatment at all. Trauma survivors may perceive greater interference in daily functioning due to problems with sleep, relationship dysfunction, chronic pain, or panic attacks, whereas avoidance of trauma reminders is viewed as helpful. Many trauma survivors view their ability to contain or compartmentalize trauma memories and suppress or “bottle up” emotions as signs that they are coping well with the events in their lives. In short, therapists treating individuals with PTSD will benefit by preparing to manage a wide variety of challenges.

## **Using the Case Formulation Approach to Guide Treatment**

Quite often patients bring many problems to the table and the task of organizing and prioritizing them to formulate a plan of treatment can be daunting. The case formulation approach (Persons & Tompkins, 2007) (Zayfert & Becker, 2007) is a fundamental tool for managing complex clinical presentations by organizing evidence-based formulations of specific client problems to guide treatment according to an overarching individual case formulation. Using this approach the clinician considers both the relevant evidence-based problem formulations and the unique needs of the individual seeking treatment. The therapist develops a road map for treatment that incorporates hypotheses about the interactions among multiple presenting problems and relies on the evidence-based formulations available for the

various problems. This approach produces a treatment plan that maximizes clinical utility because it enables the therapist to respond flexibly through a systematic hypothesis-testing approach. We can apply it to Tom, whose case we described at the beginning of this chapter.

### ***Case Formulation***

The first step in developing Tom's case formulation is to assemble his problem list. Tom's problems include poor sleep, nightmares, and daytime intrusions relating to events from Iraq and childhood physical abuse, social anxiety and isolation, alcohol and marijuana abuse, depressed mood, daytime fatigue, guilt, panic attacks cued by trauma reminders and social encounters, negative self-judgment because of pain and treatment needs, and back pain and associated guilt. Tom's diagnoses are PTSD, major depressive disorder, social phobia, and alcohol abuse, with further information needed to rule out marijuana abuse and an Axis II diagnosis. Treatment planning begins with the hypothesis that PTSD is the principal, or anchoring, diagnosis.

The next step in case formulation is to examine relevant evidence-based formulations of the presenting issues. In Tom's case these include cognitive-behavioral models of PTSD (Clark & Ehlers, 2004), depression (Beck, Rush, Shaw, & Emery, 1979), social phobia (Hofmann, 2007) comorbid PTSD and substance abuse (Najavits, 2002), and comorbid PTSD and pain (Sharp & Harvey, 2001). The template is individualized by including Tom's specific biological and somatic factors (pain from back injury), behavioral factors (avoiding social situations in order to avoid social anxiety or triggering questions, avoiding internal experiences by using substances), and cognitive factors ("It's my fault my friend died"; "I don't deserve to complain about this pain"; "No one will understand me"; "People can look at me and see I've done wrong"; "People are generally dangerous and not to be trusted"; "I am weak for coming in for help").

Lastly, Tom's therapist generated specific hypotheses about the relationships among the presenting problems in order to guide treatment planning. Given the absence of reported psychiatric history prior to deployment, Tom's therapist hypothesized that his traumatic experiences in Iraq interacted with existing avoidance behaviors and beliefs about the world derived from his childhood physical abuse and his social anxiety to produce his current set of symptoms. Beliefs first generated by the earlier abuse, such as "I have to be on guard all the time" and "People want to hurt me," were further confirmed by his experiences in Iraq. Tom's social anxiety was exacerbated by the perception that others could see that he had let a friend die and the expectation that others would ask uncomfortable questions about the war and would not understand his responses. Dealing with the abuse by pushing his feelings aside and being "strong" as a child predisposed him to avoid the emotions associated with his Iraq experiences, which served to maintain and increase through negative reinforcement the anxiety, guilt, and shame related to those experiences. Beliefs about his inability to cope with his own emotions, as well as negative thoughts about people and the world, led to a general hopelessness.

Hopelessness, grief over the loss of his buddy in Iraq, and withdrawal from positive social connections and leisure activities also contributed to depression. The therapist further hypothesized that the strong emotions associated with the trauma memories, as well as the efforts to “not think about them,” kept their associated memory networks highly potentiated and made them salient in Tom’s mind. Tom used alcohol and marijuana to achieve a sense of control over the memories and emotions and to avoid thinking about them.

Tom’s therapist planned to begin with several sessions of psychoeducation with the goals of normalizing Tom’s reactions to the war as an early way to decrease shame, providing a conceptual framework that would allow him to see the connections among his symptoms, and maximizing the likelihood he would see treatment as necessary and acceptable. The treatment would then shift to a brief course of emotion-regulation skills based on dialectical behavior therapy (Linehan, 1993a). Given the presence of shame and guilt about treatment as well as about the trauma, Tom’s therapist planned to implement cognitive restructuring with the aim of attenuating distress related to shame and guilt and preparing Tom to focus on fear during subsequent exposure. Treatment would then shift to prolonged exposure therapy, which would begin with environmental cues Tom avoided, such as hot places, bearded men, and sand. Next, exposure would target memories of war experiences and nightmare content related to the war and childhood trauma. Finally, exposure would focus on social situations that Tom avoids. Because substance abuse was hypothesized to function as a means of managing PTSD, the therapist decided not to target it directly but rather to have Tom keep a diary of his use throughout treatment, so that adjustments could be made if it increased. Similarly, depression was hypothesized to be secondary to issues that would be targeted in the initial plan, and therefore might be expected to improve in response to these interventions. Thus, the therapist opted to monitor depression levels and suicidality weekly, and consider additional interventions focused on mood if depression persisted beyond resolution of PTSD. Self-reported PTSD symptom levels, nightmare frequency, sleep impairment (including daytime fatigue), alcohol use, and daily activity would be used to assess outcome and, if necessary, inform adjustments to the initial plan.

## **Strategies and Tools for Troubleshooting**

As indicated above, an array of challenges face clinicians in implementing CBT for PTSD. We will discuss some of the common issues that emerge and strategies for handling them and refer the reader elsewhere for further guidance (Zayfert & Becker, 2007).

### ***Handling Multiple Trauma Memories***

Like Tom, the majority of trauma survivors report multiple traumatic events and/or events that were prolonged or repeated over time. Such patients often experience symptoms relating to more than one memory. For example, Tom reported weekly

nightmares about his time in Iraq as well as distress triggered by seeing parents disciplining their children. Though they may be significant, the therapist may not be aware of events not reported by the patient. Some patients disclose only the least painful events during the early stages of assessment, or may not consider other events to be significant. Maintaining focus on less distressing events can serve to divert attention from a more painful event. In addition, the treatment setting may affect inquiry or disclosure of traumas. For example, because Tom served in a war zone and sought treatment at a Veterans Affairs Medical Center, he assumed it was only permissible to discuss military trauma. Had his therapist not specifically inquired about childhood and non-combat military trauma, he might have failed to identify clinically important events.

Thorough assessment of trauma exposure and re-experiencing symptoms from the outset will help the clinician to identify the array of potentially traumatic experiences throughout a patient's life that may be relevant to treatment. When all identified traumas have been addressed in treatment and symptoms persist despite successful extinction of distress related to them, other undisclosed traumatic events may be the subject of re-experiencing and avoidance. The therapist also should consider the possibility of undisclosed events when a patient's symptoms do not seem consistent with the reported traumatic events (as when a woman who presents for treatment of motor-vehicle-accident-related PTSD reports that she avoids being alone with men).

One of the most important tasks of treatment planning is to identify the specific memories and stimuli to target to maximize the overall treatment effect. Patients then rate the distress associated with these intrusive memories, distressing dreams, and avoided stimuli, and these ratings are used to construct hierarchies that will guide the progression of exposure. The therapist works together with the patient to select the memories and stimuli that are most suitable for exposure and are candidates to produce generalizable treatment results. Although starting with the most distressing memory on the hierarchy will enhance downward generalization to other (particularly similar) memories or stimuli on a hierarchy, beginning with a moderately distressing memory has the advantage of providing a less aversive initial experience, thereby enhancing enthusiasm for the exposure process.

The therapist should conduct periodic re-assessment of re-experiencing symptoms using a standardized self-report measure (administered at least monthly, ideally weekly) and review the hierarchy after achieving anxiety reduction for a particular memory to determine the next memory to target with imaginal exposure. In a scenario where the patient shows engagement and fear reduction during exposure and completes assigned exposure homework but shows little improvement in PTSD symptoms, it is possible that relevant traumas were not identified, or the hierarchy shifted during treatment. Additional memories may emerge or increase in prominence during treatment. After intensive work on one memory, a patient may be more "ready" to address other, perhaps even more distressing memories. For example, Tom's therapist understood from him that a day-long firefight with many casualties in Iraq was the worst event that he had experienced. After several weeks of exposure to memories from this day and extinction of anxiety related to this memory, Tom's

PTSD symptoms showed no change. Upon discussion, his therapist became aware that Tom was increasingly bothered by a previously undisclosed memory of trying to save a child who eventually died from sniper fire and that this had become a more prominent source of distress.

### ***The Influence of Other Emotions: Sadness, Guilt, Shame, and Anger***

Though PTSD is currently classified as an anxiety disorder, an array of other emotions, including sadness, guilt, shame, and anger, are frequently sources of distress for trauma survivors (Holmes, Grey, & Young, 2005). Research on the interplay of various trauma-related emotions is in its early stages. Emotions other than anxiety not only are frequent and prominent aspects of peri-traumatic responding, they also are likely to play a significant, albeit as yet under-studied, role in maintaining the disorder. For example, Andrews et al. found that shame and anger experienced in the first month after a criminal assault were the strongest predictors of PTSD 6 months later (Andrews, Brewin, Rose, & Kirk, 2000). Therapists should, therefore, assess these emotions and be prepared to grapple with them in addition to anxiety. Identifying these emotions at the outset of treatment will be conducive to developing a treatment plan aimed for success. This can be done with specific assessment instruments, such as the Trauma-Related Guilt Inventory (Kubany et al., 1996), the guilt and anger items of the Clinician Administered PTSD Scale (CAPS) (Blake et al., 1990), or unstructured clinical interview. Therapists should listen carefully for trauma-related beliefs associated with anger, guilt, or shame (e.g., “It shouldn’t have happened that way,” “It was all my fault”) that patients may express during the assessment process or during imaginal exposure.

Non-anxiety emotions have the potential to interfere with the effectiveness of exposure therapy (Foa et al., 1995; Grunert, Weis, Smucker, & Christianson, 2007) or decrease the patient’s motivation to disclose or confront specific trauma-related beliefs or memories. If the patient exhibits a pattern of initial activation of anxiety during exposure followed by little or no decrease in reported distress over time (i.e., no within-session extinction), the therapist should consider the possibility that reported distress predominantly reflects other emotions. The absence of facial expressions of fear is a further clue and is predictive of poor outcome from exposure therapy (Foa et al.). Failure to show reductions in peak anxiety across sessions is another indicator that other emotions may be activated during exposure, influencing ratings of distress and interfering with fear reduction. It is important to detect these emotions when they emerge in order to facilitate processing them effectively.

#### **Sadness**

Intense sadness often is associated with depression, a comorbid problem for approximately 50% of patients seeking treatment for PTSD (Zayfert, Becker, Unger, & Shearer, 2002). The dilemma for the clinician in treating comorbid depression with

PTSD is whether the depression is so severe that it warrants targeted treatment prior to trauma-focused CBT, or whether it will resolve when PTSD is successfully ameliorated (Foa et al., 2005). Examining the issues underlying the individual's sadness will help the clinician decide how to manage sadness and depression and avert treatment failure due to worsening depression during trauma-focused treatment. Sadness can be associated with grief over a loss incurred during the trauma (such as Tom's grief over the death of his army buddy). Sadness that is connected to grief is likely to habituate during exposure (Shear, Frank, Houck, & Reynolds, 2005) and need not be an obstacle to proceeding with either exposure or cognitive therapy; indeed, protocols for cognitive processing therapy often contain an optional session specifically addressing traumatic grief. If, however, the grief is about loss of a meaningful life experience or role, such as the loss of one's childhood or the loss of the soldier's role, then the therapist may also consider strategies aimed at recreating life meaning.

Sadness may also be related to loss of self-worth in connection with traumatic events, and in this way is often connected to shame and/or guilt. For example, for most of his life, Tom had a sense that he was "no good," something he repeatedly heard from his father. This sadness was related to shame. Since returning from Iraq, he has struggled with feeling responsible for his buddy's death, and this magnified his low appraisal of his own worth ("I didn't deserve to live when I let him die"), an example of sadness related to guilt. Sadness related to guilt and shame about aspects of the trauma can often be successfully reduced using cognitive restructuring to target maladaptive beliefs about self-worth connected to the trauma.

### **Shame and Guilt**

Guilt and shame are two distinct, though related, emotions that are often experienced in relation to traumatic events. Guilt involves feelings of remorse or regret accompanied by the belief that one has done something "wrong" or "bad" and that one should have thought, felt, or acted differently according to internal standards (Kubany & Watson, 2003). Guilt is distinct from shame in that the focus is on behavior, while the self-concept remains intact. In contrast, shame, typically associated with an urge to hide from others, is a more devastating and painful emotion in which the entire self, not just the behavior, is negatively evaluated. Shame theoretically involves feelings of worthlessness and powerlessness, and shame (but not guilt) is associated with depression (Tangney, Wagner, & Gramzow, 1992).

Individuals who feel intense guilt or shame about a traumatic event may be highly motivated to avoid thinking about the event, thereby interrupting processing of the memory. Guilt and shame interfere with engagement with anxiety, so when they become the focus during imaginal exposure they can prevent new learning, thereby impeding therapy progress (Riggs et al., 2006). In some instances, disengagement may take the form of dissociating completely from the memory and even from the present reality, such that the patient is no longer mentally present in the therapist's office. It is important to note that guilt may be present but may not activate during exposure therapy, thus causing no interference with treatment. Indeed,

outcome studies have demonstrated that exposure therapy reduces overall levels of guilt (Taylor, 2004). There is some evidence, however, that cognitive processing therapy may be more efficacious than exposure for decreasing trauma-related guilt related to hindsight bias or perceived lack of justification for one's behavior (Resick et al., 2002).

## **Anger**

Clinical lore long held that angry patients were not good candidates for exposure therapy (Jaycox & Foa, 1996). In support of this, Foa et al. (1995) found that when anger is activated during exposure the patient disengages from anxiety and distress ratings elevate but do not diminish, a process which impedes the effectiveness of exposure therapy. As in the case of guilt, however, anger that does not activate during exposure may not impede treatment success. Evidence suggests that patients with high levels of anger often benefit from exposure and are likely to experience reduction in anger as a result (Cahill, Rauch, Hembree, & Foa, 2003). Thus, as with guilt, it is important for therapists to be aware of the presence of anger and its potential for interfering with treatment and then to monitor possible effects anger may have on treatment progress. Clues that a patient is experiencing anger during imaginal exposure include non-verbal behavior such as facial expressions, tone of voice, or actions. Also, when anxiety ratings do not decline during exposure, the clinician may inquire whether the ratings reflect anger. For example, during exposure to a memory of being abused as a child, the therapist noted that Tom's distress ratings did not change over several sessions despite homework compliance. The therapist asked about his thoughts and feelings during exposure, and Tom reported that he was angry with his father for having ruined the family. As a result of his focus on anger, he no longer felt anxious when recalling this memory.

## **Overcoming Guilt, Shame, and Anger**

When guilt, shame, or anger is prominent, the therapist should consider whether to include cognitive restructuring or imagery rescripting (Arntz, Tiesema, & Kindt, 2007; Grunert et al., 2007; Rusch, Grunert, Mendelsohn, & Smucker, 2000) in the initial treatment plan, or pending response to exposure interventions. Although imagery rescripting can take various forms, the concept entails altering disturbing images associated with the trauma in a manner that enhances the individual's sense of mastery or control of the experience. Recent data suggest that augmenting exposure with imagery rescripting produces greater effects on anger, guilt, and possibly shame (Arntz et al., 2007). If exposure is implemented first and the patient's anxiety fails to extinguish across exposure sessions, the therapist should assess whether other emotions have been activated. If guilt or shame has been activated, the therapist should consider postponing exposure and using cognitive restructuring or adding imagery rescripting to decrease guilt and shame. If re-experiencing symptoms persist, resume exposure.

If anger has been activated, the therapist should start by encouraging the patient to “refocus” away from the anger to the primary emotions associated with the memory (Riggs et al., 2006). It is often helpful to validate the empowering effects of anger. For most people, anger feels better than feeling frightened or vulnerable. In particular, men are socialized to view emotions such as sadness and fear as signs of weakness; in contrast, anger is more socially acceptable for men to feel and express.

If the patient is unable to maintain focus away from the anger, the therapist should next inquire as to its source. Often, patients are able to identify another emotion – such as loss, shame, or powerlessness – that underlies the anger and that the anger allows them to avoid. If the primary emotions associated with the memory can extinguish via exposure (e.g., fear, sadness), the therapist can encourage the patient to focus on the underlying emotion instead of the anger. With his therapist’s help, Tom was able to identify a strong sense of loss of his childhood and his family life underlying the anger that emerged during exposure to a memory of abuse. After acknowledging and validating this loss, Tom was able to focus on the fear he felt during that episode and exposure resumed. If shame or guilt underlies anger, cognitive restructuring may facilitate processing of those emotions so that exposure may resume. Validating anger should always be part of this process. In some cases, focusing on acceptance of past wrongs, injustices, or losses and examining consequences of maintaining attention on anger can help the patient to “let go.”

Sadness, anger, shame, and guilt that emerge in response to PTSD symptoms or the decision to seek help often are involved in perpetuating PTSD symptoms and can interfere with treatment success (Ehlers & Clark, 2000). Many cultures (for example, the military) have expectations that individuals should be able to tolerate significant discomfort and seeking mental health care is a sign of individual weakness. Patients may feel ashamed of their perceived weakness, sad about the fact that they need treatment, or guilty for not coping more effectively. Clinicians should listen for statements or questions about the implications of seeking mental health care (e.g., “I can’t believe I’m in a place like this;” “Does this mean I’m crazy?”) or ask directly about such attributions. Often, validation coupled with psychoeducation can alleviate anxiety about the help-seeking process. Cognitive restructuring may be necessary to decrease distress and increase the likelihood of treatment completion.

### ***Facilitating Affect Regulation***

Several of the strategies for dealing with sadness, shame, anger, and guilt depend on the patient being able to attend to and discriminate various emotions. Many patients with PTSD have deficits in these areas. Trauma survivors, particularly those with a history of childhood abuse, may lack basic emotion-regulation skills. They tend to have difficulty regulating intense emotions and they may have difficulty recognizing and responding effectively to emotions (Tull, Barrett, McMillan, & Roemer, 2007). The absence of these basic skills can impede trauma-focused therapies. Cloitre, Koenen, Cohen, and Han (2002) noted that difficulty tolerating and managing unpleasant emotions, higher likelihood of dissociation, and difficulty

developing and maintaining a good working relationship are characteristics of both childhood abuse survivors and patients who do not fare well in exposure-based treatments. In addition, survivors of childhood abuse were more likely than other patients with PTSD to score high on measures of expressed and subjective anger, which, as noted above, may complicate exposure therapy (Chard, Weaver, & Resick, 1997; Franklin, Posternak, & Zimmerman, 2002).

When treatment is not proceeding smoothly, the therapist should attend to indicators of poor affect-regulation skills (Chard et al., 1997; Franklin et al., 2002). These can include difficulty disentangling emotions, being overwhelmed by emotions for long periods between therapy sessions, or dissociating in the face of moderately intense emotion. Such patients may have trouble engaging with anxiety during exposure therapy or may fail to extinguish anxiety during exposure. They also can have trouble identifying relevant thoughts to challenge in cognitive restructuring. Difficulties with trust and poor interpersonal skills that often are features of poor affect regulation can result in problems maintaining an effective therapy relationship. This may in turn affect acceptance of the treatment rationale and willingness to take the necessary risks in therapy, or result in interpersonal conflicts that distract from the therapy activities.

Broadly speaking, the therapist can modify the patient's skill level or modify the treatment. Modifying the patient's skill level usually entails adding treatment components that are designed to increase distress tolerance and the ability to experience and manage emotions. Several approaches have provided affect-regulation skills training before delivering CBT for PTSD (e.g., Chard et al., 1997; Cloitre et al., 2002; House, 2006). Similarly, we have found that integrating components of dialectical behavior therapy (Becker & Zayfert, 2001) (Linehan, 1993b) or acceptance and commitment therapy (Orsillo & Batten, 2005) can improve patient's ability to tolerate focusing on trauma and consequently may increase their likelihood of completing treatment. Emotion-regulation approaches are grounded in theory and research, though their specific benefits for enhancing treatment of PTSD awaits further research. Nonetheless, they offer the clinician constructive options for working with challenging cases.

### ***Working Through Psychosocial Crises***

Emotional or pragmatic crises often arise during treatment and many patients with PTSD lack adequate coping skills to manage them. For example, patients sometimes lose their jobs, housing, or marriage, are notified of re-deployment to a war zone, or experience a new trauma, such as an accident or assault in the course of treatment. These crises can adversely affect treatment in two different ways. First, they may interfere with session attendance or homework compliance. Patients may report that they did not have time to complete assignments or come to session because they were out looking for a job or trying to call their lawyer. Second, psychosocial crises can divert session content away from interventions formulated to target symptoms. Protocols for exposure and cognitive therapy address the symptoms that are thought

to cause patients' difficulties with everyday stressors, but do not directly address those stressors. A focus within session on those stressors therefore represents a departure from the treatment plan.

Commonly, treatment protocols advise that clinicians respond to psychosocial crises by noting the potential for treatment interference, explaining that decreasing PTSD symptoms will improve ability to cope with the crisis and maintaining focus on PTSD treatment (Riggs et al., 2006). This can be effective if the patient agrees with the rationale, has adequate problem-solving skills, does not become emotionally overwhelmed, and is able to maintain focus on the therapy. However, many PTSD patients lack the problem-solving skills to resolve crises and the affective regulation skills to modulate the intensity of their emotional responses. As a result, life problems can quickly escalate and derail treatment. Pre-treatment functioning is an indicator of overall problem-solving ability and emotional stability that can inform decision-making in this area. Patients who were not functioning well before treatment often have difficulty maintaining trauma treatment focus under crisis conditions. It is sometimes necessary to suspend trauma-related treatment and provide a brief course of training in problem-solving and affect-regulation skills, with the clear message that these skills can be applied to the patient's new or ongoing psychosocial crises in order to maintain focus on the trauma-related treatment goals that will bring greater long-term emotional stability.

### ***Substance Use Disorders***

Rates of substance use disorders (SUDs) are higher among individuals with a history of trauma than in the general population (Chilcoat & Breslau, 1998; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), and rates of PTSD are higher among individuals with SUDs (Greeley & Oei, 1999). The potential for substance use to interfere with treatment of PTSD symptoms has been the subject of much theory and research (Cloitre, Miranda, Stovall-McClough, & Han, 2005). The chemical effects of the substance may prevent the patient from focusing attention on treatment materials and experiencing arousal. Patients may use substances to avoid unpleasant thoughts or emotions associated with re-experiencing symptoms or trauma treatment. Alcohol use has been related to negative outcome of anxiety treatment (Keijsers, Kampman, & Hoogduin, 2001; van Minnen et al., 2002) and thus it is not surprising that active substance dependence is a common exclusion criterion in controlled trials of PTSD treatments.

The effect of substance use on treatment varies in relation to the severity of the use and the function it serves. A thorough assessment of substance use, abuse, and dependence, including the extent to which substances aid avoidance of trauma-related emotions, is key for developing a clinically useful PTSD case formulation. The assessment information should help the therapist to determine the potential for interference with PTSD treatment and whether specific substance use treatment should precede trauma-focused treatment. For a patient who is using daily and

unable to work or maintain their activities of daily living, detoxification and intensive substance-abuse treatment should be initiated before trauma-focused treatment. When patients have detoxified from substances and have maintained a period of abstinence (many specialized PTSD programs require 30 days of sobriety before admission), the case formulation approach may be employed to elucidate the relationship between PTSD and substance use and to assess the deficits (e.g., poor affect regulation, minimal problem-solving skills) that place the patient at higher risk for using substances as a way to cope. For patients in the early stages of recovery, there are useful treatment approaches that combine the development of skills to maintain sobriety in the face of trauma-related symptoms with early education on how trauma and substances interact (Batten, 2009; Najavits, 2002).

Substance use need not be severe enough to warrant detoxification in order to interfere with PTSD treatment. Even patients who work full-time and fulfill familial responsibilities may use substances as a way to decrease thoughts and emotions associated with trauma. The substance use does not merit intervention, but it has the potential to interfere with trauma-focused treatment. In many cases, trauma-focused treatment can proceed by including discussion of the role of substance use in the individual's avoidance of trauma-related thoughts and emotions and monitoring substance use throughout treatment. Tom's case is an excellent example of this. His drinking caused few problems in his life, yet his therapist noted that it could provide a means of avoiding uncomfortable beliefs and emotions when completing therapy homework. During the education and rationale phase of treatment, Tom and his therapist discussed the function that his alcohol use served, and Tom agreed not to drink for the duration of treatment. He also agreed to monitor his urges to drink using a daily diary during treatment and to inform his therapist if his urge to drink increased. Although many clinicians strictly adhere to a policy of achieving complete sobriety before addressing trauma, many individuals are not able or willing to achieve complete abstinence before PTSD treatment, or even afterward. Ongoing collaborative discussions around the role of substances in managing PTSD symptoms will help the therapist guide the client toward optimal decisions.

### ***Titrating Engagement with Emotions During Exposure Therapy***

Parameters of treatment can be modified to facilitate. As noted above, emotional engagement is an important ingredient for successful outcome from exposure therapy. Yet over-engagement also can impede the progress treatment of therapy, as some patients will be unwilling to continue exposure without experiencing some relief of their distress. Failure of distress ratings to diminish across exposure trials is predictive of poor outcome (Jaycox, Foa, & Morral, 1998). There are a variety of reasons patients may over- or under-engage traumatic memories and emotions, some of which have been described earlier (e.g., poor ability to regulate emotions, efforts to avoid memories or the accompanying emotions, beliefs about the potential consequences of experiencing emotions, such as losing control). One approach is to

initiate exposure as soon as possible and then problem-solve to remove any obstacles that may present during the procedure. An advantage of this approach is that patients may experience reduction of distress more quickly, which can enhance their confidence in the treatment. A disadvantage of providing exposure immediately is that patients who have difficulty titrating affect are “set up” for failure, which is not conducive to a good treatment experience (Lee, 2006). An alternate approach is to assess thoroughly before initiating exposure and seek to eliminate obstacles before beginning. The disadvantage here is that delay of symptom reduction may increase likelihood of dropout. Indeed, Zayfert et al. (2005) found that patients who started exposure therapy in a clinical setting were more likely to complete CBT for PTSD successfully than patients who did not start exposure therapy.

Patients who under-engage typically present the trauma account with minimal visible or self-reported emotional arousal. Important and likely uncomfortable details (e.g., what a soldier was thinking after he was shot; the initial penetration during a sexual assault) may be missing entirely, and the account of the experience may be brief. The patient may provide the “6 o’clock news” version of the trauma, a short summary that glosses over anything difficult. There are a number of strategies therapists can implement to increase engagement with trauma-related emotions so as to maximize the likelihood of treatment success (Jaycox et al., 1998). Minimally, the therapist may remind the patient of the rationale for exposure and the consequent importance of engaging with the memory and related emotions. The therapist may also modify the parameters of the exposure exercise, asking the patient to relate the memory in the present tense, with eyes closed, and with a greater detail of events, thoughts, and feelings. Asking questions during the exposure can increase focus on particular details, thoughts, or emotions. Cognitive restructuring of thoughts about fear of losing control of emotions can help the patient feel less threatened by the task of engaging. Similarly, assessing for and targeting guilt, shame, or anger may enable the patient to engage with the trauma memory more directly. For those who are high in anxiety sensitivity (fear of fear), it can be useful to engage the patient in interoceptive exposure exercises, such as hyperventilation, to expose the patient to the physical sensations of fear independent of the trauma memory and stimuli (Barlow & Cerny, 1988).

Patients who are over-engaged will report extremely high levels of anxiety during imaginal exposure and may express concern about the effects of experiencing emotions at high levels. Over-engaged patients may also dissociate during imaginal exposure, causing them to lose engagement with the memory and the associated emotions. The main goal in working with over-engaged patients is to establish and maintain a sense of safety during exposure activities so that the patient is able to access and engage the trauma memory. The therapist should emphasize to patients that though distress (and PTSD symptoms) may increase in the early stages of treatment, it tends to decrease for most patients as treatment progresses and the increase in symptoms does not mean they will not ultimately benefit (Foa et al., 2002; Nishith, Resick, & Griffin, 2002). The therapist may also take steps to titrate anxiety, such as instructing patients to keep their eyes open while recounting the trauma, interacting verbally with them during exposure, beginning the exposure in written form, and

dividing the memory into manageable segments. In some cases, it may be useful to make a plan in advance that if the patient begins to “flash back,” the therapist will gently touch them on the leg or shoulder to re-orient them. Once anxiety has extinguished under modified conditions, exposure can resume in its original form (e.g., verbally with eyes closed).

## Summary

Cognitive-behavioral therapies have been shown to be efficacious for treatment of PTSD. However, there are a number of obstacles that can interfere with the implementation of evidence-based CBT for PTSD or that predict poor outcome of treatment. Some of these obstacles can be overcome by providing patients with skills to manage them, and others can be navigated by flexible application of existing treatment protocols. The case formulation approach is an important treatment planning tool for clinicians faced with patients with multiple problems. A thorough assessment of trauma history, PTSD symptoms, and comorbid problems informs working hypotheses about the relationships among presenting problems. These working hypotheses guide formulation of treatment plans, and continual assessment of change during treatment may lead to revision of these hypotheses and adjusting the treatment plan accordingly. Clinicians should, whenever possible, adhere as closely as they can to the structure of evidence-based CBT, while maintaining flexibility to respond effectively to roadblocks to treatment success.

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# Avoiding Treatment Failures in Social Anxiety Disorder

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## The Case of Paul

Paul<sup>1</sup> was a 24-year-old white male. He was of medium build, attractive, and cooperative during the interview. He was well articulated but spoke very softly and avoided eye contact with the interviewer. The main reason for which Paul sought help was for debilitating and pervasive social anxiety that he had been experiencing since middle school. Paul reported that he desired companionship but avoided any social contact with other people for fear of being criticized or rejected. He even felt restraint when interacting with his parents and other relatives. When asked why he was seeking help, he noted that he wanted to move out of his parents' home, get a job, have relationships, and lead a normal life. It was apparent that Paul showed a clear readiness for change and had clear short-term and long-term goals for himself. However, he viewed his competence to implement such changes as insufficient. Paul was offered group treatment at the clinic, which is the standard treatment for social anxiety disorder. However, he refused to participate because he was too fearful of interacting with other group participants.

## *Psychosocial History*

Paul's psychosocial history revealed that his social anxiety had interfered to a significant degree with his life. He reported that he dropped out of high school in tenth grade when he was asked to give a presentation in front of his class. In order to avoid this assignment, he ran away from home and spent the night in a local park. When the police found him the next day, his parents told him that he did not have to return to school. Since that time, Paul had been living at home with his parents. He did not return back to school and did not see any of his classmates again.

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<sup>1</sup>This is a real case, but the name of the patient and other identifying information was altered. A detailed description of this case is given in Hofmann & Scepkowski (2006).

Paul described the relationship with his family as difficult. Both of his parents were born and raised in Greece and immigrated to the United States when they were in their twenties. Paul's family spoke Greek at home and followed Greek traditions. He described his father as politically conservative, domineering, authoritarian, and over-involved, and his mother as submissive and conflict-avoidant. Paul reported having two older sisters. He considered his second oldest sister to be his best friend. He reported that he spoke to her once a week for a few minutes on the phone. His family was unaware of the fact that he was seeking treatment for his social anxiety.

In addition to social anxiety, Paul also reported that he felt depressed and that he worried excessively about various minor matters. Paul stated that, due to his problems, he had occasional thoughts about suicide but denied any intent or plan. Paul had never received any psychological or pharmacological treatments.

### ***Diagnostic Information***

As part of the regular intake procedure, Paul underwent a structured diagnostic interview, the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV, DiNardo, Brown, & Barlow, 1994). A second interview by an independent clinician was conducted one week later. The two interviews yielded consistent results and senior clinicians agreed with the diagnostic assessment. The ADIS-IV screens for all Axis I mood and anxiety disorders and assigns a clinical severity rating (CSR) on a scale from 0 (no distress/interference) to 8 (extreme distress/interference). A CSR rating of 4 or higher marks the clinical threshold. Paul met the criteria for the following Axis I diagnoses: Social Anxiety Disorder (Social Phobia), generalized subtype (CSR: 8); Generalized Anxiety Disorder (CSR: 5); Depressive Disorder Not Otherwise Specified (CSR: 5). Furthermore, he met criteria for Avoidant Personality Disorder (CSR: 8) on Axis II and scoliosis (S-shaped side-to-side spinal curve) on Axis III. No other patient at our center has ever received the maximum CSR ratings for both the social anxiety disorder and avoidant personality disorder (APD) diagnoses. In addition, the raters coded problems related to the social environment and occupational problems on Axis IV and assigned a Global Assessment of Functioning score of 45 (current and past year) on Axis V (serious symptoms or any serious impairment in social, occupational, or school functions).

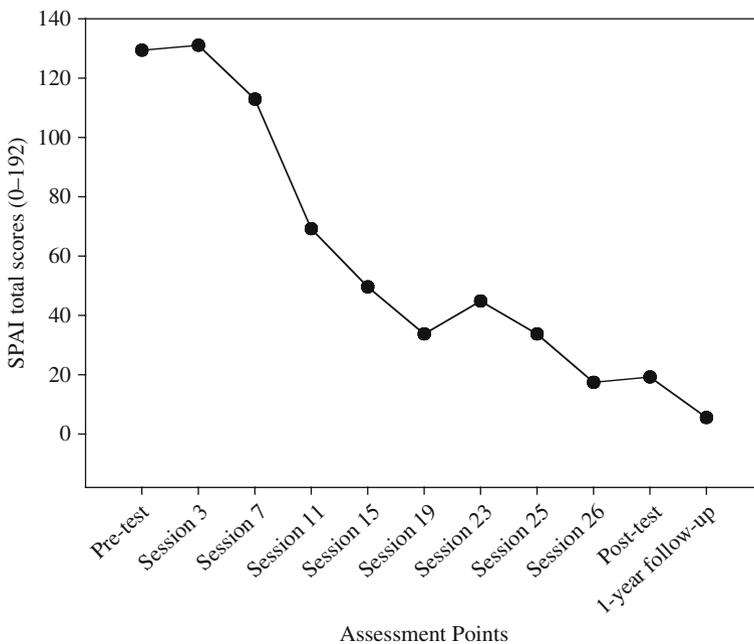
### ***Case Conceptualization***

During the interview, Paul made a number of remarks suggesting that he had a distorted image of himself. Although his attractiveness was above average, he saw himself as physically unappealing, socially inept, and inferior to others.

Paul's case showed many prototypical features of social anxiety disorder (SAD). Some of his concerns were focused on his physical appearance, which are often associated with body dysmorphic beliefs. Although the diagnostic criteria for body dysmorphic disorder were not specifically assessed, there is good evidence to

assume that Paul would have met the diagnostic criteria. As a result of these concerns, he feared and avoided social contact, but at the same time greatly suffered from social isolation. He also felt that he lacked the social competence and skills required to improve his social situation. The resulting social isolation and withdrawal was associated with, and perhaps even caused, his depressive symptoms and other anxiety symptoms. Due to the diagnostic overlap between SAD and avoidant personality disorder (APD), it is not surprising that he also met criteria for APD.

In summary, Paul represented an extreme case of SAD that highlights many commonly occurring examples of complications that might arise when treating SAD. Specifically, he had a distorted and negative view of himself and he was socially isolated and highly avoidant of social contact. At the same time, he greatly suffered from social isolation, which was closely associated with his feelings of depression. As a result, he showed features of body dysmorphic disorder, and met additional diagnostic criteria for APD and depression. Despite these significant complicating factors, Paul made remarkable improvement. In order to track Paul’s progress, he was given the Social Phobia and Anxiety Inventory (SPAI; Turner, Beidel, Dancu, & Stanley, 1989) at approximately every third session and at pre-test, post-test, and 1-year follow-up. Turner et al. suggested that a SPAI (total) cut-off score of 80 is optimal for identifying individuals with SAD among a sample of clinic patients with anxiety disorders. Figure 1 shows that the intervention



**Fig. 1** *Case of Paul.* Paul’s treatment changes in the Social Phobia and Anxiety Inventory (total score). Reprinted with permission from Hofmann and Scepkowski (2006)

led to a significant drop within the first 3 months of therapy (by session 11) from the clinical to the non-clinical range. These gains were maintained at the 1-year follow-up. Paul underwent a second diagnostic assessment at the end of treatment by a blinded clinician as part of a regular clinic procedure. At that time he no longer met any DSM-IV Axis I or II criteria. This case illustrates that, despite a number of treatment complications that will be outlined below, SAD can be effectively treated with creative techniques if they specifically target these complicating factors.

## Psychological Interventions

Contemporary theories of SAD emphasize the role of cognitive processes in the maintenance of the disorder (Clark, 2001; Leary, 2001; Rapee & Heimberg, 1997). Cognitive-behavioral treatment (CBT) protocols based on this approach show a number of similarities and notable differences. One of the first CBT protocols, Cognitive Behavioral Group Therapy (CBGT) by Heimberg (e.g., Heimberg & Becker, 2002), is an adaptation of Beck and Emery's (1985) cognitive therapy for anxiety disorders to SAD. This intervention is administered by two therapists in 12 weekly 2.5-h sessions to groups comprised of six patients and consists of several distinct but interwoven treatment components. In the first two sessions, patients are taught the Beckian CBT model as applied to SAD, and are given instructions in cognitive restructuring techniques. Specifically, patients practice identifying negative cognitions (automatic thoughts), observing the co-variation between anxious mood and automatic thoughts, examining the errors of logic, and formulating rational alternatives to their automatic thoughts. The remaining ten sessions entail largely exposure exercises, in which patients confront increasingly difficult feared situations such as public speaking (simulated in the therapy group) while applying these cognitive restructuring techniques. Behavioral experiments such as these are utilized to confront specific reactions to these exposure experiences. At the end of each exposure-based session, the therapist and group members agree on individualized assignments for exposure to similar real-life situations during the week. Patients also complete self-administered cognitive restructuring exercises before and after each behavioral homework assignment.

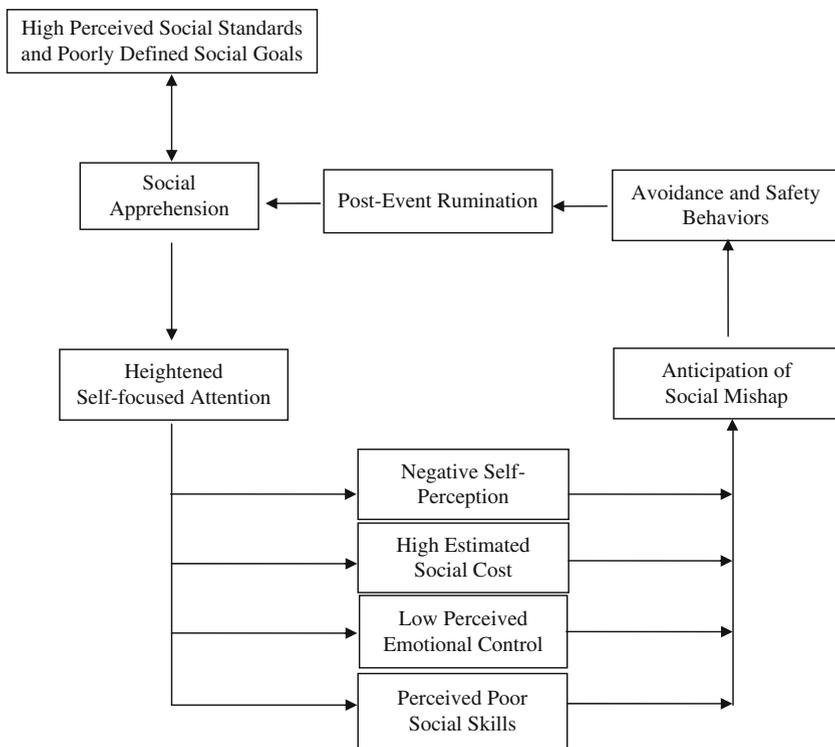
This method of intervention has stimulated a great amount of research interest. In standardized protocols, CBGT has been shown to be at least as effective as the most efficacious form of pharmacotherapy. On the other hand, however, there is clearly still room for improvement. For example, in the most recent study on the efficacy of CBGT, 133 patients with SAD were randomly assigned to phenelzine (Nardil, an MAOI commonly used to treat SAD), Educational Support Group Therapy, a pill placebo, or CBGT (Heimberg et al., 1998). After 12 weeks, both the phenelzine (65%) and the CBGT conditions (58%) had higher proportions of responders than pill placebo (33%) or Educational Support Group Therapy (27%), which served as a psychotherapy placebo condition. The criterion for treatment response was based

on a 7-point rating of change on the Social Phobic Disorders Severity Change Form (Liebowitz et al., 1992). Patients rated as markedly or moderately improved were classified as responders.

A recent modification of Heimberg's CBT protocol is Comprehensive Cognitive Behavioral Therapy (CCBT; Foa, 1994). This treatment protocol was included as a treatment condition in a recently published clinical trial (Davidson et al., 2004). The treatment protocol is derived in part from an earlier version of CBGT (Heimberg & Becker, 2002) and combines exposure techniques, Beckian cognitive restructuring therapy, and social skills training. The intervention differs from CBGT primarily in that it includes specific social skills training in addition to the conventional cognitive restructuring exercises and exposure tasks. Furthermore, the role-plays are shorter and the treatment is two sessions longer than CBGT. The study by Davidson et al. suggests that Foa's treatment shows efficacy rates that are similar to CBGT. Specifically, the study randomized 295 patients with generalized SAD to one of five groups: (1) fluoxetine, (2) CCBT, (3) placebo, (4) CCBT combined with fluoxetine, or (5) CCBT combined with placebo. The results showed that all active treatments were superior to placebo, and the combined treatment was not superior to the other treatments. The response rates in the intention-to-treat sample (using the Clinical Global Impressions scale) were 50.9% (fluoxetine), 51.7% (CCBT), 54.2% (CCBT/fluoxetine), 50.8% (CCBT/placebo), and 31.7% (placebo). These findings are comparable to the clinical trials examining "traditional" CBT approaches (i.e., CBT protocols that are based on more generic, Beckian-style CBT for anxiety disorders). Although the treatment efficacy data were relatively modest, CBT was similarly effective than pharmacotherapy. Interestingly, combining CBT and pharmacotherapy was not more effective than monotherapy.

In an attempt to enhance the efficacy of the CBT approaches for SAD, some investigators have more recently developed protocols that are designed to better target disorder-specific factors of SAD that have been identified in experimental studies (Clark, 2001; Hofmann, 2007). One such treatment model is depicted in Fig. 2.

According to this model, individuals with SAD are apprehensive in social situations in part because they perceive the social standard (i.e., expectations and social goals) as being high. They desire to make a particular impression on others but doubt that they will be able to do so (Leary, 2001), partly because they are unable to define goals and select specific achievable behavioral strategies to reach these goals (Hiemisch, Ehlers, & Westermann, 2002). This leads to a further increase in social apprehension and increased self-focused attention (Clark & McManus, 2002; Heinrichs & Hofmann, 2001; Hofmann, 2000; Hirsch & Clark, 2004; Woody, 1996), which triggers a number of additional cognitive processes. Specifically, vulnerable individuals exaggerate the probability and potential social costs involved in social situations (Foa, Franklin, Perry, & Herbert, 1996; Hofmann, 2004). This is consistent with the model by Clark (2001), which assumes that individuals with SAD assume that they are in danger of behaving in an inept and unacceptable fashion and believe that this will result in disastrous consequences. In addition, the model



**Fig. 2** *Treatment model.* Psychological factors that maintain social anxiety disorder. Reprinted from Hofmann (2008)

posits that individuals with SAD perceive little control over their anxiety response in social situations (Hofmann, 2005; Hofmann & Barlow, 2002), hold a negative view of themselves as social objects (Hofmann, Moscovitch, Kim, & Taylor, 2004), and view their social skills as very poor or inadequate to master the social task. As a result, the individual with SAD anticipates social mishaps and engages in avoidance and/or safety behaviors (Wells et al., 1995), followed by post-event rumination (Mellings & Alden, 2000; Rachmann, Grüter-Andrew, & Shafran, 2000). This leads to a positive feedback loop, leading to the maintenance and further exacerbation of social avoidance and anxiety.

An unanswered question for the field is whether a particular focus on modification of social cost can boost treatment response beyond that seen in other protocols. Comparative treatment trials have not yet been conducted to show the additive benefit with the same group of patients, but independent trials have shown particularly promising effect sizes suggesting that more patients may be able to achieve remission with this approach. For example, Clark et al. (2003) developed an individual treatment approach consisting of 16 sessions that primarily focuses on modifying safety behaviors and self-focused attention, in addition to the conventional CBT strategies. The Clark et al. trial randomly assigned 60 patients with generalized

SAD to one of three conditions: (1) cognitive therapy alone, (2) fluoxetine combined with self-exposure, or (3) fluoxetine combined with placebo. Treatment efficacy was measured by calculating a composite score that was based on six frequently used self-report measures of SAD and a rating based on a structured clinical interview. The results at post-treatment and 12-month follow-up assessments showed that cognitive therapy was superior to the other two conditions, which did not differ from one another. The results showed that the uncontrolled effect size of the severity rating based on the clinical interview was 1.41 (pre-test to post-test) and 1.43 (pre-test to 12-month follow-up) in the cognitive therapy group. Even stronger effects were found for the composite score, which was associated with an uncontrolled pre-post effect size of 2.14. Similar results were reported in an open-label pilot trial with ten patients with SAD who received a 12-session group therapy with five participants per group (Hofmann & Scepkowski, 2006). This intervention resulted in a substantial reduction of social anxiety with effect size estimates ranging between 1.54 and 2.37. These results suggest that it is possible to improve the treatment effects of CBT by targeting cognitive variables that appear to be specific maintaining factors for SAD and that have not been systematically addressed in more conventional CBT protocols.

## **Potential Predictors of Poor Treatment Response**

### ***Generalized Subtype and Avoidant Personality Disorder***

APD is an Axis II condition which is commonly comorbid with SAD and highly overlapping in symptomatology with the generalized subtype of SAD. Studies have found that both the generalized subtype of SAD and APD are associated with poor overall psychosocial functioning, greater overall psychopathology, high trait anxiety, and depression (e.g., Boone et al., 1999; Brown, Heimberg, & Juster, 1995; Herbert, Hope, & Bellack, 1992; Holt, Heimberg, & Hope, 1992; Turner, Beidel, & Townsley, 1992). It has been suggested, therefore, that this additional diagnosis may simply represent increasingly severe manifestations of social anxiety, which range on a continuum from specific (nongeneralized) SAD, to generalized SAD without APD, to generalized SAD with APD (Hofmann, 2000b; McNeil, 2001; Hofmann, Heinrichs, & Moscovitch, 2004).

Whereas some studies support the notion that APD is a predictor of poor treatment response (Alden & Capreol, 1993; Feske, Perry, Chambless, Renneberg, & Goldstein, 1996) other studies have not shown APD to be a significant moderator of treatment change (Brown et al., 1995; Dreessen & Arntz, 1998; Hofmann, Newman, Becker, Taylor, & Roth, 1995; Hope, Herbert & White, 1995; Mersch, Jansen, & Arntz, 1995; Van Velzen, Emmelkamp, & Scholing, 1997). Similar inconsistent results have been reported in studies investigating the generalized subtype as a predictor of poor treatment outcome (Brown et al., 1995; Gorman, Liebowitz, Fyer, Campeasa, & Klein, 1985; Liebowitz et al., 1992; Turner et al., 1992; Uhde, Tancer, Black, & Brown, 1991).

## ***Depression***

Depression is highly comorbid with SAD (Kessler, Stang, Wittchen, Stein, & Walters, 1999), and there is evidence to suggest that a diagnosis of SAD increases the risk of subsequent depression (Alpert et al., 1999; Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Stein et al., 2001). Furthermore, a study by Chambless and colleagues found that a high level of depression is a predictor of poor SAD treatment response (Chambless, Tran, & Glass, 1997). In contrast, however, Erwin, Heimberg, Juster, and Mindlin (2002) did not observe a differential treatment response of SAD patients with or without depression. Instead, the authors found that SAD patients with comorbid mood disorders, but not comorbid anxiety disorders, were more severely impaired than those with no comorbid diagnosis both before and after 12 weeks of CBGT. Moreover, a study by Moscovitch, Hofmann, Suvak, and In-Albon (2005) found that changes in SAD mediate changes in depression during CBT for SAD. The role of comorbid depression in relation to SAD is covered in more detail in Chapter 10.

## ***Social Skills***

The perception of one's social skills and abilities appears to be an important component of perceived self-efficacy in SAD. Although it remains uncertain whether socially anxious individuals are in fact deficient in any of their social skills (Clark & Arkowitz, 1975; Glasgow & Arkowitz, 1975; Halford & Foddy, 1982; Hofmann, Gerlach, Wender, & Roth, 1997; Rapee & Lim, 1992; Stopa & Clark, 1993), they do tend to appraise their own performance in social situations more negatively than non-anxious individuals, even when actual differences in performance are accounted for (Alden & Wallace, 1995; Glasgow & Arkowitz, 1975; Rapee & Lim, 1992; Stopa & Clark, 1993). These results and others call into question the value of social skills training modules (Stravynski & Amado, 2001). Although social skills training seems to be effective in reducing social anxiety (Stravynski, Grey, & Elie, 1987; Stravynski, Marks, & Yule, 1982), there is no clear evidence to suggest that it is more effective than exposure therapy or cognitive-behavior therapy for reducing social anxiety, even for individuals who were judged to have poor social skills (Mersch, Emmelkamp, Bögels, & van der Sleen, 1989; Mersch, Emmelkamp, & Lips, 1991; Wlazlo, Schroeder-Hartwig, Hand, Kaiser, & Münchau, 1990).

## **Core Elements of Treatment and Common Sticking-Points in Therapy**

One of the most important components of CBT is the therapy rationale itself. Three aspects are important to convey: (1) the therapy is an active, goal-directed, behavioral-based intervention to eliminate avoidance and reduce subjective distress; (2) cognitive errors contribute to social anxiety; and (3) avoidance behaviors maintain social anxiety. The following brief dialogues will exemplify selected aspects of the treatment.

## *Motivation for Therapy*

Treatment motivation is crucial for any exposure-based therapy of highly avoidant individuals. In addition to establishing rapport with the patient and discussing the treatment model, the first session also has an important motivational goal. Discussing the treatment model, the role of avoidance for the maintenance of SAD, and the importance of exposure practices can generate a great degree of distress and increase the likelihood of avoidance tendencies and even treatment dropout. Therefore, it is important to “inoculate” the patient for avoidance behaviors. In other words, it is often useful to prepare patients for situations in the future when they will want to avoid exposure assignments, other homework tasks, or even coming to sessions. In order to counter this tendency, the therapist should explain that not doing exposure practices in sessions and homework assignments (i.e., tasks to be completed between sessions every week after the second session) and missing sessions are all forms of avoidance behavior. For example, the therapist might introduce this issue at the end of the first session as follows:

T: Before we end this session, I would like to say something that I think is very important. Avoidance has many faces. And sometimes it might be difficult to recognize a behavior as avoidance behavior. This is partly because avoidance has developed into a habit; and habits occur on a subconscious level. Avoidance behaviors are particularly hard to identify if you can give yourself other reasons why you avoided. That way, you can avoid doing something unpleasant and at the same time can tell yourself that you didn't do it not because of your anxiety, but because your car broke down, you had a deadline at work, or because your dog got sick. Your avoidance is a strong habit and it will always be easy to find reasons why you can't do it, some might be more convincing to yourself and other people than others. But the bottom line is: it is still avoidance. Every time you avoid, you are making a decision against an independent and anxiety-free life and for a life that is controlled by your anxiety. And every time you don't avoid, you are courageous and choose the hard-way with the goal to free yourself from your anxiety. I want you to be fully aware of this. Patients I have worked with who succeeded were the ones who were committed to getting better. I recommend you do the same – nothing should be more important than coming to our sessions and practicing the homework exercises.

Some patients may be resistant and hesitant to undergo therapy because they believe that this is the way they are, and changing their social anxiety would require fundamental changes in personality traits. The following dialogue illustrates this issue:

T: Social anxiety, the fear of social situations, is something really interesting. You are constantly confronted with social situations in your daily life. Just think about how often you interact with people during your day. And yet,

in the absence of treatment, social anxiety can persist for many years or decades. What keeps this anxiety going? Why don't people get used to it? Do you have any ideas?

P: I am just very shy. I have always been.

T: People clearly differ in how shy they are. But shyness itself is not the problem. The problem develops when social anxiety interferes with your life and when you can't do things you want to do because of your fear of social situations. We discussed earlier the many ways in which social anxiety interferes with your life. The goal of treatment is to make you feel more comfortable in social situations so that you can do the things you want to do. So the goal is not to change you as a person. Instead, the goal is to accept yourself the way you are in order to feel less uncomfortable in social situations.

### *Challenging Cognitive Errors*

A very common cognitive error that is often resistant to change is estimated social cost. This refers to the belief that social mishaps would lead to disastrous consequences. The following dialogue exemplifies the Socratic method of cognitive therapy to deal with this error:

P: But what if this unlikely event really does happen? What if I really do lose my train of thought and my mind goes blank?

T: Yes, good point. So what if your mind really does go blank? What do you think would happen?

R: This would be awful.

T: A real catastrophe?

P: Yes.

T: But what exactly would be so terrible about it?

P: It would be embarrassing!

T: Why would it be embarrassing?

P: Because I will make a fool of myself in front of other people.

T: What does "making a fool of yourself in front of other people" exactly mean? What would happen?

P: They would laugh about me and think that I am a total loser.

T: How do you know what other people think of you? Do you have a crystal ball?

P: What do you mean?

T: You are making a number of assumptions here that may or may not be correct. Interestingly, you choose out of many possible alternatives the one that threatens your self the most. For example, you are assuming that if you lose your train of thought, everybody will notice it, laugh at you, and think that you are incompetent. This scenario would then elicit embarrassment in you. If people don't notice that you lost your train of thought or if they do, but are not the least bit hostile, you wouldn't have any reason to feel embarrassed any more. Isn't that right?

P: I guess so.

T: Furthermore, while this scenario is not completely impossible, it is not very likely. It assumes that the social world out there is hostile and aggressive, and that people are out to get you. But let's assume for a moment that this scenario actually does happen, and that you happen to speak in front of an audience that consists of some very hostile people, and that you would indeed embarrass yourself. Then what? Have you been in any embarrassing situations before in your life?

P: Of course I have.

T: How many times?

P: Oh, many times. More than I can count.

T: And are you still alive?

P: (Laughs)

T: My point is that even if your mind does go blank, even if people notice that, and even if they are in fact hostile and think that you are an incompetent loser, which causes you a great deal of embarrassment, it is not a catastrophe. You have been embarrassed before, and so has everyone else. And life will go on.

### ***Eliminating Avoidance Behaviors***

It is recommended to define *avoidance* as anything the patient does or does not do that prevents him/her from facing his/her anxiety. This includes not entering the feared situation, escaping out of the feared situation, distracting oneself, using breathing techniques, or behaving in a way that makes oneself feel more comfortable (which are termed as safety behaviors).

Exposure situations serve a number of different purposes, one of which is providing an opportunity to practice goal setting and re-evaluate social standards. For this purpose, the therapist should discuss with the patient what the social expectations (standards) of a given situation might be, and should help the patient to state at least one clear (e.g., behavioral, quantifiable) goal (e.g., asking a particular question). At the beginning, it is important to provide very clear instructions about what the exposure task should look like. The therapist's role during these early exposures is similar to that of a movie director who provides the patient with a clear script of his/her expected behavior. If the situation requires a complex social interaction (e.g., returning an item to the same sales person minutes after it was purchased) the therapist should clearly specify when a particular action should be shown. For example, rather than simply instructing the patients to "return a book minutes after you bought it," the therapist should instruct the client to "purchase the newest Harry Potter book, walk with it toward the exit door, and when reaching the exit doors, turn around, find the same sales person again, and ask for a refund of this book by saying: 'I want to exchange this book that I just bought because I changed my mind.'" The goal of this task may be to say this particular sentence.

Effective situations for individuals with SAD differ from exposure situations to treat other phobic disorders. First, it often requires performance of complicated chains of interpersonal behavior during exposure. Second, the SAD patient's specific anxiety-eliciting situations are not always "available." For example, an agoraphobic individual may go for a walk away from home at almost any time, but the patients with SAD may confront that feared staff meeting only once weekly. Other situations may occur only sporadically and be beyond the individual's realistic control. Therefore, in-session exposure, especially at the beginning of therapy, is recommended. Public speaking or initiating and maintaining a conversation with a stranger can be easily created in the session. Further along in treatment, it is recommended to conduct in vivo exposures outside the safe environment of the therapist's office. Later exposure practices should further specifically target estimated social cost. Examples of in vivo exposure tasks to challenge this cognitive error are given below. The patient should be instructed to conduct similar and more individually tailored exercises as part of his/her homework assignments. A monitoring form will aid the patient specifically to examine and challenge the perceived social costs and probability associated with these situations. For example, the patient may be asked to order a bagel, and then to "accidentally" drop it on the floor, and ask for a new one (without paying for it) or to go to a local video store, rent a DVD, and immediately return it, saying "I forgot I don't have a DVD player." For a more extensive account, see Hofmann and Otto (2008).

## **Flexibility for Dealing with Challenging Cases**

The exposure assignments should be designed to be challenging for all patients. With adequate motivation by the therapist, patients should be able to perform the assigned exposure tasks, even if they significantly violate the patient's perceived social norms. Some patients, however, may feel unable to conduct the exposures. In those cases, the therapist should show the adequate degree of flexibility and modify the task accordingly. People who severely fear public speaking, for example, might answer simple questions that the therapist or some audience member ask rather than give an impromptu speech, or they may be asked to simply read a text paragraph in front of the audience. Conversely, the situation should be made more challenging if the patient does not experience enough anxiety or discomfort. For example, the patient may be asked to give a presentation about negative personality characteristics rather than a speech about their hobbies. Simple physical exercises prior to the exposure task (e.g., push-ups) that induce intense physical sensations and sweatiness can further heighten the anxiety during an exposure exercise. We believe the optimal level of anxiety during the anticipation phase of a social task is between 5 and 7 on a scale from 0 (no anxiety) to 10 (extreme anxiety).

Homework practices, and especially the lack thereof, are another significant challenge to treatment. As part of the homework assignments, patients are asked

to perform behaviors or place themselves in situations that were previously avoided or tolerated only with excessive anxiety. If the patient repeatedly refuses homework assignments, the therapist should discuss the importance and negotiate attainable goals and practices. During these discussions, patients should be told that homework is a very important element of this treatment and that not doing the homework is a form of avoidance. At the same time, successful exposure practices, or even simple but honest attempts, should be rewarded by the therapist. Moreover, patients should be asked to reward themselves by doing something special or buying something if they did engage in the exposure practice. Discussion of the homework is an opportunity to reinforce successful behavior and for the therapists to identify the parameters of the patient's feared situations. In addition, it provides an opportunity for the therapists to reinforce the model.

## Summary

SAD is a common and debilitating disorder. Traditional CBT approaches have shown reliable effects that are comparable to pharmacologic interventions and superior to psychological and pharmacological placebo treatments. More recent CBT protocols that have been specifically tailored to SAD demonstrated effects that appear to be stronger, although comparative treatment trials are lacking. Nevertheless, there is still considerable room for improvement. So far, empirical studies have failed to reliably identify predictors of poor treatment response or treatment complications, such as the generalized subtype, avoidant personality disorder, depression, and social skills. Instead, treatment complications may arise if patients: (1) lack treatment motivation and are deficient in setting appropriate treatment goals; (2) show cognitive errors that enhance social anxiety in response to actual or imagined social threat; and (3) exhibit avoidance strategies that lead to the maintenance of social anxiety. It is recommended to flexibly tailor CBT approaches to the individual patient and idiosyncratic psychopathology in order to maximize treatment efficacy and minimize or resolve treatment complications.

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# Avoiding Treatment Failures in Generalized Anxiety Disorder

Evelyn Behar and T.D. Borkovec

## Resolving Treatment Complications in Generalized Anxiety Disorder

Generalized anxiety disorder (GAD) first appeared as a diagnosis in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-III)*; American Psychiatric Association (APA), 1980). Since that time, programmatic research on the nature of worry and GAD has led to advancements both in its diagnostic criteria and in its treatment. With the publication of *DSM-III-R* (APA, 1987), the primary defining symptom of GAD became excessive anxiety and worry about more than one topic. Later, *DSM-IV* (APA, 1994) retained excessive worry as GAD's cardinal feature and further stipulated that the worry must be difficult to control. GAD is highly comorbid with other Axis I conditions, particularly with other anxiety and mood disorders (Kessler, 1997; Noyes et al., 1992). Thus, its pervasiveness in other conditions makes the thorough understanding and successful treatment of worry an important aspect of mental health care in general.

Although cognitive-behavioral treatments for GAD have been shown to be effective in treating this condition (Borkovec & Ruscio, 2001) and in reducing symptoms of comorbid Axis I conditions (Borkovec, Abel, & Newman, 1995), cognitive-behavioral therapy (CBT) alone evidences only a 50% rate of high end-state functioning among treated patients (Borkovec, Newman, Pincus, & Lytle, 2002). Consequently, knowledge resulting from (a) research on predictors of treatment responsiveness and (b) laboratory investigations on the etiology and maintenance of GAD has been applied in attempts to increase the efficacy of traditional CBT techniques. In this chapter, we will describe traditional cognitive and behavioral techniques, as well as more recent approaches incorporating emotional and interpersonal therapy, mindfulness-based therapy, metacognitive therapy, emotion regulation therapy, and therapy focused on increasing tolerance of uncertainty.

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Moreover, we focus on the resolution of complications in treatment by identifying predictors of poor response to treatment and highlighting methods to increase efficacy of treatment for those individuals.

## **Predictors of Response to Treatment**

Knowledge regarding the individual differences that may enhance or compromise responsiveness to treatment can assist in the development of treatment packages that are able to successfully treat a larger percentage of GAD patients. Newman, Crits-Cristoph, Connelly Gibbons, and Erickson (2006) conducted a review of the participant factors that predict responsiveness to treatment for anxiety. Among demographic variables, gender (Gould, Otto, Pollack, & Yap, 1997; Tyrer, Seivewright, Ferguson, Murphy, & Johnson, 1993) and intelligence (Haaga, DeRubeis, Stewart, & Beck, 1991) do not seem to be significant predictors of treatment outcome for GAD patients. On the other hand, findings show that high socioeconomic status predicts maintenance of treatment gains over 12 months following GAD treatment (Durham, Allan, & Hackett, 1997) and is associated with less severe GAD symptoms during the treatment phase (Tyrer et al., 1993). Also, research indicates that older patients (over 35 years of age) are less likely to drop out of treatment (Edlund et al., 2002), suggesting that therapists treating younger patients may want to pay special attention to building a strong alliance, addressing and mending alliance ruptures, providing a believable rationale for treatment, and addressing patients' motivation difficulties in therapy.

Data on whether severity of GAD symptoms appears to be a predictor of treatment outcome are mixed. Although three studies suggested that patients with higher self-reported anxiety (Butler & Anastasiades, 1988), assessor-rated anxiety (Butler, 1993), and clinician-rated global severity and number of symptoms (Yonkers, Dyck, Warshaw, & Keller, 2000) at pre-treatment responded less well to therapy, a host of other studies failed to find a relationship between outcome and symptom severity as rated by assessor (Barlow, Rapee, & Brown, 1992; Butler, 1993), Hamilton Anxiety Scale (Barlow et al.; Biswas & Chattopadhyay, 2001; Durham et al., 1997), or self-report (Barlow et al.; Durham et al.; van den Brink et al., 2002). Research on whether duration of symptoms, or chronicity, predicts treatment outcome is less equivocal. Longer duration of GAD has been shown to predict worse outcome from treatment (Biswas & Chattopadhyay, 2001; van den Brink et al.). Furthermore, the presence of comorbid Axis I conditions seems to predict poor response to therapy. For example, patients are more likely to maintain treatment gains if they lack a comorbid Axis I diagnosis in general (Durham et al.) or comorbid dysthymia or panic disorder specifically (Tyrer, Seivewright, Simmonds, & Johnson, 2001).

Certain cognitive variables have also been found to predict response to treatment in GAD. Butler (1993) found that the degree to which ambiguous external information is interpreted as threatening predicts outcome in patients being treated with CBT. Also, high internal locus of control predicts positive response to treatment

following cognitive therapy for GAD (Biswas & Chattopadhyay, 2001). Together, these findings suggest that cognitive therapy may be useful in the treatment of GAD symptoms, particularly among patients with interpretive biases and external loci of control.

Finally, several areas of research on predictors of treatment response converge to suggest that interpersonal problems predict a poor response to treatment among GAD patients. First, Borkovec et al. (2002) found that interpersonal problems remaining at treatment termination predicted poor outcomes at post-therapy and follow-up assessments. Second, personality pathology seems to play a role in response to therapy. Sanderson, Beck, and McGinn (1994) found that patients with Axis II diagnoses were more likely to drop out early from cognitive therapy for GAD. Tyrer et al. (1993) reported that personality disorder traits were associated with poorer response to cognitive therapy and self-help treatments among GAD patients. Also, findings from longitudinal studies suggest that cluster B (Yonkers et al., 2000) and C (Massion et al., 2002; Yonkers et al.) personality disorders are associated with diminished likelihood of remission from GAD. Third, research on the effects of social support converges with the above findings on interpersonal relationships. Durham et al. (1997) found that married (as opposed to single, widowed, or divorced) patients are more likely to show sustained improvement following treatment for GAD, and that among married patients, high levels of marital tension are associated with lower sustained improvement from treatment. These findings suggest that a focus on interpersonal functioning may be highly valuable in treating chronic worriers who present with troubled relationships, personality pathology, and limited social support systems.

Throughout the course of this chapter, we will highlight some of these and other predictors of treatment response and, where appropriate, suggest methods for treating individuals whose characteristics may make them less responsive to traditional cognitive-behavioral techniques. Also, as will be evident below, there are a number of variations by which treatment can be targeted and delivered, focusing alternatively on exposure interventions, cognitive interventions, and modification of autonomic functioning. At the present stage of the literature, the best guidance is that all of these strategies have efficacy, and when treatment resistance is confronted, consideration of alternative strategies within the domain of these interventions is warranted.

## **Theoretical Conceptualization of Worry**

The avoidance theory of GAD is explained in detail by Borkovec, Alcaine, and Behar (2004). Theoretical conceptualizations of GAD rest largely on Mowrer's (1947) two-stage theory of fear. Mowrer proposed that fear emerges via classical conditioning and is maintained via operant conditioning in the form of negatively reinforced behavioral avoidance of conditioned fear stimuli. Mowrer's theory is the foundation of traditional exposure-based treatments for anxiety disorders, which

seek to decrease anxious reactions to feared stimuli by repeatedly exposing individuals to those stimuli while preventing avoidance responses that would otherwise be negatively reinforced by temporary reductions in that fear.

According to the avoidance theory of worry, GAD differs from the other anxiety disorders in a key way. Whereas all of the other anxiety disorders are often characterized by *motoric* avoidance of feared stimuli, GAD patients do not typically avoid disorder-specific, discrete situations or stimuli. Instead, threat exists in the nonexistent future, and there is therefore no behavioral avoidance response at the worrier's disposal. To satisfy the need to take immediate action to cope with this threat in the absence of behavioral options, patients utilize *cognitive* avoidance (trying to come up with ways to prevent the bad outcomes or to prepare oneself for them) in response to threatening material. As a predominantly verbal-linguistic (as opposed to imagery-based) phenomenon (Behar, Zullig, & Borkovec, 2005; Borkovec & Inz, 1990), worry is remote from physiological and affective experience, and a period of worrisome activity has an inhibitory effect on physiological reactivity during subsequent anxiety-provoking tasks (e.g., Borkovec & Hu, 1990). Given the central role of physiological reactivity to emotional processing (Foa & Kozak, 1986), worry's inhibitory effects therefore are hypothesized to prevent successful emotional processing of fear and thus preclude anxiety extinction.

Patients with GAD are stuck in a variety of non-adaptive, habitual, and nonflexible ways of thinking, behaving, and experiencing emotion. Our approach to treating GAD stems from the cognitive avoidance conceptualization of worry, and from our understanding of the specific cause-and-effect relationships that contribute to the etiology and maintenance of these non-adaptive habits. Therapy techniques target those relationships in order to maximize the efficacy of interventions. The overall goal of these techniques is to replace automatic, anxiety-maintaining spirals with more adaptive, flexible, and anxiety-incompatible responses. Therapists encourage patients to repeatedly rehearse coping skills both in session and in their daily lives so that these alternative responses become habitual. Initial coping skills provide stepping stones for later therapeutic work. By loosening up their rigid, maladaptive behaviors, cognitions, and emotions, patients create new, less threatening meanings and develop more flexible ways of behaving, thinking, and feeling. As laboratory investigations have led to increased knowledge about the nature of worry, the development of interventions for GAD has focused on targeting non-adaptive areas of functioning for the GAD patient. The foundation of current cognitive-behavioral treatment entails teaching patients to objectively observe, or self-monitor, their physiological functioning, behavioral responses, cognitive processes (thoughts and images), interpersonal behaviors, and emotional experiences. By providing patients with interventions targeting many or all of these relevant response systems, the ultimate goal is to cause change within the whole, cohesive organism, so that patients can achieve freedom from ruminative worrying, experience adaptive thoughts and emotions, and have an enhanced ability to experience and process each moment of their lives as it occurs in the present moment. Below, we discuss each of these areas of functioning and describe the techniques designed to treat them effectively.

## Self-Monitoring

The cognitive world of chronic worriers is rigidly focused on threat. Worriers spend large amounts of time engaged in rumination about potential future catastrophes, they are frequently detecting threat cues in their immediate environments (Bradley, Mogg, White, Groom, & de Bono, 1999), and they often interpret ambiguous information as threatening (Mogg, Bradley, Miller, & Potts, 1994). As a result of their attentional resources being spent predominantly on the detection of threat, worriers' awareness of their present-moment reality is compromised. Furthermore, given that the success of treatment in part depends on patients' ability to attend to internal and external states so that they can implement therapeutic techniques at the appropriate moments, such non-adaptive patterns of awareness must be targeted as an initial step in treatment.

The first step in treating non-adaptive awareness is to equip patients with self-monitoring skills. In self-monitoring, the therapist helps the patient to learn to attend to his/her internal experiences and external environments and to become familiar with how different response systems interact with one another. Patients are asked to notice all thoughts, emotions, behaviors, and physiological experiences surrounding a particular situation and to note the consequences of these responses. By becoming more attuned to their automatic responses and how these responses relate to one another and to external situations, patients can become familiar with how their anxiety spirals and where and how in the sequence they can intervene. Take, for instance, the following exchange between a patient (P) and her therapist (T) after they established that one of the patient's most common triggers for worry and anxiety was waiting for her children to get home from school.

T: Adele, I'd like to help you get into the habit of being very aware of what happens before, during, and after an intense worry episode like the one you just told me about. If you're comfortable doing so, close your eyes for a few moments and try to imagine the scene you just told me about from yesterday. Imagine yourself sitting at your desk in your office. It is 2:45 pm – about 15 min before your kids are due home. Tell me what you see in the image before the worry sets in.

P: I can see my desk, and my computer. I can also see my telephone very clearly; it almost seems to be the focus point of the picture I'm conjuring up. I am trying hard to focus on the email I need to write and send out, but my eyes just keep wandering to the telephone. It's almost like I'm willing the phone to ring. I want to know that the kids are safely in the house, and that nothing bad has happened to them.

T: Good. Try to stay with that image as much as you can, and continue seeing it in your mind's eye as vividly as possible. Tell me what is going on for you internally in the situation at that moment.

P: I feel myself getting very nervous, and starting to worry that something awful has happened to the kids. I imagine my youngest child run over by a car. I

pick up the phone and call the house to see if they're home yet, but there's no response. I start to get even more worried and I call my husband, check the local news online, and make sure I have the phone number to the elementary school.

- T: As you're saying this, I can see how worried you become before 3:00 pm every day. You seem to go straight for the catastrophic thinking, landing on all sorts of awful possibilities of what may have happened. Tell me, as you're thinking about this scene, what do you notice in your body?
- P: I can feel my shoulders and my back – they're very tense, even here and now. I also feel a knot in the pit of my stomach, and I notice myself remembering all sorts of awful news stories about children who have been kidnapped or run over. I feel guilty that I'm not picking them up from school myself. . .that instead, I have a job and a life away from them.
- T: OK, so physically, you feel tension and I can also see from here that you are furrowing your brow and clenching your jaw. Your thoughts focus on your quality as a mother and on thoughts of bad things happening to your kids. Also, you mention that you engage in some checking behaviors to decrease your worry – doing things like calling your husband. What do you notice as a result of that? What does it feel like the moment you get off the phone with him?
- P: I feel better for about five seconds, because he seems to be calm and hasn't heard of any accidents, but I immediately just start to worry again when I begin to think of all the other possible sources of information I haven't checked. I just get more and more tense. Then I look at the phone, and the email I was trying to write, and start to worry all over again.

In this exchange, it is clear that the patient's anxiety spirals out of control when faced with this daily event. By imagining the scenario and the sequence of events in this and similar situations, and by generating anxiety in the session to identify specific events and reactions, the therapist can work with the patient to identify how the patient's anxiety develops from moment to moment. Therapists should encourage their patients to catch the anxiety spiral early (in this patient's case, the therapist might ask her to monitor her internal state beginning much earlier in the day in order to detect precisely when the first hint of worry and anxiety begins in anticipation of the 3:00 pm call).

Therapists should also help patients to understand the causal relationships between thoughts, feelings, and behaviors. In the exchange above, for instance, the therapist might help the patient come to the realization that her anxiety is not merely an effect of the fact that her children have to get themselves home safely. She is creating much of her own anxiety by her checking behavior, her catastrophic images, her automatic thoughts, her physiological tensing reactions, and her attempts to distract herself by composing an e-mail. This may be especially useful for patients who have a strong external locus of control and therefore may believe that there is little they can do to change their patterns (a negative predictor of treatment response;

Biswas & Chattopadhyay, 2001). By becoming aware of their own contributions to their anxiety, they may be more likely to believe that they can exert control over it.

As part of self-monitoring, therapists should frequently ask patients to use a 0–100 scale to rate the degree of tension, anxiety, and distress they experience moment to moment in the session. When therapists see or infer an increase in a patient's anxiety as issues are being discussed, they can ask “What is your number now?” in order to quickly gather information about shifts in the patient's state. This allows therapists and patients to more accurately identify precursors and consequences of anxiety, as well as to identify early stages of the anxiety spiral. This scale is also used later to assess in-session impacts of new techniques being deployed (e.g., applied relaxation, generation of new cognitions).

Self-monitoring provides an introduction to patients about focusing on the present moment. By attending to the immediate reality of the world around them, they can observe themselves and their environments objectively. They learn to accurately process information in the present and to open their awareness to this information. As therapy progresses, patients are encouraged to begin observing *all* of the information contained in the present moment, especially positive information. In contrast to their usual attention to negative aspects of the environment, which contributes to confirmatory biases that bad things will happen in the future (Faust, 1984), patients learn a more balanced processing of the present in which they can flexibly choose whether or not they want to attend to negative elements. Making the choice to attend to what actually exists frees them from their automatic responses and promotes genuine experiences – a connectedness between the environment, the self, and emotion. As discussed later, self-monitoring is also useful in teaching and facilitating emotional processing. Patients learn to attend to and fully process all emotions that occur in reaction to present-moment events. By generating perspectives that cultivate approach to daily life and joy in its engagement, patients are more likely to process positive, value-directed information.

## **Relaxation Training**

Unlike patients with other anxiety disorders, who show sympathetic activation when confronted with feared stimuli, GAD patients show a suppression of sympathetic activation during worry and during anxiety-eliciting stimuli following worry periods (Borkovec & Hu, 1990). Along with sympathetic reductions, worriers show reductions in cardiovascular variability indicative of parasympathetic deficiency (Hoehn-Saric, McLeod, & Zimmerli, 1989; Thayer, Friedman, & Borkovec, 1996) as well as reduced EMG variability (Hazlett, McLeod, & Hoehn-Saric, 1994). The only physiological elevation evident in GAD patients is in muscle tension (Hazlett et al.). Both this muscle tension and the parasympathetic deficiency are addressed in treatment by the use of applied relaxation training. Progressive muscle relaxation (PMR) and slowed, paced diaphragmatic breathing are two powerful methods that can help patients cultivate physiological and psychological states opposite to those

induced by worry and anxiety, both throughout the day in order to reduce baseline anxiety and in response to incipient anxiety spirals.

In PMR, patients are taught to tense and then let go of the tension in a variety of muscle groups. Scripts for guiding patients through PMR are available (Bernstein, Borkovec, & Hazlett-Stevens, 2000), and focus initially on 16 muscle groups that are combined gradually over sessions. One of the goals patients and therapists work toward is to eventually be able to induce a relaxed state without employing muscle tension at all, but rather by merely recalling states of relaxation. PMR can help patients develop greater ability to quickly decrease tension levels as soon as they are detected within the anxiety spiral. Furthermore, PMR can help patients create a pleasant present moment, which they can recall as they practice “letting go” of tension, negative thoughts, and catastrophic images.

Patients are also trained in diaphragmatic breathing methods. Unlike PMR, which requires patients to set aside time to complete the exercises, diaphragmatic breathing can induce a relaxed state in a very short period of time. Slowed, paced breathing through the diaphragm is compared in session to shallow, rapid chest breathing. Therapists can use the 0–100 rating scale to communicate regarding the degree of tension and anxiety patients experience during each type of breathing. Diaphragmatic breathing can be a powerful tool in demonstrating to patients that they have the power to create positive physiological and emotional states in themselves very rapidly and even during situations that previously elicited anxiety and worry.

Finally, patients are taught to practice engaging in relaxing imagery and in meditative techniques for relaxation. They are encouraged to shift flexibly between all of the relaxation techniques at their disposal so that they can use whichever techniques work best in specific circumstances, and as a way of encouraging a flexible, non-rigid lifestyle.

One complication that may arise in treatment is that particular patients may become increasingly anxious as a result of relaxation training. Research on relaxation-induced anxiety (RIA) shows that patients who become anxious during PMR show poorer responses to therapy (Borkovec, Mathews, Chambers, Ebrahimi, & Nelson, 1987). For this reason, therapists are advised to look for signs of RIA in their patients and to (a) continuously target (via PMR) those muscle groups that induce RIA if repeated practice is leading to reductions in RIA, (b) focus primarily on those muscle groups that produce less RIA if symptoms of RIA persist despite repeated practice, and/or (c) rely more heavily on diaphragmatic breathing and relaxing imagery for patients who are made anxious by the tensing of muscles.

By practicing their relaxation skills twice per day for 10 minutes each time, patients can develop the ability to intervene more successfully whenever stressful or worrisome situations occur. By eliciting these calm states frequently throughout their daily lives, patients become increasingly familiar with this tranquil state, reduce their background anxiety levels, and apply their relaxation skills upon detection of the earliest signs of impending anxiety and worry, including detection of the absence of this relaxed state. Using self-monitoring to detect incipient anxiety, patients can implement their relaxation skills to interrupt anxiety spirals.

Furthermore, by inducing calm states frequently throughout the day, patients have the opportunity to attend to the positive present moment instead of over-attending to potential threat.

## **Self-Control Desensitization**

In self-control desensitization (SCD; Goldfried, 1971), the vivid imagery and relaxation skills patients learned previously are used in combination to rehearse their coping responses. Patients are asked to vividly imagine a situation that causes them to worry and experience high levels of anxiety. While vividly engaged in imagery, the patient indicates to the therapist (e.g., by a raised finger) when he/she notices actual anxiety cues beginning to occur. The therapist then encourages the patient to continue imagining the anxiety-inducing scene, but also to vividly imagine deploying his/her relaxation skills in that scene. The patient continues engaging in this imagery until anxiety dissipates, at which point the patient indicates to the therapist (e.g., by lowering the raised finger) that anxiety is significantly reduced or absent. At this point, the therapist has the patient continue imagining relaxing in the situation for a little while longer so that the patient further experiences successful relaxation in the midst of the situation. Finally, the patient is asked to terminate the image and deepen the relaxed state. This procedure is repeated until anxious responses decrease quickly upon imagery of using relaxation skills or until the initial anxious image fails to elicit incipient anxiety cues in the first place.

Once patients begin to learn cognitive therapy techniques for dealing with their anxieties and worries (described below), during subsequent SCD image presentations they can rehearse both relaxing themselves and shifting their perspectives to more adaptive ones within the scene.

## **Stimulus Control**

Stimulus control treatments are used for a variety of emotional problems, including insomnia (Bootzin & Epstein, 2000), issues related to developmental disabilities (e.g., Falcomata, Roane, & Pabico, 2007), and eating disorders (Linden, 1980). Stimulus control treatment for worry developed in response to the fact that worry can occur anytime and anywhere. Thus, worry is under poor stimulus control. It becomes associated with countless external cues (e.g., time of day, physical location, contact with specific people), and this can in turn make worry especially easy to elicit, given that individuals are likely to come into contact with at least some of those external cues quite often. By teaching their patients stimulus control techniques, therapists can help their patients gradually limit the number of environmental conditions that come to elicit worry. These techniques have been shown to be effective for chronic worriers (Behar, Tishk, & Zalewski, 2002; Borkovec, Wilkinson, Folsensbee, & Lerman, 1983).

Two variations on stimulus control treatment can be implemented with patients. In the first, patients are asked to self-monitor throughout the day so that they can detect anxiety cues as early as possible, and they are then instructed to postpone worry to a later half-hour worry period. This half-hour “worry time” is established at a consistent time each day, in a consistent location. During this time, patients can worry intensely to provide for some habituation, but later in therapy they are encouraged to use this time instead to engage in effective problem-solving, employ techniques they learn in cognitive therapy (described later), and use other coping strategies they learn in treatment. This approach helps patients to develop greater discriminative stimulus control so that (a) worry does not pervade their lives throughout the day and (b) they are able to focus on employing effective coping techniques for their worries in a structured, time-limited setting.

A second stimulus control technique involves having patients establish a “worry-free zone,” a time or place in which they will not worry, letting go of or postponing the worry to just outside of that zone. Once the patients notice lessened worry in that zone, they establish a second worry-free zone in some other place or time during their day. They continue to add additional zones to gradually reduce the number of situations that elicit worry and anxiety. When patients are in these worry-free zones, they can employ self-monitoring, mindfulness techniques (e.g., Roemer, Salters-Pedneault, & Orsillo, 2006), and any other therapeutic skills that help them remain focused on the present moment.

## **Cognitive Therapy**

As mentioned earlier, the use of cognitive therapy may be useful in the treatment of GAD symptoms, particularly among patients with less adaptive cognitive styles (e.g., those who have strong interpretive biases and who believe that their fate is guided by external, as opposed to internal, circumstances). GAD patients’ responses on the Dysfunctional Attitude Scale suggest that they have non-adaptive core beliefs (Behar & Borkovec, 2002). Cognitive therapy for GAD (adapted from Beck, 1976) focuses on identifying core cognitions about the self, the past, and the future. To help identify these non-adaptive cognitions, patients are first encouraged to employ self-monitoring to detect incipient anxiety cues, and to then record the thoughts that preceded and followed those cues. By reviewing the themes that run through these automatic thoughts, therapists and patients can collaboratively challenge and modify those cognitions and the underlying beliefs that give rise to them. A primary goal of cognitive therapy for GAD is the generation of multiple perspectives. By generating numerous possible interpretations of an event or predictions of the future, patients can begin to loosen up some of their inflexible ways of thinking. It can be helpful in the beginning to introduce the concept of multiple perspectives by using emotionally neutral situations for brainstorming many ways of seeing the situation without regard for accuracy. Then the creation of multiple perspectives can be applied to more emotionally relevant material. Take, for instance, the case of Adele,

the patient who worried uncontrollably about her children's safety while coming home from school:

- T: This exercise of writing down your thoughts was very helpful, because I think it helped us identify what some of your knee-jerk reactions tend to be. You – along with lots of people who struggle with anxiety – seem to fall into, as you put it, “thinking traps” when things go wrong or when you’re worried about something. If it’s OK with you, I’d like to try something out. . . a small exercise. Are you willing to give this a shot with me?
- P: Sure.
- T: Great. Now, you’ve mentioned in the past that you are an avid runner.
- P: Yes, I love running. Sometimes I think it’s the only thing that keeps me sane!
- T: I’d like you to imagine that you haven’t run for ten consecutive days. Can you play the part of an unbiased observer and list for me some reasons why Adele hasn’t run in ten days?
- P: Hmmm. . . you mean you want me to just come up with possible reasons?
- T: Yes, that’s right. Just form some hypotheses, without worrying whether they are accurate or likely or acceptable. I’ll contribute some too.
- P: OK. Well, maybe Adele has been very busy. Or maybe she got shin splints and her doctor told her to lay off for a couple of weeks.
- T: Excellent. Maybe Adele has been sick and wants to recover fully before she gets back into the swing of things.
- P: Yeah, or maybe her kids have been ill and she hasn’t wanted to leave them in the house alone while she goes out.
- T: Maybe Adele is tired of running and wants to take a month or two off from it. . . or maybe even more.
- P: Or maybe the weather has been very bad and her treadmill is broken. Or maybe she is trying to gain a few pounds if she is very thin.
- T: Excellent. These are all wonderful possibilities that you’ve generated. Now, can you do the same thing regarding the *consequences* of her not running? Can you generate lots of perspectives about those?
- P: Well, she may end up gaining too much weight from not exercising. Or she may lose lots of muscle and have a hard time getting back into it. Or maybe she’ll really benefit from the time off and wind up being a better runner when she starts up again. Maybe she’ll enjoy spending the extra hour with her children and never go back to it, or maybe she’ll start to feel depressed and realize that she really needs it in her life.
- T: OK, great. Thank you for doing that. You seem to be very comfortable generating lots and lots of ways of looking at one small event or situation. Let’s shift our focus a bit now and do the same thing with one of your biggest “thinking traps” from the worry records. You seemed to have lots of knee-jerk negative thoughts about the day last week when you had to step out of your office just before 3:00 pm when your colleague needed you to sign something and you missed the phone call from your kids letting you know they were home safely. Here in your worry record, you wrote, “I am a bad

mother. . .If it had been an emergency I would not have been available and something awful could have happened. . .My children may end up thinking I am not there for them.” What I’d like to do now is have you again play the part of the unbiased observer and generate lots of ways of looking at this situation. Let’s not pass judgment on any of them, or worry too much about whether they are right or wrong. Let’s just come up with lots of ways of looking at it.

- P: [Hesitating]. . .This is harder to do when it’s something I feel really strongly about.
- T: Yes, this is definitely tougher. Why don’t you start with one, and I’ll contribute one too. Remember to take the stance of the casual, unbiased observer; that may help you think about it more easily.
- P: OK. Well, I guess I could make the obvious statement that Adele simply had to step out for a minute, and that stepping out for a minute doesn’t make her a bad mother.
- T: OK, that’s good. How about this: By not being responsive to colleagues’ needs or by not signing things on time, Adele might be viewed as a less competent worker, which may influence her job satisfaction and therefore her ability to be emotionally present for her kids.
- P: It’s also possible, of course, that Adele *is* a bad mother, and puts her work before her kids’ safety.
- T: Yep, that’s a fair one to bring up, certainly. Can you come up with another one?
- P: Hmm. Well, maybe by not always being immediately available, Adele’s kids can learn to be more independent.
- T: I’ve got one, too: Maybe the kids can leave a voice mail, and Adele will get the message as soon as she returns to her office.
- P: Or maybe something *is* wrong, but they can call back.
- T: OK, I think we could probably come up with another dozen interpretations, but let’s stop there for now. What was that experience like for you?
- P: A little weird. Usually, when something like that happens, I just think of 3 or 4 really bad things that led up to it or that can come of it, and then I get worried and start all of my usual behaviors of checking the news, calling my husband, and getting frantic. This didn’t seem natural for me.
- T: I can imagine that it would feel very foreign for something so emotional and anxiety-provoking, and for someone who doesn’t generally follow that train of thinking.
- P: At the same time, though, I found myself believing some of those, you know? Like. . .maybe it *will* make my kids a little more independent if I’m not always there checking up on them.
- T: Wow, we went from “I’m a bad mother if I’m not immediately available” to “Maybe I’m a bad mother if I am always immediately available.” It’s almost hard to know which is accurate. . .they’re both kind of believable at some level. But that is precisely the most important part of this exercise.

P: I think I know what you're getting at. Just because I have a knee-jerk reaction, doesn't mean my knee-jerk reaction is right. I guess in some ways, there is no way to know which reaction is the right one to have. Maybe they are all a little right and a little wrong.

In this exchange, we can see that the patient's rigid thinking begins to slowly loosen up. She will undoubtedly need to practice this many times (including rehearsals within SCD) before it can become more habitual. However, in this instance, by completing the exercise only once, she was able to reach the conclusion that just because she has a thought, it does not mean that the thought is accurate, and indeed that it is not possible to know which thoughts are accurate or inaccurate. As cognitive therapy progresses and the patient becomes increasingly skilled at generating multiple perspectives, the therapist can help the patient to choose useful perspectives among the various ones generated by assessing their relative accuracy, their advantages and disadvantages in the patient's life, and each perspective's impact on the patient's emotional functioning.

Therapists can further aid cognitive change by employing the Socratic method to challenge patients' automatic thoughts, as well as a host of other traditional cognitive therapy techniques. For example, when patients are catastrophizing about the outcome of a worry, repeatedly asking "What would be so bad about that?" can help them identify the worst potential outcome of their worry. Therapists can help patients come to the conclusion that even the realistic worst-case scenario is often better than what they had anticipated, and that they have the skills to effectively cope with negative outcomes. This can also be achieved through the use of worry outcome diaries, in which patients record four things: their worries; what the outcomes of those worries are; whether the actual outcomes, when they occur, were better, equal to, or worse than they had initially anticipated; and, in the case of worries that came true, how well they were able to cope with the outcome. As a result of this technique, many patients find that the majority of their worries do not come true, and when they do, patients are able to cope with the outcomes better than they expected (Borkovec, Hazlett-Stevens, & Diaz, 1999).

One complication that may arise during cognitive therapy for worry is the patient's espousal of positive beliefs related to worrying. Specifically, many patients report the beliefs that worrying actually makes it less likely that negative outcomes will occur, that worry motivates them to take action, and that worrying prepares them to cope with the worst-case scenario (Borkovec & Roemer, 1995). One cognitive technique that specifically targets such positive beliefs about worrying is the use of ABAB single-subject designs. Adapted from Barlow and Hersen (1984), patients employing ABAB designs are encouraged to engage in a task that typically engenders worrisome thinking while worrying and while not worrying and to rate their anxiety and performance throughout the course of the exercise. The goal is for patients to employ a scientific approach to evaluate whether worrying actually does have positive effects on their performance or affects whether good or bad outcomes eventually occur. When used in the Penn State GAD treatment program, patients

have routinely reported that they are less anxious during the tasks when not worrying and are able to perform the tasks as competently and efficiently (often more so) when they are not worrying compared to when they are worrying.

Another cognitive technique aimed at reducing positive beliefs about worry is to conduct a cost–benefit analysis of worrying. Two possible foci of cost–benefit analyses are to (a) help patients recognize the advantages of producing small reductions in negative affect resulting from worrying relative to the disadvantages of the distress caused by worrying about events that infrequently occur and to (b) help patients compare the likelihood of a worry actually coming true to the emotional energy spent on that worry. To illustrate this latter technique, consider, again, our patient who worries about her children’s safety after school:

- T: We’ve talked a lot about your levels of worry between 2:30 and 3:00 pm every day, and how distressing it can be for you. I imagine that half hour period every day is just torture for you.
- P: You said it – it’s pure torture. It’s like living through hell, imagining all the terrible things that might be happening to my kids. Honestly, you would think that if it’s so terrible a feeling, I’d be motivated to not worry so much!
- T: Do you ever think that worrying is having some positive effect?
- P: You know, it’s funny you should say that. I worry all the time, and nothing has happened to them so far. So sometimes I’m afraid that if I stop worrying, it’ll cause the bad stuff to finally happen. I’ve never really said that out loud before. Geez, that really sounds crazy.
- T: Actually, you’re not the first person to believe that worrying has some positive effects. I wonder, though, if we were to really examine the effect of worry – if we were to submit it to a bit of a cost-benefit analysis – how it would do.
- P: What do you mean?
- T: Well, let’s take your biggest worry – that on the way home, a car will hit your son, since you say he’s sometimes absent-minded when crossing the street. On a scale of 0–100, how likely do you think it is that a car will *actually* hit him?
- P: Probably about 2%.
- T: How likely do you think it is that he’ll be killed as a result?
- P: About 1%.
- T: OK, so we’ve got a 1–2% chance that your worst worry will come true, correct?
- P: That sounds about right.
- T: Now, if you had to estimate, again using our 0–100 scale, how upset you get for that half hour each day, what number would you give it?
- P: I’d say about a 90.
- T: So it sounds like you’re investing 90% of your emotional energy on something that is 1 or 2% likely to happen.
- P: Oh boy [laughing]. That’s not good. That’s pathetic.
- T: Well, let’s not pass too much judgment on it. Let’s just be unbiased observers of this assessment.

- P: Can I play devil's advocate?
- T: Of course. Please. . .
- P: Well, maybe the cost of my getting upset is, you know, much higher than the likelihood of Joey actually getting killed in an accident. But if he *did* get killed, well, I'd be upset at about a 300 out of 100. And if worrying helps to fend that off, then getting upset at a 90 is worth it!
- T: Ah, I see, so you're saying that you're more than willing to suffer at a level of 90 every day for 30 min if it decreases the likelihood of suffering at a 300 for the rest of your life.
- P: Yep.
- T: Well, I can certainly see where you're coming from. I wonder, though, if the picture looks the same when we consider how realistic it is that the worry is actually having a positive effect. Let me ask you this: How much do you really believe that your worry is keeping him safe?
- P: Hmm. Well, rationally, I know it's not. It's more of an emotional thing. That makes me think of all the times you've said that just because we feel something, doesn't make it true.
- T: Obviously, there are some *potential* pros and *definite* cons here to the worry. Can you step away from the emotionality of it for a moment and evaluate it, again, as an unbiased observer? What's the verdict on this worry?
- P: I think I know what you mean. Chances are, Joey will live to see adulthood whether I worry or not. Meanwhile, I'll have been a mess for all those years, all for no good reason. Because I bet if I magically stopped worrying tomorrow, he'd be no worse off every day on the way home from school. I'll probably never see that 300, and meanwhile I'm giving myself a daily dose of 90.

In this exchange, one can see how strongly the patient holds on to her positive beliefs about worrying. She is willing to suffer each day for the sake of avoiding much more intense negative emotions if her worst fear were to come true. However, when the therapist asks her to conduct an assessment of how effective she thinks her worry is in having positive effects, she comes to realize that the combination of (a) daily suffering and (b) low likelihood of a pay-off resulting from that suffering simply does not tip the scales in the direction of benefit over cost.

## Interpersonal and Emotional Processing

Given the limitations of CBT in achieving high end-state functioning among a large number of patients, clinical scientists have utilized knowledge gained from research investigations to develop adjunct treatments for GAD that may increase the number of individuals who benefit from therapy. One such finding is that chronic worriers who present with troubled relationships, personality pathology, and limited social support systems may not benefit as much from therapy, suggesting that attending to

interpersonal problems may be a fruitful path to helping chronic worriers. Although no specific studies on emotional deficits as predictors of treatment response exist, extant research suggests that targeting the emotional lives of individuals with GAD may be fruitful. For instance, alexithymia studies in GAD suggest that worriers have trouble identifying and describing their emotions (Yamas, Hazlett-Stevens, & Borkovec, 1997), and research on GAD patients' reactions to emotions suggests that they are fearful of emotional experiences (Mennin, Heimberg, Turk, & Fresco, 2005) and find emotions to be aversive (Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). As mentioned earlier, the avoidance theory of GAD posits that worry is remote from somatic and emotional experience, and that worry's inhibitory quality precludes emotional processing of fear cues (Borkovec et al., 2004).

In interpersonal and emotional processing therapy (IEP; Newman, Castonguay, Borkovec, & Molnar, 2004), therapists help patients improve their functioning in two major areas, namely interpersonal relationships and the depth with which they experience authentic, primary emotion. In interpersonal therapy for GAD, therapists and patients together conduct a functional analysis of interpersonal behavior, paying close attention to behaviors that increase or decrease the likelihood of fulfilling interpersonal needs and of maximizing the quality of their relationships with significant others. The therapeutic relationship is also used as an important sample of patients' behaviors in their everyday lives, and the therapist provides valuable, constructive feedback on how a patient's behavior impacts the therapist in the moment. Through this feedback, patients learn about natural contingencies and have the opportunity to practice new skills within a safe context in which the significant other (here, the therapist) provides genuine feedback and positive regard. Patients then practice these new behaviors in their actual relationships and conduct assessments of which behaviors are successful at helping them attain their goals and satisfy their interpersonal needs.

In emotional processing therapy for GAD, therapists help patients to undergo repeated exposures to emotional experiences. The goal is to help patients to become increasingly comfortable with both positive and negative affect, which helps them both to experience and express more authentic emotions in their lives and to identify underlying interpersonal needs which facilitate the development of effective interpersonal behaviors. Therapists may employ several techniques from experiential therapy to help patients to deepen their emotional experiences. For example, in the empty-chair technique, the patient has the opportunity to communicate with a significant other (whom he/she can imagine sitting in the "empty chair") regarding an issue that may elicit strong emotions in the patient.

Researchers at Penn State recently completed an investigation examining the effects of adding IEP therapy to CBT (CBT+IEP), as compared to CBT plus supportive listening (CBT+SL, where SL was used to control for various common factors related to therapy). Preliminary analyses indicate that the addition of IEP to CBT does not enhance efficacy of treatment as measured by most of the primary outcome measures, although 24 months after treatment termination this combined condition did evidence a significantly higher rate of high end-state functioning.

Although these results prevent us from routinely recommending the use of IEP therapy with all GAD patients, these techniques may be useful for individual patients if functional analysis of those patients' behaviors indicates that these areas of functioning are maladaptive, contribute to the patient's anxiety, and are appropriate targets of intervention. Within this clinical trial, for example, it was found that patients who had highly dismissive attachment styles assessed at pre-therapy and received the IEP component had significantly better post-therapy and follow-up outcome than all other patients, whereas those who did not have enmeshed relationships with their primary care-giver in childhood did particularly well when IEP was part of their treatment (Newman, Castonguay, Fisher, & Borkovec, 2008).

A further example is given below in the case of our patient, Adele, who is discussing with her therapist a common interpersonal pattern that the therapist has noticed:

T: Adele, with your permission, I'd like to take a few moments to make an observation about our interactions. It's something that I've noticed over the course of our meetings, and I'd like to bring it out in the open so that we can discuss it. Sometimes when we're talking about emotional issues in here – whether it's about your relationship with your mom, or your marriage, or even about how you feel about your sister's death – it sometimes almost feels like you're not completely "here."

P: I don't understand. You've asked me to talk about those things, and I have.

T: Yes, you definitely have, and I know it's been hard for you to discuss some of those tough issues. Maybe I'm not being very clear about what I mean. . .let me try harder, with a more concrete example. Last time, about halfway through our session, I thought there was a moment when we had a "disconnect" of sorts. The way I remember it – and please speak up if you think I'm not doing it justice, or misremembering it – you were talking about your sister's death. At some point, you sort of shifted to a different topic. . .I think we somehow started talking about the political elections. . .and I tried to redirect the conversation back to talking about your sister. I couldn't tell exactly what had happened, but it felt from my end that you had become frustrated with me. And, thinking about it later, I realized that in twelve sessions we've always briefly mentioned your sister's death, but we've never really discussed it in detail. The truth is, this is an enormous part of your emotional life, and I don't know much about it. The topic seems to be a slippery one, and we seem to often lose our hold on it.

P: Well, you know, talking about her is really hard.

T: I can only begin to imagine just how hard it is. Did you feel that I was being too pushy last time?

P: It's not just that. I mean. . .sometimes I think about her, and I just. . .I miss her a lot. Talking about her is hard, because it makes me remember her and miss her. She was my closest friend, and it sometimes feels like I can't find

anyone who understands me the way she did. [Patient getting choked up, with tears in her eyes.]

- T: You know, there is something about sibling relationships that's really special. Siblings, in many ways, are our first friends.
- P: Yeah. You know, I look at my kids sometimes and watch them playing or fighting or negotiating something, and it's kind of funny to see them like that. [Patient brightens up a bit.] The other day, Joey and Hannah were watching cartoons, and I could hear them laughing from the other room. They've gotten really into this new cartoon on TV, and they wait all day until 5 pm to watch it. My husband had the brilliant idea of using it as a reward or punishment, and I don't think he's had this much power over them in ages!
- T: Adele, I'd like to bring us back to chatting about your sister. I can't help but feel again like we've slipped away from that topic. I know it's hard to focus on, but I just can't help but notice that when we go there, you seem to run away.
- P: [Pause. . .] I'm afraid to think about her. I'm afraid it'll be just like the year after her death, when she was all I could focus on.
- T: I can understand that. It must be a scary feeling. I can tell that you loved your sister a lot.
- P: [Crying openly now.]
- T: Tell me what you're feeling right now.
- P: So many things. Love, sadness, longing, anxiety, guilt. I'm thinking about my wedding, when she went out of her way to make everything so special for me. I'm thinking about the birth of my first child, when she held my hand in the hospital room as I pushed. I'm thinking about all the times she got on a train to come see me when I needed her, and all the times I went down to Virginia when she needed me. I'm thinking about our coffee dates, and the time we went to see the Phantom of the Opera and she got sick in the theater and how funny we thought it was later [laughing, crying]. I wish she could be here now, so that I could show her how similar my daughter is to her. I miss her so much. . . .
- T: [silent]
- P: Sometimes I just can't believe she's gone. Sometimes I wish it could have been me instead of her. But other times, I'm relieved it wasn't me, because I have kids and she didn't. But I feel so guilty that I even think that. I can't even believe I just said it. I hate myself for thinking it.
- T: Adele, let's try to stay with that for a moment. Tell me more about that feeling of guilt. I know it's hard, but please try.
- P: You know, I wasn't even there for her when she died. I could have gotten there sooner, but I was just so wrapped up in my own life. Meanwhile, my little sister was dying. And now that she's dead, I feel relief that it wasn't me! Relief! It's a disgusting thing for me to feel. I just feel so. . .dirty when I think that. How can I think that, when she deserved to live? I feel so guilty. I'm awful.

- T: It sounds like you have two big emotions here. On the one hand, you love your sister and miss her dearly. On the other hand, you feel guilt over your relief that it wasn't you.
- P: I feel so overwhelmed by all of this. I miss her. I miss her so much. I wish I could see her right now, but I still feel so guilty. [Crying harder for a couple of minutes.]
- T: You know, Adele, even though this has been really hard for you to focus on, I think this is the most genuine I've ever seen you be in here. You feel much more real to me right now.
- P: I spend so much time avoiding feeling like this that I sometimes forget how much I miss her.

In the above exchange, the therapist began by discussing an interpersonal issue, namely the loss of a loved one and the possibility that the therapist had caused a rupture in the therapeutic alliance by trying to have the patient talk about this highly emotional event. The discussion elicited an emotional reaction from the patient, who quickly became avoidant of that path of discussion and any emotional engagement. Notice how the therapist gently redirects her, while pointing out the pattern she notices and encouraging the patient to label her emotions and stay with the emotional experience. The therapist also points out the patient's apparent conflicting emotions, and reinforces the patient's genuine expressions of emotion. Notice also how the therapist relates this genuine emotional moment to a real interpersonal issue between the two of them, namely that the patient's emotionality and openness has made their relationship itself more genuine ("You feel much more real to me right now"). For a detailed description of emotional processing techniques in the treatment of GAD, the reader is referred to Newman et al. (2004).

## Future Directions

There remains important work to be done in the quest to develop effective treatments for GAD that benefit larger numbers of patients. Recently, several investigators have drawn on existing GAD research to suggest specific strategic approaches to treating chronic worry. First, individuals with GAD find uncertain or ambiguous situations to be less tolerable relative to individuals without GAD (Dugas, Gagnon, Ladouceur, & Freeston, 1998). Indeed, "intolerance of uncertainty" reliably distinguishes individuals with GAD from non-anxious individuals (Ladouceur, Blais, Freeston, & Dugas, 1998). Dugas and his colleagues have developed an intervention to specifically target intolerance of uncertainty. Thus far, evaluations of this intervention in both an individual (Ladouceur et al., 2000) and group (Dugas et al., 2003) therapy format suggest that this treatment evidences significant improvements over a waiting-list condition. Future evaluations employing comparisons against active control groups will help to further inform the potential value of targeting this area of functioning in the lives of individuals with GAD.

Second, the metacognitive model of GAD (Wells, 1995, 1999, 2004) proposes that when individuals engage in worry, negative beliefs about that worry are engendered, leading them to engage in worry about the worry process (or “meta-worry”). This meta-worry is hypothesized to produce a number of ineffective coping strategies aimed at reducing worry, such as reassurance seeking, checking behaviors, thought suppression, distraction, and avoidance of potential worry-inducing situations (Wells, 1999, 2004). Metacognitive therapy for GAD focuses on the use of behavioral experiments and examination of the evidence to modify beliefs that worry is uncontrollable, that it is dangerous, and that it has positive consequences. In an open trial investigating the effects of metacognitive therapy for GAD, 10 patients showed significant improvements at post-treatment on measures of worry, anxiety, and depression (Wells & King, 2006). As pointed out by the authors, controlled investigations of metacognitive therapy are needed to evaluate its potential unique contribution to the treatment of GAD.

Third, the emotion dysregulation model of GAD (Mennin, Turk, Heimberg, & Carmin, 2004) posits that individuals with GAD (a) experience emotions more intensely than do others (Turk et al., 2005); (b) have a poorer understanding of emotions than do others; (c) show greater negative reactions to emotions relative to most people; and (d) use ineffective strategies to regulate their emotions, thereby leaving them in even worse emotional states. A therapeutic intervention based on the emotion dysregulation model is currently being developed by Mennin (2004), and combines elements of traditional CBT for GAD with treatment components specifically targeted to address problems with deficits in emotion regulation. A focus on emotion dysregulation in GAD contains hope for increasing the efficacy of traditional CBT, and future controlled outcome investigations will surely provide valuable information on the nature of the emotional lives of individuals with GAD.

Finally, Roemer and Orsillo (2002, 2005; Roemer et al. 2006) have drawn upon Hayes’ work on experiential avoidance (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996) to expand the theoretical framework in which GAD is conceptualized. Roemer and Orsillo reiterate the avoidance theory’s view of worry as cognitive avoidance of physiological and emotional experience, and further argue that individuals with GAD fear losing control of their emotions and that they are less mindful of internal and external events relative to individuals without GAD. They also stress the importance of attending to patients’ core values in treatment as a way of enhancing intrinsic motivation and intentional action. Thus, their treatment for GAD incorporates traditional CBT approaches combined with efforts to reduce fear of emotions and experiential avoidance through the use of mindfulness- and values-based therapy (Roemer & Orsillo, 2005). In a recent open trial of this treatment, 16 patients evidenced significant improvement in GAD severity and self-reported symptoms of anxiety, depression, and fear/avoidance of internal experiences (Roemer & Orsillo, 2007). Future controlled investigations of mindfulness-based therapy for GAD hold promise for informing the nature of worry and approaches to its treatment.

Finally, additional research aimed at identifying predictors of treatment responsiveness will be crucial to future efforts to devise techniques that can make GAD

treatment successful for a greater number of patients. For example, research on specific emotion-related variables may elucidate whether emotional processing therapy techniques may be beneficial for specific types of patients (e.g., those who display deficits in expressions of negative or positive affect, or those who display deficits in processing highly arousing emotions). As researchers identify specific individual factors that make patients more or less responsive to therapy, treatment packages can eventually help larger numbers of chronic worriers.

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# Avoiding Treatment Failures in Specific Phobias

Georg W. Alpers

## Treatment Complications in Specific Phobias

When systematic research on phobias began, fears related to specific objects or situations were still called “simple phobias” (see Marks, 1969), a term that misleadingly implied that these phobias were of low severity. In fact, specific phobias can incur serious life impairment, in the range of other mental disorders (Becker et al., 2007). Since the introduction of DSM-IV (American Psychiatric Association, 1994), they are listed by the preferable term specific phobias (see Hofmann, Alpers, & Pauli, 2009).

Specific phobia is characterized by intense and persistent fear cued by exposure to, or anticipation of, a clearly discernible and circumscribed object or situation such as certain animals or insects, blood/injury/injection (BII), natural environmental events (e.g., thunder), or other stimuli (e.g., vomiting, contracting an illness). Although adults with phobias realize that these fears are irrational, they avoid confrontation in order to avoid triggering panic or severe anxiety. If the fear is confronted, it is common to experience emotional discomfort and marked autonomic, respiratory, and endocrine reactivity (Alpers, Abelson, Wilhelm, & Roth, 2003; Alpers & Sell, 2008; Alpers, Wilhelm, & Roth, 2005). Some symptoms are specific to certain fears, such as body sway, which is closely associated with fear of heights (Alpers & Adolph, 2008; Hübeler, Kandil, Alpers, & Gerlach, 2009).

The *DSM-IV* (APA, 1994) lists five discrete subtypes of specific phobias: (1) animal type: if the fear is cued by animals or insects; (2) natural environment type: if the fear is cued by objects in the natural environment, such as storms, heights, or water; (3) BII type: if the fear is cued by seeing blood or an injury or by receiving an injection or other invasive medical procedure; (4) situational type: if the fear is cued by a specific situation such as public transportation, tunnels, bridges, elevators,

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This work was supported by a grant to Georg W. Alpers from the German Ministry of Research and Education (BMBF FKZ 01GV0617).

flying, driving, or enclosed places; and (5) other types: if the fear is cued by other stimuli (including choking, vomiting, or contracting an illness, fear of falling, and children's fears of loud sounds or costumed characters).

Previous studies estimated the lifetime prevalence of specific phobias to be between 1% and 19% of the population. More recent data suggest that the lifetime and 12-month prevalence rates of specific phobia are about 13% and 9%, respectively (Becker et al., 2007; Kessler et al., 2005; Kessler, Chiu, Demler, & Walters, 2005; Stinson et al., 2007). These data suggest that specific phobia is the most common form of anxiety disorder. Prevalence rates are generally higher in women than in men (ratios vary between 2:1 and 4:1 from study to study) with much variance between subtypes of phobias (Becker et al., 2007; Bourdon, Boyd, Rae, & Burns, 1988; Curtis, Magee, Eaton, Wittchen, & Kessler, 1998; Fredrikson, Annas, Fischer, & Wik, 1996; Stinson et al., 2007). The highest prevalence rates have been found for specific animal phobias in women and claustrophobia in men (Curtis et al., 1998). Prevalence rates also differ with ethnicity; the rates of specific phobias are almost twice as high in African-American individuals (Eaton, Dryman, & Weissman, 1991; Stinson et al., 2007). Considerable variation in prevalence rates can be found between cultures worldwide (Good & Kleinman, 1985; Shen et al., 2006).

Impairment has been found to strongly correlate with the number of phobic symptoms a patient experiences (Curtis et al., 1998). In addition to the high prevalence of multiple phobias, comorbidity with other mental disorders is high: 84% of all phobic patients have one or more comorbid disorders. Aside from other anxiety disorders, affective disorders and substance-related disorders are common (Kushner, Krueger, Frye, & Peterson, 2008). The phobia more often preceded (in 57% of the cases) the comorbid disorder (Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996). In spite of the high prevalence and considerable impairment, only 8% reported treatment specifically for specific phobia (Stinson et al., 2007).

### *The Origin of Specific Phobias*

Rachman (1977) suggested that there are three routes to develop a phobia: First, by classical conditioning due to a traumatic experience in a specific situation or in the presence of a specific object; second, by vicarious learning; and third, by instruction (usually the parents) or information (e.g., the media). Empirical data suggest that specific phobia develops following a traumatic experience in 36% of the cases, the observation of fearful behavior or observation of a trauma to others in 8%, and the instruction by others in 8%. This means that about 50% of the phobic patients do not recall how or why they developed the phobia (Kendler, Myers, & Prescott, 2002).

A number of longitudinal studies clearly demonstrate the link between traumatic experiences, such as traffic accidents or episodes of unexpected panic, and the later development of a specific phobia (Blaszczynski et al., 1998). Although these data show that some phobias date back to aversive experiences, this does not explain why certain classes of typical cues (e.g., spiders and snakes) often elicit phobic reactions

despite rarely inflicting harm, while others often inflict harm (e.g., knives) but are rarely feared. This seeming paradox can be explained if one assumes that the typical phobic cues are prepared fear stimuli (Seligman, 1971). Indeed, several experiments consistently confirmed that fear responses conditioned to typical fear cues are more resistant to extinction (Mineka & Öhman, 2002). Also, vicarious learning of fear responses that has been documented for laboratory-reared monkeys who learned to avoid snakes from a model (Mineka, Davidson, Cook, & Keir, 1984) seems to be preparedness for evolutionary relevant fear cues (Cook & Mineka, 1989).

Based on the resistance to extinction documented for experimentally acquired responses, the preparedness hypothesis has been widely accepted and entered almost every textbook on biological and on abnormal psychology, although evidence for other characteristics of preparedness (ease of acquisition, irrationality, and belongingness) is much more limited (McNally, 1987). The basic premise of the theory that preparedness helps to protect humans from dangerous predators has recently been questioned for a common specific phobia, i.e., spider phobia (Gerdes, Uhl, & Alpers, 2009).

In humans learning avoidance behavior from models (Gerull & Rapee, 2002) and fear acquisition by instruction (Field & Lawson, 2003) seem to be particularly important routes to the development of anxiety. Moreover, it has been shown in longitudinal studies that experience with certain challenges such as heights or water is needed to unlearn certain inborn fears (Poulton & Menzies, 2002).

Theoretically, the characteristic avoidance behavior phobic patients display can be explained by the two-factor model (Mowrer, 1947). This model assumes that classically conditioned fear stimuli elicit a fear response that is then reduced or ameliorated by the instrumental avoidance behavior. The conclusion that this dysfunctional avoidance helps to maintain the fear response can be extended to fears acquired through other routes than classical conditioning. Aside from these learning-based considerations of how phobias may be acquired, there is strong evidence for a significant genetic disposition to acquire an anxiety disorder (Kendler, Neale, Kessler, Heath, & Eaves, 1992).

### ***Differential Diagnosis and Comorbidity***

A challenge to adequate differential diagnosis is the fact that anxiety disorders are highly comorbid (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Stinson et al., 2007). However, structured interviews such as the Anxiety Disorders Interview Schedule for DSM-IV and adequate training in its application lead to good diagnostic accuracy and interrater reliability for specific phobias (Brown, Di Nardo, Lehman, & Campbell, 2001). Although most subtypes of phobic disorder can also be differentiated reliably (Fyer et al., 1989), a major difficulty is the diagnostic distinction of phobias and panic disorder. With the exception of phobias of the animal type, panic disorder patients – especially those with marked agoraphobia – often fear typical phobic cues such as heights, invasive medical procedures, public transportation, or contracting illness. Moreover, patients with specific phobias

often experience panic attacks with marked physical symptoms during exposure to the feared cues, especially in the situational subtype (Ehlers, Hofmann, Herda, & Roth, 1994; Lipsitz, Barlow, Mannuzza, Hofmann, & Fyer, 2002). Symptoms of these situational panic attacks markedly overlap with typical symptoms experienced by panic disorder patients (Craske, 1991); for example, the symptom profile of the phobic fear of enclosed places is most similar to that of panic disorder and the fear of enclosed places is frequent in panic disorder patients. A further complication arises because the phobias trace back to a spontaneous panic attack in many patients (Himle, Crystal, Curtis, & Fluent, 1991). In spite of these similarities, phobic fear and panic disorder are clearly distinguished by most theoretical accounts because they are either uncued (i.e., as seen in panic disorder) or cued (i.e., as seen in specific phobia) (Barlow, 2002).

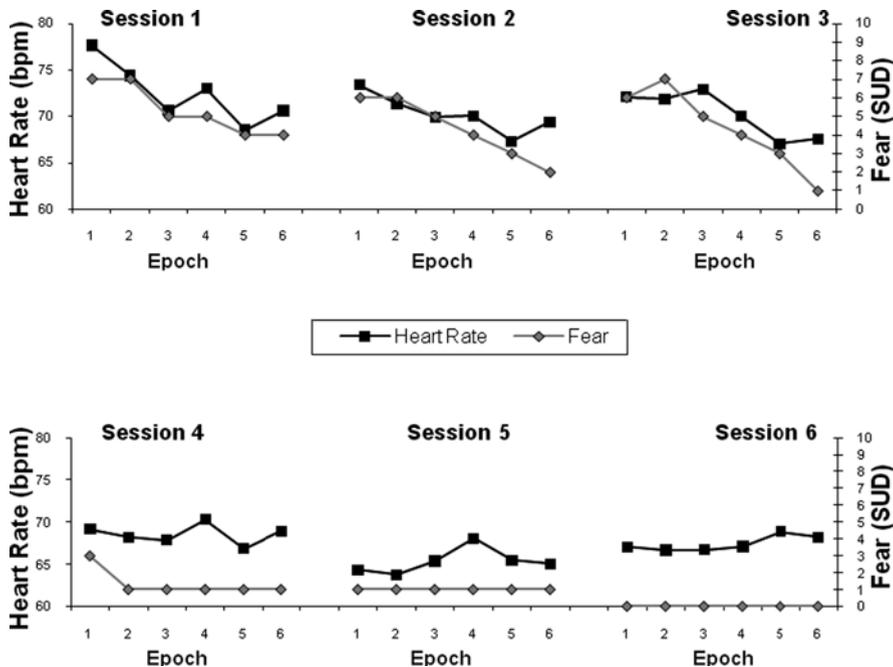
### ***Gold Standard for Therapy: Exposure***

A systematic review of treatment studies published between 1960 and 2005 shows that research has been conducted on the usefulness of systematic desensitization (or imaginal exposure), in vivo exposure, cognitive therapy, for a multitude of different specific phobias and applied tension for BII phobia specifically (Choy, Fyer, & Lipsitz, 2007). The limited data on medication are disappointing (Choy et al., 2007).

Although some textbooks still recommend systematic desensitization (Wolpe, 1962) as the typical strategy for the treatment of phobias, today, the gold standard for the treatment of specific phobias clearly is exposure therapy unaccompanied by relaxation instruction. Response to in vivo exposure has proven to be superior to systematic desensitization in several direct comparisons (Choy et al., 2007). The effects of exposure are generally not limited to behavioral changes but extend to improvement in cognitive measures of fear as well (Booth & Rachman, 1992). Figure 1 illustrates extinction of self-reported fear and heart rate within and across repeated exposure sessions in a claustrophobic patient.

### ***Efficacy of Exposure***

A recent meta-analysis systematically compared the efficacy of different strategies used to treat specific phobias (Wolitzky-Taylor, Horowitz, Powers, & Telch, 2008). Data from 33 randomized treatment studies were compared. First, exposure-based treatment approaches produced large effect sizes relative to no treatment (wait list). Second, exposure also outperformed placebo conditions and alternative active psychotherapeutic approaches (e.g., relaxation training). Third, in vivo exposure with the phobic cue also outperformed alternative forms of exposure therapy (e.g., imaginal exposure, virtual reality) at post-treatment. There are now several well-established manuals providing self-help materials as well as support and information for the clinician (e.g., Barlow & Craske, 1994; Bourne, 1998, 2005). In vivo exposure is often conducted in a graded fashion, beginning with a relatively mild challenge and progressively moving upward on the hierarchy of fearful cues.



**Fig. 1** Self-reported fear (subjective units of distress, SUD) and heart rate (HR) in one selected patient in the course of the six exposure sessions. The patient was 52 years of age. His HR at quiet sitting after session 6 was 64 bpm. His scores on a claustrophobia questionnaire (CLQ, Radomsky, Rachman, Thordarson, McIsaac, & Teachman, 2001) changed from 57 before to 28 after the sixth session (with permission from Alpers & Sell, 2008)

A review of studies using in vivo exposure therapy reported that between 80% and 90% of treatment completers were able to complete the behavioral avoidance task, a measure of clinically significant change, at the end of treatment (Choy et al., 2007). The difference between treatments was sometimes as large as 25% of completers being able to touch a snake after systematic desensitization versus 92% after in vivo exposure (Bandura, Blanchard, & Ritter, 1969). Importantly, there has been no evidence for symptom substitution. Instead, the gains obviously generalized to other domains, even to those not addressed during exposure treatment (Götestam & Götestam, 1998).

Follow-up assessments of in vivo exposure were gathered for several specific phobias with periods ranging from 6 months to 14 months. In general, treatment gains are either maintained or further improved over time (Choy et al., 2007). However, superiority of in vivo exposure to alternative approaches was less pronounced in some studies at follow-up than at the post-treatment assessment following exposure therapy (Wolitzky-Taylor et al., 2008). For both findings (the increasing effects and the decline of treatment-specific differences) a possible explanation may be that even moderate therapeutic improvement may motivate all

patients to engage in self-guided in vivo exposure so that they can improve further. On the other hand, some patients who were successfully treated with in vivo exposure may cease to self-expose after treatment has terminated and may return to some of their avoidance behavior. In the clinical setting, this needs to be assessed in booster sessions on an individual basis. The few studies that examined very long-term outcomes call for caution. Lipsitz and colleagues reassessed symptoms 10–16 years after treatment and found considerable rates of relapse (Lipsitz, Mannuzza, Klein, Ross, & Fyer, 1999).

Interestingly, very brief exposure treatments have garnered substantial support. For example, Öst and coworkers have demonstrated that prolonged exposure can be successful even if there is only one extended session (usually between 2 h and 4 h) with a therapist (Öst, 1989). The effectiveness has been demonstrated most clearly for small animal phobias (Gotestam, 2002; Hellstrom & Oest, 1995; Koch, Spates, & Himle, 2004; Thorpe & Salkovskis, 1997) and flying phobia (Öst, Brandberg, & Alm, 1997). However, in the meta-analysis mentioned before (Wolitzky-Taylor et al., 2008), multi-session treatments marginally outperformed single-session treatments on domain-specific questionnaire measures of phobic dysfunction, and moderator analyses revealed that more sessions predicted more favorable outcomes.

Although it is often assumed that some phobias may be more difficult to treat than others, effect sizes for the major comparisons of interest were not moderated by the type of specific phobia in the meta-analysis (Wolitzky-Taylor et al., 2008). Interestingly, the meta-analysis also revealed that placebo treatments were significantly more effective than no treatment, which suggests that patients with specific phobias are moderately responsive to placebo interventions.

Although beyond the scope of this chapter, it should be mentioned that phobias are common in children as well. Although their characteristics often differ from those in adults, there is some overlap. Because phobias start early in life, and pose a risk for developing a second mental disorder, and because of the chronic duration, the need for treatment in childhood and adolescence is obvious (Becker et al., 2007). Exposure in vivo is also the treatment of choice for small animal phobias in children (Muris, Merckelbach, Holdrinet, & Sijnsenaar, 1998).

## **Core Elements of CBT**

### ***Cognitive Preparation or Psychoeducation***

Most treatment approaches for specific phobias include some form of psychoeducation, but the core CBT strategy to overcome phobic fear is unanimously some variant of exposure and response prevention. In this context, psychoeducation has the purpose of introducing the therapeutic rationale, and typically addresses the nature of (non-clinical) fear and its protective function. It usually also involves a review of the individual's risk assessment in a given situation and some cognitive restructuring if this assessment seems to be exaggerated. Then, the principles of

fear acquisition and extinction are reviewed, with the purpose of raising motivation and clarifying expectations for the exposure therapy to follow. Together, these aspects of psychoeducation are sometimes summarized as cognitive preparation. Significant predictors of treatment success are credibility of the treatment rationale and the motivation for psychotherapy in general (e.g., Öst, Stridh, & Wolf, 1998; Southworth & Kirsch, 1988).

### ***Exposure to Fear Cues***

In the preparation for exposure therapy, patients are asked to identify and arrange cues that evoke undue fear from the least to the most frightening. Starting at a cue representing moderate fear, patients are asked to gradually expose themselves to the cue, usually for up to an hour or more at a time, allowing ensuing feelings and thoughts to occur without escape, and to continue the exposure until these feelings of discomfort subside. Such homework is frequently assigned on a daily basis. Exposure may be conducted with or without a therapist (e.g., Öst et al., 1998) and/or guided by appropriate self-help books or computer systems (e.g., Kenwright, Marks, Gega, & Mataix-Cols, 2004). Phobic cues are frequently live situations or objects, but when difficult to arrange, pictures, film material, or computer animations (see Virtual Reality) or imaginal stimuli may be used. A number of studies now suggest that virtual reality may be effective in flying and height phobia, but this needs to be substantiated by more controlled trials (Choy et al., 2007; Krijn, Emmelkamp, Olafsson, & Biemond, 2004; Wolitzky-Taylor et al., 2008).

### ***A Case Example: Treating Spider Phobia***

Jen, aged 35, consulted a therapist for her severely handicapping and inexplicable fear of spiders. She had never really liked spiders and her fear had intensified over the years. Whenever she saw a spider she panicked helplessly and felt frozen in place. Her heart raced, her palms sweated, and she felt embarrassed because she depended on other people because of this fear. She avoided walking across a lawn or going into her basement or garage lest she encountered spiders there. Having unsuccessfully tried to prevent spiders entering her home, she was about to move elsewhere. Jen was told her symptoms were typical of a phobia and that she could endure them for long enough to get used to whatever was frightening her. Even the mere thought of looking at a spider evoked extreme fear and disgust. In therapy, she learned to open a book with pictures of spiders in the therapist's office. She took the book home and made herself touch the pictures with her fingers. Next, she looked at a spider in an empty glass jar for at least 30 min without her usual attempt to remove it or turn away from it. Jen was encouraged to do exposure without her usual subtle avoidances that stopped her experiencing the fear fully and getting used to it. Thus she examined the spider and her own reactions in detail, she was fascinated at not

being overwhelmed by fear. Her distress decreased during each exposure session and across repeated such sessions. She became more confident exposing herself to spiders at home. After 10–50 min weekly sessions and several hours of practice at home she touched a large spider and let it crawl across her palm. When she had accomplished this, she expressed doubt that her family would actually believe what she had just done, and the therapist spontaneously decided to take a few pictures of how she handled the spider. She was proud and happy to take home her therapy graduation pictures. Jen then cleaned out her garage, kept a spider in a jar in her kitchen, and went to bed without checking for spiders. Improvement continued at follow-up 8 weeks later.

## **Factors that May Interfere with Exposure Success**

### ***Treatment Engagement***

As in CBT for panic disorder with or without agoraphobia (see Sanderson & Bruce, 2007), lack of engagement in behavioral exposure or non-compliance is the most important reason for sub-optimal treatment response in phobia therapy as well. For example, compliance with self-exposure homework during weeks 0–8 predicted more improvement 2 years later (Park et al., 2001). An older and relatively small study suggests that positive interaction (“warm therapist behavior”) contributes to better adherence (Morris & Magrath, 1979). Even in completed treatments, negative cognitions have been identified as a predictor of poor treatment response (Rachman & Levitt, 1988; Shafran, Booth, & Rachman, 1993). Accordingly, cognitive interventions should be considered to address possible negative expectations, as a strategy to reduce drop-out and to improve outcome. It is important to point out that patients who fail to improve with one treatment, for example, relaxation training, retain a strong chance of responding after subsequent crossover to exposure therapy (Park et al., 2001).

### ***Duration of Exposure***

Generally, clinicians strongly emphasize that exposure sessions need to be of adequate duration to result in a reduction of fear across sessions. Exposure appears to be more effective as the patient is given more time to experience the reduction of anxiety in the presence of the phobic cue. For example, exposure was found to be more effective when no anxiety was experienced for at least 1 min compared to a condition where exposure was ended when the highest level of anxiety was reached (Marshall, 1985). Likewise, longer exposure results in more fear extinction than shorter exposures even if the total duration of exposure is held constant (Stern & Marks, 1973; but see de Silva & Rachman, 1984). Several studies with fixed durations (e.g., 30 min) of exposure have also been found to be effective independent of fear levels (e.g., Alpers et al., 2005); the most crucial aspect of these repeated brief exposures may be that patients are taught that they will have to return to exposure

soon after the first exposure has terminated, even if it was terminated at a relatively high level of anxiety.

### ***Multiple Phobias***

When more than one situation or object is feared, which is quite typical (Hofmann, Lehman, & Barlow, 1997; Stinson et al., 2007), patient and therapist need to decide which fear to target first. If too many issues are targeted at the same time it may be difficult for the patient to monitor gradual change in the course of the intervention. As such, one recommendation is that extinction should be clearly observable in the course of exposure exercises with one situation before targeting the next task.

Often, there are several distinct facets to one specific phobia. For example, in some cases with driving phobia it makes a big difference whether the patient is the driver or a passenger (Alpers et al., 2005; Ehlers et al., 1994). Exposure exercises will have to target these specific circumstances; the patient will have to be the driver or the passenger. Similarly, if patients with such a specific phobia experience panic attacks while driving, they may be very concerned that these attacks might impair their ability to drive. Here, cognitive restructuring will have to address these risk estimations (see below). On top of this, the car is a confined space that cannot be left at any time, that is, claustrophobia may make things worse. As indicated above, this aspect can be targeted separately. While driving, the driver might be exposed to unpleasant temperatures or lighting conditions or dizziness – intolerance of unpleasant bodily experiences, i.e., anxiety sensitivity may contribute to symptom development. Specific interventions targeting interoceptive exposure (Antony, Ledley, Liss, & Swinson, 2006) may be indicated to treat fear of bodily sensations. Also, while driving one is exposed to being far away from home when driving long distances – a typical agoraphobic challenge that can be the topic of a homework exercise even when the patient is not prepared to drive yet. When there are different facets to a specific phobia, the different circumstances and conditions need to be thoroughly explored and evaluated. Any case formulation will have to take these patient-specific factors into account.

### ***When Other Unpleasant Emotions/Sensations Come into Play***

*Disgust.* Although the characteristic emotional experience in phobias is fear upon exposure to the phobic cue, some cues may also elicit the distinct emotion *disgust* (Davey, 1994; Woody, McLean, & Klassen, 2005), which seems to change with therapy on different gradients (Smits, Telch, & Randall, 2002). Disgust also decreases during exposure treatment. However, in one study, the decline of disgust ratings was found to lag behind that of fear ratings in a direct comparison (Smits et al., 2002). Interestingly, disgust levels at pretreatment did not moderate the level of fear activation or fear reduction during treatment in this study. Although an experimental

analysis seems to indicate that disgust does not have a strong influence on the return of fear (Edwards & Salkovskis, 2006), the observation that not all emotions extinguish at the same rate may be relevant for treatment. The goal of exposure may not only be to reduce fear but also to reduce the level of other unpleasant emotions.

*Nausea.* Nausea is frequently related to anxiety disorders (Haug, Mykletun, & Dahl, 2002). More specifically, the fear of vomiting can make it difficult for a patient to expose herself to fear-provoking cues. Research with patients who are specifically afraid of vomiting (i.e., emetophobia) suggests that this fear may be closely related to the fear of losing control, and that vomiting phobia reflects this underlying problem (Davidson, Boyle, & Lauchlan, 2008). Such a fear of losing control should be explored and targeted by specific cognitive interventions which are aimed at increasing the tolerance of uncertainty (see Keefer et al., 2005; Robichaud & Dugas, 2006). There is evidence from a few case reports that also other worries about gastrointestinal problems, such as being afraid of having diarrhea (Hedberg, 1973) or excessive need to urinate (Myers, MacKinnon, & Corson, 1982), have been successfully treated with behavioral interventions. Physical therapy with vestibular rehabilitation exercises may benefit phobic patients with vestibular dysfunctions (Jacob, Whitney, Detweiler-Shostak, & Furman, 2001).

*Lightheadedness/Fainting.* Related to disgust and nausea is the fear of BII. Patients with BII phobia often faint when exposed to the relevant cues (Dahlloef & Öst, 1998; Sarlo, Buodo, Munafò, Stegagno, & Palomba, 2008). This particular psychophysiological pattern has been addressed in a specific intervention, applied tension (Öst, Fellenius, & Sterner, 1991; Öst, Lindahl, Sterner, & Jerremalm, 1984; Öst & Sterner, 1987).

*Shame.* Patients with a specific phobia can feel utterly debilitated as a consequence of their fear. They are dependent on family members and friends. In many cases they have to tailor their job search around conditions allowing them to avoid whatever they are afraid of. As in cases of social anxiety disorder, the focus of maladaptive cognitions can be placed on the consequence of public scrutiny and subsequent negative evaluation (“I’m going to make a fool of myself”). Some treatment strategies can also result in embarrassment (e.g., riding an elevator up and down for extended periods of time or staying in small fitting rooms while other people rush through the department store). This needs to be addressed when exposure is planned or it may result in undue insecurity or early cessation of prolonged exposure. Importantly, the patient needs to be aware of the different emotions aroused in a given situation (i.e., claustrophobic fear versus embarrassment because the exercises may draw passengers’ attention).

### ***When Skill Deficits Accompany the Phobia***

Longstanding phobias (with onset in childhood) – phobias of animals, the natural environment, and BII type often start in childhood while most situational phobias usually start in early adulthood (e.g., Becker et al., 2007; Lipsitz et al., 2002) – may have prevented development of appropriate skills for the phobic situation.

For example, individuals with longstanding fears of dogs may not have developed skills for appropriately interacting with animals, and may require training in how to approach, touch, or interact with an animal (e.g., Rentz, Powers, Smits, Cogle, & Telch, 2003). Similar considerations are apt for driving fears, where the therapist should remain vigilant to poor driving habits which may both increase the fearfulness and the actual risk of the driving experience (see Taylor, Deane, & Podd, 2007).

### ***When Anticipation Is Worse than Exposure***

The maladaptive cognitions associated with the phobias tend to be future-oriented perceptions of danger or threat (e.g., what is about to happen, what will happen). This sense of danger may involve either physical threat (e.g., having a heart attack) or psychological (e.g., anxiety focused on embarrassment). In addition, these cognitions tend to focus upon a sense of uncontrollability over the situation or symptoms of anxiety.

Related to the issue of risk assessments is the fact that anticipatory anxiety is often worse than fear during exposure. A striking overprediction of fear before the exposure compared to the limited increase in fear during the exposure itself has frequently been observed (Alpers & Sell, 2008; Johansson & Oest, 1982; Rachman & Bichard, 1988).

In a study with a height exposure task in a theme park we showed that, for all participants, fear, dizziness, and body sway were increased during exposure (Alpers & Adolph, 2008). However, anticipated fear most reliably predicted body sway during exposure. In addition, persons scoring high on trait fear of heights anticipated and experienced more fear during exposure, but this relationship was not found for any objective measure.

Attending to overestimations in the likelihood of catastrophic events is one way of addressing these anticipatory fears. A focus on danger or harm is most frequently associated with the natural environment and situational subtypes (Lipsitz et al., 2002). Patients with a fear of flying may overestimate the risk of plane crashes, those with claustrophobia may exaggerate the risk of suffocation in an elevator, and those with the specific phobia of driving are often concerned with their own ability to drive. To help with anticipatory anxiety, patients may be asked to research facts about the feared situation prior to the initiation of exposure. This information is then applied in a cognitive-restructuring format to help patients generate more accurate expectations prior to exposure.

### ***When Patients Use Cognitive Avoidance During Exposure***

Even if patients agree to pursue exposure, this strategy may sometimes fail to result in a sizable extinction of fear responses. One reason is related to cognitive avoidance during exposure. Theoretically, avoidance or distraction should result in less

extinction (Foa & Kozak, 1991, 1986; Rodriguez & Craske, 1993). This is supported by studies that found less treatment response in patients who were distracted from experiencing the full extent of fear during exposure (Grayson, Foa, & Steketee, 1982; Kamphuis & Telch, 2000; Mohlman & Zinbarg, 2000) or more return of fear (Haw & Dickerson, 1998). However, the detrimental effects of safety behavior (subtle avoidance strategies designed to reduce fear during exposure) have not always been replicated (Antony, McCabe, Leeuw, Sano, & Swinson, 2001; Milosevic & Radomsky, 2008).

In a recent study with claustrophobic patients it was evident that those who were encouraged to use safety behaviors during exposure showed significantly more fear at post-treatment and follow-up relative to those encouraged to focus and reevaluate their core threats during exposure (Sloan & Telch, 2002). Particularly during imaginal exposure, safety behavior is frequent and linked to poorer outcome (Rentz et al., 2003). Thus, patients should be instructed not to use subtle avoidance strategies and to fully experience the symptoms and feelings provoked by exposure.

### ***Vigilance to Threat***

Eysenck's (1992) hypervigilance theory proposes that anxious people scan their environment excessively and broaden their attention span when searching for a fear-relevant stimulus and narrow it while that stimulus is processed. This hyperactive alarm system may lead to frequent and intense false alarms in fearful subjects (Becker & Rinck, 2004). The subsequent narrowing of attention may explain why patients have difficulties to disengage attention from spider distractors before moving on to the target. In contrast to several theoretical accounts, there is little evidence that attentional engagement to phobic cues actually occurs automatically (Alpers et al., 2009). Instead, patients with a spider phobia seem to have a deficit in disengaging their attention from spider cues (Gerdes, Alpers, & Pauli, 2008; Gerdes, Pauli, & Alpers, 2009). Under naturalistic conditions, phobic patients have also been shown to scan their environment for threatening stimuli (Lange, Tierney, Reinhardt-Rutland, & Vivekananda-Schmidt, 2004). Although specific interventions to address the dysfunctional allocation of attention are not yet available outside of the laboratory (see Mathews & MacLeod, 2002), patient behavior should be closely observed and observations such as excessive scanning should be addressed.

### ***When Self-Report Is Not Predictive of Emotional Processing***

Assessing verbal report during treatment is often not enough to assess emotional activation. Multiple response theory states that phobic fear is reflected in autonomic nervous system activation, self-report, and avoidance behavior (Lang, 1968). Despite the positive reaction this statement has created with theorists (e.g., Foa & Kozak, 1991), physiological measurement is often neglected, partly because its contribution of useful and unique information has not been convincingly documented. However, in a recent study we documented that although claustrophobic

patients are activated both in self-report and in physiology while being exposed to a fear-related situation such as a small confined space, measuring heart rate can provide incremental information (Alpers & Sell, 2008). Although initial fear activation during exposure as indicated by self-report is not predictive of therapeutic change, higher heart rate responses to exposure correlate with more therapeutic change from before to after exposure treatment. Thus, whenever possible, physiological measurements should be consulted to monitor the effects of exposure treatment.

### *Enhancing Memory of Success*

The return of fear in successfully treated patients is often thought of as a process similar to reinstatement of extinguished fear in the conditioning model (Bouton, 2002). To aid retention of exposure across the multiple contexts in which a phobic cue may be experienced in the future, extinction training in multiple contexts may be important (e.g., for animal work see Gunther, Denniston, & Miller, 1998). A superiority of variable context exposure training can also be observed in therapy with humans, especially in longer-term follow-up assessments (Vansteenwegen, Vervliet, Hermans, Thewissen, & Eelen, 2007; for review see also Craske et al., 2008). Hence, variability in the way in which exposure is conducted (in different settings, at different times of day, with and without therapist accompaniment) may aid longer-term fear reduction.

Also, there are now data suggesting that pharmacological cognitive enhancers (e.g., d-cycloserine) may increase the efficacy of exposure-based interventions in specific phobias (acrophobia Ressler, Rothbaum, Tannenbaum, Anderson, Graap, Zimand et al., 2004). Exposure therapy combined with d-cycloserine resulted in significantly larger reductions of acrophobia symptoms on all main outcome measures. Promising initial findings have also been reported for the augmentation of exposure therapy for claustrophobia with yohimbine, another putative cognitive enhancer for extinction learning (Powers, Smits, Otto, Sanders, & Emmelkamp, 2009). However, whether such adjunctive treatment with putative memory enhancers can help reduce the rate of treatment non-response to CBT has not been systematically evaluated.

### **Conclusion**

It is appropriate that specific phobias are no longer called simple phobias. Specific phobias are highly prevalent and they can result in significant life impairment. The multitude of different fears and the considerable level of comorbidity imply that treatment needs to be individually tailored. There are several etiological pathways to the development of specific phobias and the individual's learning history should be considered for treatment. Exposure and response prevention is the gold standard for treatment. Variants of this treatment approach have proven to be highly effective with lasting effects on multiple levels of responses (self-report, physiological reactivity, and behavioral avoidance). The specific ways in which exposure is conducted

likely influences the robustness of outcome. This chapter attended to factors that need to be considered in planning and for adequate exposure situations, sufficient frequency, and duration of exposure exercises. Possible treatment complications, when other emotions come into play and when risk assessments call for specific interventions, were highlighted.

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**Part III**  
**Treatment Complications**  
**in Special Populations**

# Resolving Treatment Complications Associated with Comorbid Depression

Christen M. Deveney and Michael W. Otto

## The Impact of Comorbid Depression on Treatment for Anxiety

Brad is a 30-year-old male who presents to the clinic with complaints of frequent, intense, unexpected panic attacks as well as symptoms of major depression. Following a functional analysis of his symptoms, Brad and his therapist agree to an initial treatment focus on panic disorder. As treatment progresses, and despite understanding the treatment model and reporting motivation for conducting exposure exercises, Brad has difficulty completing his exposure homework. He reports that it is hard for him to gather the motivation to conduct the exposures without help from his therapist and only manages to practice one exposure per week. He considers this to be a sign that he is a “failure.” During treatment sessions he sits slouched in his chair with a sad expression on his face. Brad sighs and says that he is considering stopping treatment, because “it isn’t working.” This thought quickly spirals into a series of thoughts about how he will never get better and will never be able to support himself” so he will have to return home and live with his parents or live on the street.” This coincides with a week of frequent and intense panic attacks, significant anticipatory anxiety and avoidance, as well as an exacerbation of depression symptoms – including passive suicidal ideation.

Brad’s case effectively represents some of the issues encountered by clinicians in the treatment of comorbid anxiety and mood disorders. The choice to initiate a treatment focus on the anxiety disorder, if it is the primary source of distress and disability, rather than the depression is well supported by the treatment literature. In many cases, the empirical literature suggests that cognitive behavioral therapy (CBT) for anxiety disorders is resilient to comorbid depression. There is also evidence that depression symptoms improve with treatment of a primary anxiety disorder (see below). Nonetheless, as illustrated by Brad’s case, there are treatment challenges introduced by the depression. Depression has been associated with increased clinical severity and therefore may require additional treatment sessions and/or additional treatment strategies to fully address all anxiety and depression symptoms. Depression may influence motivation for treatment in general as well as the perceived ability to complete specific assignments and may change the evaluation of treatment progress. Also, depression brings with it waxing and waning

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symptoms that may demand attention therapeutically, ranging from variable levels of distress in session to the importance of monitoring and intervening with suicidal ideation.

In the sections that follow, we will review the nature and impact of depression comorbid with anxiety disorders. We will also address treatment modifications that can be important for treating anxious individuals who have comorbid depression.

## The Impact of Comorbid Depression

Comorbidity between anxiety and major depressive disorders (MDDs) is associated with greater severity of anxiety symptoms. For example, increased panic disorder severity, help seeking, role impairment, and symptom persistence have been routinely observed among patients with panic disorder and comorbid major depression relative to non-comorbid individuals (e.g., Brown, Antony, & Barlow, 1995; Joermann, Kosfelder, & Schul, 2005; McLean, Woody, Taylor, & Koch, 1998; Roy-Byrne et al., 2000). Elevated panic symptoms frequently remain after treatment among individuals with higher depression scores (e.g., Keijsers, Hoogduin, & Schaap, 1994). Symptom severity was also higher in a social anxiety disorder and comorbid major depression group relative to social anxiety disorder alone and social anxiety disorder and comorbid anxiety disorder comparison groups (Erwin, Heimberg, Juster, & Mindlin, 2002). At times, it appears that the driving force behind this increased clinical severity may reflect an increase in general anxiety rather than elevations in symptoms related to specific anxiety disorders. For example, general anxiety, but not disorder-specific symptoms, was more severe among individuals with primary obsessive-compulsive disorder (OCD; Overbeek, Schruers, Vermetten, & Griez, 2002) or social anxiety disorder (Turner, Beidel, Wolff, Spaulding, & Jacob, 1996) and comorbid depression relative to a non-depressed group.

Depression is also associated with an intensification of anxiety-related cognitions. For example, social anxiety disorder with depression was associated with even more marked negative evaluations of social performances than a non-comorbid group (Ball, Otto, Pollack, Uccello, & Rosenbaum, 1995; Wilson & Rapee, 2005 see also Bruch, Mattia, Heimberg, & Hoit, 1993). A similar relationship has been noted for somatic sensation fears among panic disorder patients with comorbid depression relative to a non-comorbid group (Otto, Pollack, Fava, Uccello & Rosenbaum, 1995).

Another impact of comorbid depression is reduced motivation for treatment. Marks (1987) reported reduced motivation to engage in treatment or complete self-directed exposures among anxiety patients with comorbid depression. Also, two studies found an association between decreased post-traumatic stress disorder (PTSD) treatment adherence and pretreatment depression scores (e.g., Bryant, Moulds, Guthrie, Dang, & Nixon, 2003; McDonagh et al., 2005; c.f. Resick, Nishith, Weaver, Astin, & Feuer, 2002). One source of this adherence difficulty may be the negative attitudes and self-evaluations that are endemic to depression. As discussed by Telch (1988), depressogenic cognitions should be expected to impact

evaluations of success following exposure exercises. For example, an individual with social anxiety disorder and comorbid depression may be more likely to judge his/her exposure performance in the context of larger depressogenic beliefs (e.g., “I wasn’t perfect in the exposure, which is just one more sign of my worthlessness”). In addition, failure to recognize the positive outcome of an exposure exercise (e.g., “I just did something very difficult and my worst fears did not happen”) may minimize the safety learning considered to be essential for the successful resolution of anxiety symptoms. These depressive cognitions may limit treatment gains by reducing patients’ expectations or ability to learn from anxiety treatment interventions (Safren, Heimberg, & Juster, 1997).

Another independent effect of depression on adherence may be via an impact on problem solving. Stress management and problem solving appears to be reduced by depression, and may lead patients with depression to feel that much more overwhelmed by stressors. As a result, homework assignments, which by their nature are stressful, may be perceived as an unmanageable burden and therefore avoided. Finally, symptoms such as anhedonia in combination with sad mood and decreased energy may reduce motivation for the benefits that exposure therapy can bring – the ability to engage in more personal, social, and role activities. Hence, the “vision” of what successful anxiety treatment may bring in terms of enhanced functioning may be both less salient and less important for individuals with severe depression.

Increased symptom severity, increased negative cognitions, and decreased willingness and ability to engage in treatment-related as well as pleasurable activities may all combine to reduce an individual’s ability to engage in anxiety treatment. Missed appointments and low motivation have been associated with poorer treatment outcome (Dugas et al., 2003; Tarrier, Sommerfield, Pilgrim, & Faragher, 2000), even in studies where comorbid major depression itself did not impact treatment outcome (Tarrier et al., 2000). Hence, clinicians should be prepared to work harder to help patients with depression engage adequately in treatment.

## **Impact of Comorbid Depression on Anxiety Treatment Outcome**

A critical issue for consideration is the degree of impact comorbid depression has on anxiety disorder treatment outcome. In the following section we review the existing literature for panic disorder, social anxiety disorder, OCD, and PTSD. Although there are some equivocal findings for each diagnostic group under consideration, overall there is evidence that CBT for anxiety disorders is often robust in the presence of comorbid depression.

### ***Panic Disorder***

Empirical investigations of the impact of comorbid depression upon CBT for panic disorder have been the most extensive (see Mennin & Heimberg, 2000 for review) and indicate that CBT for panic is frequently resilient to comorbid depression.

Despite some mixed findings, it appears as if neither pretreatment depression symptom severity (e.g., Beck Depression Inventory scores; Başoğlu et al., 1994; Black, Wesner, Gabel, Bowers, & Monahan, 1994; Jansson, Öst, & Jerremalm, 1987) nor the presence of a diagnosable mood disorder (Barlow, Gorman, Shear, & Woods, 2000; Brown et al., 1995; Kampman, Keijsers, & Hoogduin, 2008; Laberge, Gauthier, Cote, Plamondon, & Cormier, 1993; Maddock & Blacker, 1991; McLean et al., 1998; Wade, Treat, & Stuart, 1998) reduces the efficacy of CBT for panic disorder. Similarly, the presence of a comorbid mood disorder at pretreatment does not appear to influence panic relapse (Otto, Pollack, and Sabatino, 1996). The durability of CBT for panic disorder extends to more naturalistic settings as CBT for panic disorder was not markedly influenced by the presence of major depression, despite the greater clinical impairment among the comorbid group, as evaluated in a German community clinic (Joormann et al., 2005). Consistent with our prior discussion of increased symptom severity among individuals with comorbid depression, the literature indicates that despite greater symptom severity at baseline in the comorbid group, individuals with panic disorder and comorbid depression are able to make similar treatment gains as compared with individuals with panic disorder only (McLean et al., 1998).

Despite these promising outcomes, some studies have linked pretreatment major depression to poorer acute outcome (Başoğlu et al., 1994; Maddock & Blacker, 1991; Steketee, Chambless, & Tran, 2001) as well as increased panic symptoms and decreased social adjustment over a long-term period (Noyes et al., 1990). There is less evidence for a relationship between treatment outcome and pretreatment depression severity scores (e.g., Başoğlu et al., 1994). Accordingly, we are left with the conclusion that in many cases, CBT for panic disorder is robust over the long term in the face of comorbid depression (see Mennin & Heimberg, 2000 for review). Yet, given the initial starting and ending severity differences, more treatment may be required to bring patients with comorbid depression to the desired outcome.

### ***Social Anxiety Disorder***

As with panic disorder, several clinical trials indicate that the presence of comorbid depression does not have a marked impact on treatment outcome for social anxiety disorder (e.g., Van Velzen, Emmelkamp, & Scholing; Turner et al., 1996). The strongest evidence comes from a study by Erwin et al. (2002), who indicated that individuals with social anxiety disorder exhibited similar rates of improvement, regardless of whether they had comorbid major depression. These findings are echoed by a recent meta-analysis by Lincoln and Rief (2004) which indicated that treatment outcome for social anxiety disorder did not differ depending on whether comorbid major depression was permitted or excluded from the clinical trial. Indeed, effect sizes for the two groups were virtually identical ( $d = 0.091$  for studies including comorbid major depression and  $d = 0.92$  for studies excluding comorbid major depression; Lincoln & Rief, 2004).

Despite these promising results, two studies indicate that pretreatment depression is associated with poorer treatment outcome for social anxiety (Chambless, Tran, & Glass, 1997; Scholing & Emmelkamp, 1999). In the Chambless et al. (1997) study, pretreatment depression scores predicted negative treatment outcome beyond that of other measures of anxiety and anxious apprehension. However, pretreatment depression scores may be most related to early measures of treatment outcome, as there was no relationship between pretreatment depression and outcome at an 18-month follow-up assessment in the study by Scholing and Emmelkamp (1999). Accordingly, given the compelling findings from the meta-analysis, clinicians can be at least moderately confident that outpatients with social phobia and depression will have the opportunity to improve to similar degree as outpatients without comorbid depression, especially when assessed over a long-term period.

## ***OCD***

Empirical investigations of the impact of depression on treatment outcome for OCD are more limited than for panic or social anxiety disorders, yet there are some similarities with the preceding literature. As with other anxiety disorders, comorbid depression has been associated with higher OCD symptom scores both before and after treatment relative to non-comorbid individuals (Abramowitz & Foa, 2000). Yet this increased severity does not appear to hinder the ability of individuals with comorbid major depression to make equivalent gains in treatment as compared with their non-comorbid counterparts (e.g., Abramowitz, Franklin, Street, Kozak, & Foa, 2000; Abramowitz & Foa, 2000). Indeed, two studies suggest that OCD treatment efficacy was not affected by the presence of pretreatment depression (Abramowitz & Foa, 2000; Orloff et al., 1994), and pretreatment depression scores did not differ between treatment completers and noncompleters in at least one study (Franklin, Abramowitz, Kozak, Levitt, & Foa, 2000).

This small literature does include some evidence suggestive of a negative impact of depression on OCD treatment outcome. Steketee et al. (2001) reported that the presence of comorbid depression has an adverse effect on post-treatment and 6-month follow-up outcome. Work by Abramowitz et al. (2000) suggests that treatment gains are limited among individuals with severe (relative to mild or moderate) depression. Finally, a recent meta-analysis associated comorbidity (including depression) with reduced treatment effect sizes (Rosa-Alcázar, Sánchez-Meca, Gómez-Conesa, & Marín-Martínez, 2008). Although this literature is somewhat mixed, several studies indicate that individuals with OCD and comorbid depression can benefit from CBT for OCD and that comorbid depression is unrelated to treatment compliance. Much less confidence is appropriate when treating an individual with severe depression, as this may be related to poorer outcome. Additional research is necessary in order to clarify the influence of comorbid depression upon CBT for OCD.

## ***PTSD***

As with OCD, empirical investigations of the impact of comorbid depression on treatment outcome for PTSD are limited. Several studies indicate that the presence of comorbid depression is unrelated to PTSD treatment outcome (Ehlers, Clark, Hackmann, McManus, & Fennell, 2005; Gillespie, Duffy, Hackmann, & Clark, 2002; TARRIER et al., 2000) and treatment attrition (e.g., Resick et al., 2002), although additional treatment sessions may be required to attain the same clinical outcome (Gillespie et al., 2002). A negative impact of comorbid depression on treatment outcome for PTSD is documented in three studies. Taylor et al. (2001) noted that higher pretreatment depression scores were associated with partial relative to full response to CBT, and two studies related decreased treatment adherence with increased pretreatment depression scores (e.g., Bryant et al., 2003; McDonagh et al., 2005; c.f. Resick et al., 2002). Together, the evidence suggests that response to CBT for PTSD may not be affected by the presence of comorbid major depression, although additional treatment sessions or particular attention to patient engagement in treatment may be required in order to maximize treatment outcome.

## ***Conclusions from the Treatment Outcome Literature***

In an effort to clarify the scope of our chapter, the following limitations are important to note. First, the literature reviewed above concerned treatment for populations with a primary anxiety disorder and secondary depression. Primary depression may negatively impact treatment for secondary anxiety more substantially than our review above suggests. Indeed, several of the studies excluded individuals with depression that was severe enough to require hospitalization, re-emphasizing the secondary nature of the depression considered in the studies reviewed above. Second, anxiety disorders are frequently comorbid with *bipolar* disorder, yet treatment outcome studies concerning the impact of bipolar depression on anxiety treatment are lacking in the published literature. Consequently, this review focused on the effects of *unipolar* depression on anxiety treatment. Third, many of these studies reviewed above included small sample sizes that may be underpowered to detect small influences of comorbid major depression on treatment outcome. Therefore, while it is possible that depression has a small effect on anxiety treatment outcome, it is unlikely to have a marked negative impact upon treatment response in this population. Finally, no studies examined whether the duration or early onset of depression predicted anxiety treatment outcome. Given the evidence that depression chronicity is a predictor of poor outcome in depression treatment studies (Keller et al., 1992; Stewart et al., 1989), it is possible that individuals with early onset of recurrent depressive episodes may exhibit lower treatment responses to CBT for anxiety and later-onset comorbid depression may have a negligible effect on anxiety treatment. Therefore, additional research is necessary before definitive conclusions

can be drawn about the exact nature of the relationship between comorbid MDD and anxiety treatment outcome. What is clear is that despite increased symptom severity before and after treatment, treatment gains need not be limited by the presence of a comorbid mood disorder, but these patients may require additional attention in treatment.

## **Impact of CBT for Anxiety Disorders on Depression Symptoms**

One particularly promising finding is that in many cases depression symptoms appear to resolve significantly following anxiety treatment with CBT. For example, the percentage of individuals with panic disorder who met criteria for comorbid depression (MDD and dysthymia) decreased from 18% to 6% following panic treatment (Tsao, Mystkowski, Zucker, & Craske, 2002). Significant decreases in the percentage of individuals meeting criteria for MDD have also been observed following CBT for PTSD (Blanchard et al., 2004; Resick & Schnicke, 1992).

Depression severity reductions have also been observed across several anxiety populations including panic disorder (Joormann et al., 2005; Öst, Thulin, & Ramnerö, 2004; Tsao et al., 2002), social anxiety disorder (Joormann et al., 2005), generalized anxiety disorder (GAD) (Borkovec & Costello, 1993; Ladouceur et al., 2000; Öst & Breitholtz, 2000), and OCD (Rosa-Alcázar et al., 2008). Indeed, almost all studies of PTSD indicate a significant decrease in depression severity scores following CBT (e.g., Bryant, Harvey, Dang, Sackville, & Basten, 1998; Bryant et al., 2003; Cloitre, Koenen, Cohen, & Han, 2002; Ehlers et al., 2003; 2005; Foa et al., 1999; Marks, Lovell, Noshirvani, Livanou, & Thrasher, 1998; Paunovic & Öst, 2001; Resick et al., 2002; Resick & Schnicke 1992, see Foa, Rothbaum, Riggs, & Murdock, 1991 and McDonagh et al., 2005 for failures of CBT to reduce depression scores to a greater degree than a wait-list control group). Two studies suggest that the impact of CBT for anxiety disorders on depression symptoms is dramatic. Moscovitch, Hofmann, Suvak, and In-Albon (2005) indicated that social anxiety symptom improvement mediated 91% of subsequent depression symptom improvement and one meta-analysis suggests that CBT for (GAD) reduced depression symptoms to a greater degree than pharmacotherapy (Gould, Otto, Pollack, & Yap, 1997). Therefore, the majority of studies indicate that CBT for anxiety disorders results in a reduction of comorbid depression, even when depression symptoms are not a target of treatment.

Given research suggesting that the addition of CBT specifically aimed at reducing depression after the conclusion of anxiety treatment successfully reduces remaining depressive symptoms (Woody, McLean, Taylor, & Koch, 1999), augmenting treatment with a booster session focusing on depression may be implemented for those individuals whose depression scores are not alleviated by CBT for anxiety. In addition, some evidence suggests that anxiety interventions that

target cognitive restructuring may result in greater depression severity reductions than interventions that rely on exposure interventions (Bryant et al., 2003; Harvey, Bryant, & Tarrier, 2003). Consequently, a therapist may choose a treatment protocol that emphasizes cognitive restructuring as well as exposure interventions for use with comorbid patients.

There are a number of ways in which common treatment elements for anxiety and depression may facilitate symptom reductions in both disorders. For example, although negative thinking patterns in anxiety often involve fears of catastrophic consequences and are distinct from the more self-punitive thoughts observed in depression, the cognitive restructuring skills typically employed in CBT provide patients with skills to identify, evaluate, and modify maladaptive negative thinking styles more generally. Training individuals to treat their thoughts as ideas to be considered and evaluated rather than taken as fact may facilitate a reduction in maladaptive responses to both anxiogenic and depressive thoughts. For example, restructuring negative automatic thoughts common in social anxiety disorder (“I will stumble over my words and look like an idiot”) requires similar techniques as restructuring negative thoughts associated with MDD (“I am unlovable”). As a result, depression-related thoughts, and therefore depression symptoms, may be reduced following anxiety treatment.

Treatment for anxiety and depression encourages a systematic approach toward symptom reduction and re-engagement with the world. In anxiety, the creation of fear hierarchies and gradual conduct of exposure exercises encourage patients to decrease avoidance behavior. Increased engagement in pleasurable activities is a core component of CBT and behavioral activation treatments for MDD (Jacobson, Martell, & Dimidjian, 2001). It is possible that the reduction of avoidance behaviors following anxiety treatment promotes active engagement in meaningful and pleasurable activities thereby ameliorating both anxiety and depressive symptoms. For example, a panic patient’s increasing comfort using public transportation or driving long distances following exposures may result in increased visits to friends and family. The patient’s increased social engagement may then decrease depressive symptoms associated with loneliness and isolation.

Finally, anxiety and depression are both characterized by effortful suppression and avoidance of emotional experiences (Campbell-Sills, Barlow, Brown, & Hofmann, 2006). Several treatment strategies for anxiety encourage patients to experience, acknowledge, tolerate, and accept their affective states and negative thoughts while continuing to strive toward their goals (e.g., Barlow, Allen, & Choate, 2005; Hayes, Strosahl, & Wilson, 1999; Otto, Powers, & Fischman, 2005). Exposure exercises for OCD encourage tolerance and observation of anxious states in order to view the extinction process and learn that rituals are not necessary in order for anxiety to decrease. To the extent that an acceptance of emotions represents a general treatment factor that encourages more adaptive responses to marked affective states (Barlow et al., 2005), reductions in depression symptoms may result following the acknowledgement of negative affect and associated thoughts. These

skills may be translated to other situations and emotions, leading to a reduction in both anxiety and depressive symptoms.

## **Treatment Considerations for Comorbid Anxiety and Depression**

Although it is unlikely that comorbid major depression will routinely prevent an individual from benefiting from anxiety treatment, the literature suggests that clinicians should expect some costs associated with the presence of a mood disorder at least some of the time. For example, clinicians should anticipate that individuals with comorbid anxiety and depression will exhibit higher symptom severity scores, may have poorer motivation, and may exhibit thinking patterns that exacerbate their anxiogenic thoughts and interfere with treatment compliance. Consequently, we turn to a discussion of several treatment considerations that may help clinicians to maximize successful outcome with comorbid populations.

First, both the patient and the clinician should consider that additional treatment sessions may be required due to the presence of greater pre- and post-treatment clinical severity among individuals with comorbid conditions. In addition, treatment of one disorder may fail to address the comorbid condition at all or to resolve symptoms completely. Some studies have used additional sessions as a successful way of addressing the increased symptom severity likely to be present in comorbid populations (e.g., Gillespie et al., 2002), but additional protocols or strategies for the comorbid illness may be required.

Second, an initial decision on treatment ordering is necessary when addressing comorbid conditions. As long as the anxiety disorder is primary (the primary source of distress and disability), we routinely recommend attending to this disorder first as was done in the case example that opened this chapter. However, because treatment adherence is greatest when participant expectations and treatment methods are aligned (Eisenthal, Emery, Lazare, & Udin, 1979; Schulberg et al., 1996), it is important to elicit information about the patient's most pressing concerns and treatment goals. Developing a collaborative relationship with the patient and conducting a thorough analysis of the relationship between symptom clusters may facilitate treatment ordering decisions. For example, if a patient's depression primarily results from repeated panic attacks that interfere with her social and occupational abilities, she may benefit most significantly from initial treatment of panic disorder. Depression symptoms may resolve naturally as panic symptoms subside and as skills learned in CBT for panic are translated to her depression symptoms. Additional treatment for MDD may be considered if her mood symptoms remain elevated following successful panic treatment. On the other hand, if a patient's depression is severe enough to hinder her ability to conduct or benefit from the exposure exercises, treating the depression symptoms first using cognitive restructuring and behavioral activation may lead to a more optimal outcome. See the following case descriptions for an example.

## Box 1 – Case Examples

**Case #1:** Sally arrives at the clinic requesting treatment for panic attacks occurring daily for the past 10 years. Some of her panic attacks occur out of the blue, but some are triggered by reminders of prior traumatic events resulting in a diagnosis of PTSD. The frequency of her panic attacks and PTSD symptoms has led to a lengthy and severe depression over the past several years and she has been unable to work and take care of many household responsibilities. After some conversation, it appears as if Sally's depression primarily stems from the impairment caused by her frequent and intense panic attacks. In addition, Sally and her therapist believe that some of the skills associated with panic disorder treatment may be translated into future treatment for PTSD. Consequently, they agreed to target panic symptoms first, PTSD second, and depression third, with regular assessments of symptoms in each domain throughout treatment. Sally responded well to CBT for panic disorder, noting a decrease in both trauma-uncued and trauma-cued panic attacks over time. As the frequency of panic attacks decreased, Sally was able to function more effectively at home and noticed an improvement in her social relationships which helped to ameliorate her depressive symptoms. Further, Sally reported a decrease in anxiety in response to trauma-related situations due to increased coping skills as well as enhanced motivation to begin PTSD treatment as a result of her successful treatment for panic disorder.

**Case #2:** Jill arrives at the clinic requesting treatment for frequent obsessions and compulsions. Jill spends up to 6 hours/day completing complex rituals and is unable to work. She reported having difficulty with depression throughout her life and is currently experiencing a marked period of sad mood, anhedonia, poor energy, decreased appetite, psychomotor retardation, difficulty concentrating, and passive suicidal ideation. Jill and her therapist evaluate the relationship between Jill's OCD and depressive symptoms and it appears as if Jill's depression sometimes occurs independently of her OCD symptoms, but the onset of the current depressive episode began after a marked increase in compulsions and the resulting loss of her job. Consequently, Jill and her therapist believe that Jill's current MDD symptoms are the result of the recent OCD exacerbation and they agree to target OCD symptoms before turning to address her depression. However, Jill had difficulty completing exposure exercises in session, frequently remarking that she "isn't strong enough" and that "treatment won't work for me." In addition, Jill was unable to complete homework exercises, reporting that low energy and motivation made it hard for her to purposefully challenge her anxiety. Because Jill's depressive symptoms were making it difficult for her to complete and learn from the exposure exercises, Jill and her therapist agreed to change the focus of

treatment and address Jill's depressive symptoms. Following behavioral activation and cognitive restructuring interventions, Jill's depressive symptoms remitted somewhat and she resumed OCD treatment. Once Jill's marked MDD symptoms no longer interfered with her ability to conduct exposure exercises and her behavioral activation exercises provided her with opportunities to benefit from naturally occurring exposures, Jill experienced significant symptom relief from repeated exposure exercises and the resulting habituation. This success, in turn, further alleviated her remaining depressive symptoms.

The research literature suggests that Jill (Case #2) would likely benefit from OCD treatment despite the presence of depression symptoms. However, given the impact of depression on treatment compliance, Jill and her therapist adjusted their treatment plan when it was clear that Jill's comorbid depression was influencing her ability to perform the homework exposures necessary for maximum treatment benefit. It is possible that similar changes in treatment emphasis may help individuals whose depression is impeding treatment progress to a significant degree. However, therapists will frequently encounter more subtle interference in daily sessions that can negatively impact treatment goals. For example, comorbid depression frequently results in the display of tearfulness or obvious low mood in session, whereas anxiety and avoidance may be more likely to occur outside of session and away from a clinician's immediate observation. Signs of in-session distress are often powerful and can lure a therapist into addressing these symptoms at the expense of the anxiety treatment. Deciding whether to respond to in-session distress or focus on the larger treatment goals is a difficult process. Returning to the initial functional analysis of symptoms developed in collaboration with the patient may help the therapist to focus on the long-term goals relative to the patient's short-term distress. For example, a therapist may be tempted to use cognitive restructuring techniques to challenge the negative thoughts (e.g., "I will never have friends") of an individual with social anxiety and MDD, taking away time available during the session to devote to exposures. Yet, the patient may benefit most from participating in exposure exercises designed to help him reduce his social anxiety and facilitate his ability to develop social networks. These exposure exercises may also provide him with an opportunity to challenge the maladaptive thoughts that help to perpetuate his negative mood. Given prior evidence that depression symptoms resolve following social anxiety treatment (e.g., Moscovitch et al., 2005), it may be in the patient's best interest to minimize attention to his in-session distress and actively promote the techniques that will move him toward his long-term goals. The one exception to this principle is when suicidal ideation or intent arises. Regular assessment of suicidal ideation is a must in comorbid patients and, when present, may well redirect therapeutic interventions to safety issues. Cognitive therapy emphasizing cognitive restructuring and problem solving is efficacious for those at suicidal risk (Berk,

Henriques, Warman, Brown, & Beck, 2004; Brown et al., 2005) and should be considered as part of an integrated treatment approach when serious suicidal ideation is confronted in comorbid patients.

Third, a recent study found that a brief motivational interviewing intervention prior to treatment for panic disorder, social anxiety disorder, or GAD increased engagement with CBT and resulted in improved outcome (Westra & Dozois, 2006). Consequently, attention to engaging the participant in treatment as a means of combating low motivation among anxiety patients regardless of comorbidity appears worthwhile. In order to promote engagement with the treatment, we recommend encouraging homework compliance, taking care to clearly write down all homework assignments, systematically review homework progress at each session, and emphasizing the crucial nature of at-home practice. Similarly, although treatment gains are always applauded, extra attention is given to early treatment gains to help maintain a client's motivation for treatment and facilitate the ability of patients to link their efforts with demonstrable changes in their symptom and abilities.

Fourth, because low motivation and energy can make it more difficult for individuals to fully utilize CBT strategies, extra attention is paid toward preparing patients for mood-specific cognitive biases and ways that they can challenge those biases. For example, we attempt to give patients a vivid example of the powerful effects of moods on thought patterns by examining how easily our beliefs are manipulated in movies. Although we may know that the movie sequel starring the main character is already being filmed, the ominous music and seemingly insurmountable obstacles during the movie climax contribute to a temporary conviction that the main character is doomed. Negative affect common to depression and anxiety can function like the background "mood music" present in these films and can increase the likelihood that we will believe negative and false ideas that would be evaluated differently in neutral or pleasant mood states. Therefore, thoughts can *feel* true during times of marked depressed mood. However, "feeling" more believable does not increase the *actual* accuracy of those beliefs. Thoughts about one's inability to manage anxiety during depressed moods can be discussed in this framework. Psychoeducation is paired with cognitive restructuring and coping skills designed to neutralize the negative thought that interferes with treatment progress. Practicing this skill *before* patients encounter such negative thoughts during exposure exercises may help the patients reap the benefits of these exercises to a greater degree.

Fifth, negative thinking styles characteristic of depression may impair a patient's ability to learn from exposure exercises and to acknowledge treatment progress (e.g., "this isn't working"; "I won't get better"). Consequently, practicing the recognition of and coping strategies for these thoughts during the session can have significant benefit for the patient when they encounter these thoughts on their own or during exposure exercises. For example, negative thoughts about treatment (e.g., "this won't work") are likely to interfere with the individual's willingness to conduct an exposure and a therapist may benefit from identifying and restructure this thought early in treatment (e.g., "I don't know if this will work but it can't hurt to give it a try"). Therefore, when the patient has the initial thought that treatment won't work outside of the session, s/he may already have a ready and adaptive

response to the thought which may promote compliance with exposure homework. Challenging negative thinking styles in advance may be of particular importance when addressing long-standing depressive cognitions related to core beliefs (e.g., “I am a failure”). Hofmann and Otto (2008) addressed this issue in a recent treatment manual. They discuss the case of Bob, a client with social anxiety disorder and comorbid depression whose negative self-statement, “I am a failure,” was impeding treatment of his social anxiety disorder and did not respond to traditional cognitive therapy strategies. The therapist chose to use an exposure-based technique in order to help neutralize these negative thoughts and promote progress in social anxiety treatment. Selected text from this portion of the manual is reproduced below.

## **Box 2 – Using Exposure Principles to Target Maladaptive Beliefs**

**Therapist:** Given how long you have been rehearsing that line, “I am a failure,” I am not going to try to directly break the habit of your saying it. But I do want you to know that it does not serve you well; as long as you buy into that thought, it keeps you stuck in your current position, making it hard for you to learn new ways of interacting socially. So, while respecting the power of your habit in saying, “I am a failure” to yourself, I am also going to ask you to help this phrase lose its current meaning. I want it to become a phrase that can no longer push you around emotionally or stand in your way of developing more social comfort. Starting today in session, I am going to ask you to expose yourself to this thought (“I am a failure”) by saying it over and over to yourself aloud. I want you to listen to yourself say this statement and, as you hear it, realize that this thought has traditionally pushed you around and made it harder for you to reach your goals. I want you to say it to yourself and let it lose some of its meaning. . .

Below are some of the examples of thoughts that went through Bob’s head as he was reading the phrase:

- I am a failure (boy, that is a nasty thought, generally makes a person feel lousy)
- I am a failure (yep, this is what I say to myself to make myself feel bad)
- I am a failure (pretty negative. . . this is my habit)
- I am a failure (I will just need to get bored with hearing this)
- I am a failure (yep, this is what I say over and over again)
- I am a failure (getting boring)
- I am a failure
- I am a failure
- I am a failure (yep, I use this thought to really screw myself over)
- I am a failure

I am a failure (time to really get bored with this)  
 I am a failure  
 I am a failure (time to take away all meaning from this thought)  
 I am a failure  
 I am a failure  
 I am a failure  
 I am a failure (here I go again with this old, negative thought)  
 I am a failure  
 I am a failure . . .

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*Cognitive behavioral therapy for social anxiety disorder*. New York:  
 Routledge, pp 117–118.

Finally, earlier in this chapter we discussed the similarities between systematically addressing items on a fear hierarchy and gradual participation in activities designed to increase behavioral activation. Both of these strategies emphasize systematic exposure to healthy behaviors that increase reinforcement and reduce avoidance. As individuals gradually increase their participation in activities that constitute a meaningful life, depression and anxiety symptoms should be reduced, indicating that these strategies may allow a therapist to target both symptom clusters successfully (Hopko, Lejuez, Ruggiero, & Eifert, 2003). For example, Hopko, Lejuez, and Hopko (2004) recently applied behavioral activation strategies to the treatment of an individual with comorbid panic disorder and depression (Hopko et al., 2004). Successful progression through a hierarchy of depression and anxiety-related behaviors ranked by level of ease was associated with a corresponding reduction in both symptom clusters. Consequently, when working with a population with anxiety disorders, we encourage the scheduling of activities that will help boost pleasant mood and encourage the patient to conduct exposure exercises.

## Concluding Comments

Because comorbidity rates between anxiety and depression are high in the research literature (e.g., Kessler et al., 1996) and clinical practice (e.g., Brown, Campbell, Lehman, Grisham, & Mancill, 2001), our goal in this chapter was to address the impact of depression comorbidity upon the treatment of anxiety disorders and to review strategies to resolve problems associated with this comorbidity. Fortunately, it appears as if the efficacy of CBT for anxiety disorders is not significantly reduced by the presence of MDD. Ability to engage in treatment and make treatment gains appears unaffected by the presence of MDD and depression symptoms are often significantly reduced by treatments targeting anxiety disorders. Yet the empirical literature also highlights several subtle ways in which comorbid depression may

influence anxiety treatment (e.g., increasing symptom severity, influencing motivation, decreasing ability to learn from exposure exercises). Therefore, we offered several suggestions to help minimize the smaller effects that comorbid depression may have upon treatment for anxiety disorders. We hope that these suggestions will allow clinicians to work effectively with challenging populations.

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# Resolving Treatment Complications Associated with the Presence of Comorbid Personality Disorders

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*Eva, a 36-year-old Hispanic woman, presented to an outpatient clinic for the treatment of social anxiety disorder (SAD). Eva was referred by her psychoanalyst, whom she had been seeing four times per week for the past two years, because the analysis did not appear to be improving her SAD and he believed cognitive-behavioral therapy (CBT) may be indicated. During his first meeting with Eva the CBT clinician conducted a structured diagnostic interview for Axis I disorders, which confirmed the presence of SAD as well as comorbid major depressive disorder. No assessment of Axis II disorders was performed given the case was referred as “a pretty straightforward SAD case” and the initial interview did not disconfirm this (nor did it try). The interview suggested that Eva’s depression was secondary to her SAD, as the negative automatic thoughts and other depressive symptoms she experienced seemed largely to result from her lack of engagement in pleasurable activities and her failure to have any social interactions outside of those that were completely necessary at work. Eva was referred to cognitive-behavioral group therapy for SAD, but refused to participate in a group because “I’ve had great difficulties with other women in the past. Men are always very attracted to me, and women get angry as a result, and I don’t want to participate in group therapy at this time because I don’t want all of that to get in the way of my treatment.” Eva began once weekly CBT for SAD, which initially proceeded just as planned. She attended each session on time and was very organized in session – creating a binder for all of her treatment-related materials, writing out a detailed homework list at the end of each session, and completing most of her homework assignments each week. However, several complications arose after the first few sessions. During the first in vivo exposure exercise, Eva became extremely upset. She cried for 15 minutes and made numerous self-critical statements such as “I’m so stupid! . . . I don’t deserve to get better! . . . No one will ever be with me!” The intensity and duration of Eva’s responses were much stronger than expected given the situation. A follow-up with Eva by the clinician revealed that Eva had been experiencing extremely intense responses to stressful*

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*events for many years. Moreover, she reported using non-suicidal self-injury for the past 15 years in order to help regulate this intense affect, as well as to convey to others how distressed she was feeling. Eva also reported the daily experience of intense anger toward others, feelings of emptiness and abandonment, and paranoid thoughts that other people were plotting against her at work. The clinician used the next session to conduct a thorough diagnostic assessment of Axis II disorders, which revealed the presence of borderline personality disorder and paranoid personality disorder.*

How might the presence of these comorbid personality disorders introduce complications in the treatment of Eva's SAD? What things should the clinician consider in conducting ongoing assessment in this case? What can the clinician do to manage and/or ameliorate Eva's personality disorders while also providing effective treatment of her Axis I disorders? The purpose of this chapter is to (a) describe the nature of personality disorders, (b) review the extent to which personality disorders are comorbid with anxiety disorders, (c) discuss treatment complications that can arise when comorbid personality disorders are present, and (d) describe ways in which clinicians can resolve these complications.

## **The Nature of Personality Disorders**

A personality disorder is "an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual's culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment" (American Psychiatric Association [APA], 1994, p. 629). The DSM-IV includes 10 personality disorders grouped into three overarching clusters. Cluster A includes paranoid, schizoid, and schizotypal personality disorders – all characterized by odd or eccentric thoughts, feelings, and behaviors. Cluster B includes antisocial, borderline, histrionic, and narcissistic personality disorders – all characterized by dramatic, emotional, or erratic thoughts, feelings, and behaviors. Cluster C includes avoidant, dependent, and obsessive compulsive personality disorders – all characterized by anxious or fearful thoughts, feelings, and behaviors.

Although personality disorders have existed as formal diagnoses since DSM-III (APA, 1980), their rate of occurrence in the community has been unknown until recently. Several recent surveys indicate that personality disorders occur in approximately 9.1–15.7% of adults in the community (Crawford et al., 2005; Lenzenweger, Lane, Laranger, & Kessler, 2007; Samuels et al., 2002). The largest and most representative survey reporting on the prevalence of personality disorders in the United States, the recently completed National Comorbidity Survey – Replication (Kessler & Merikangas, 2004), provides the most recent and representative rates of Cluster A (5.7%), Cluster B (1.5%), and Cluster C (6.0%) disorders (Lenzenweger et al., 2007).

Although all clinicians are quite familiar with personality disorders in general, it is instructive for the current purposes to acknowledge several important issues that can arise when working with clients with personality disorders. First, many clinicians have negative thoughts and feelings about people with personality disorders. Such individuals are often viewed as being hostile, uncooperative, and manipulative (Bowers & Allan, 2006; Potter, 2006). Although empirical work on personality disorders suggests that these are often difficult conditions to treat, it is important to remember that not all clients with a personality disorder share the undesirable qualities listed above. It may be helpful to remember that many of the behavioral patterns displayed by those with personality disorders are the result of both biological predispositions and earlier learning experiences (e.g., Siever, 2005; Ehrensaft, Cohen, & Johnson, 2006).

Perhaps more importantly, the conceptualization about what it means to “have” a personality disorder has changed recently as a result of new research on this topic. There is emerging evidence that personality disorders are not all categorical entities but in some cases represent extreme points on a continuum or dimension of personality traits or characteristics that we all possess (Guay, Ruscio, Knight, & Hare, 2007; Haslam, 2003; Trull & CDurrett, 2005; Westen & Shedler, 1999). Conceptualizing personality disorders in this way has great intuitive and clinical appeal and may help clinicians and the public better understand the experiences and behaviors of those diagnosed with a personality disorder.

Often, clinicians’ preconceived notions about what characterizes people diagnosed with a personality disorder can negatively influence the treatment offered. A common error is to confuse the intention of a client’s behavior with the effect the behavior has on the clinician. Consider the following interaction between a clinician (T) and his supervisor (S) in which the clinician came to supervision clearly frustrated by his client (P).

T: Once again P did not do her homework. In our last session I clearly told her exactly what to do for homework and even worked on getting a commitment, and then did some troubleshooting.

S: OK. What happened when she came in for the next session?

T: What always happens, she didn’t follow through. It’s so clear that she is purposely sabotaging the treatment as a way of just holding on to her relationship with me.

S: Perhaps. Did you speak with her, though, about how difficult the homework might be for her? Is it possible that this is why she has not done her homework?

T: I think it’s a given that the homework is tough. It’s homework. I don’t think we need to belabor that point. She is just being a typical Borderline who doesn’t want to get better.

S: It may be that her presentation is not atypical for someone with BPD. It also sounds, though, like you may be responding like “a typical therapist.”

The supervisor went on to suggest that the clinician might be confusing his understandable frustration as being caused by the client's deliberate lack of follow-through; however, it was clear that an element was missing in the treatment. Instead of placing blame on the client, one must take the view that the treatment must be modified. Specifically, it might be useful for the clinician to accept how emotionally challenging the homework is going to be for the client and offer validation regarding the difficulty level of the assignment.

A second important issue that can arise when working with patients with personality disorders is that although personality disorders are often thought of as life-long or at least long-term conditions, recent research examining the stability of these disorders suggests they are much less persistent than previously assumed. Results from the Collaborative Longitudinal Personality Disorder Study indicate that although personality disorders are generally more persistent than Axis I disorders and maladaptive trait constellations that characterize specific personality disorders tend to persist over time, the majority of clients who meet criteria for a personality disorder at initial assessment fail to meet criteria when followed up 12 or 24 months later (Grilo et al., 2004; Shea et al., 2002).

The fact that personality disorders are not necessarily intractable by nature suggests that their remission may be facilitated, and their probability of re-occurrence reduced, by psychological intervention. Indeed, results from the NCS-R suggest that approximately 39.0% of those with a personality disorder report receiving treatment of some kind in the past year, with the rate much higher for Cluster B (49.1%) than Cluster A (25.0%) or Cluster C (29.0%) (Lenzenweger et al., 2007). What is less clear, however, is how and why people with personality disorders come to present for treatment.

## **Comorbidity of Personality Disorders and Anxiety Disorders**

In our experience it is relatively rare for people to present for treatment with a chief complaint of a personality disorder. Instead, personality disorders are often revealed at the initial assessment or during the course of treatment for some other disorder. Prior research in this area suggests that personality disorders are significantly more likely to be present among those with an Axis I mental disorder of any kind and the odds are highest for those with mood and anxiety disorders. Indeed, those with an anxiety disorder are approximately seven times more likely to have a personality disorder than those without an anxiety disorder; and despite the apparent overlap between anxiety disorders and Cluster C personality disorders, the odds are higher for Cluster B (OR = 8.4) than Clusters C (OR = 4.0) or A (OR = 2.5) (Lenzenweger et al., 2007). Moreover, examination of the odds of having a personality disorder among each type of anxiety disorder reveals that the highest odds of personality disorder are seen among those with SAD (OR = 9.9) and panic disorder (OR = 8.0) (Lenzenweger et al., 2007). While these data are from a nationally representative survey, data from both outpatient and inpatient clinical samples also indicate that

personality disorders are fairly common among those with an anxiety disorder (29–35% of outpatient cases and 89% of inpatient cases), although the highest rates of personality disorders in such settings appear to be for Cluster C rather than Clusters B and A (Sanderson, Wetzler, Beck, & Betz, 1994; Skodol et al., 1995).

It is important to consider the ways in which comorbidity between anxiety disorders and personality disorders may arise; a case conceptualization of the relation between the presenting disorders based on the client's psychological history can help guide assessment and treatment decisions. There are many different ways in which comorbidity may arise in any particular case (Kessler & Price, 1993). It could be that the presence of anxiety disorders during childhood leads to maladaptive behavioral patterns (e.g., avoidance of others, excessive fears or concerns) that interact with contextual factors to produce personality disorders. In contrast, these disorders could share a common etiologic pathway and overlapping symptom presentations. For instance, multiple studies demonstrate significant overlap between SAD and avoidant personality disorder (e.g., McGlashan et al., 2005), both of which are characterized by persistent and impairing social inhibition and fear of negative evaluation, and some have suggested that they are actually different conceptualizations of the same disorder (Johnson, Cohen, Kasen, & Brook, 2006; Ralevski, sanislow, & Grilo 2005).

Another example of comorbid disorders with potentially shared etiology is seen in the overlap of post-traumatic stress disorder (PTSD) and panic disorder with borderline personality disorder (BPD). Prior research has consistently demonstrated strong relations between these two anxiety disorders and BPD (see Lenzenweger et al., 2007; McGlashan et al., 2005; Shea et al., 2004), and each of these disorders has been associated with the occurrence of abuse during childhood (e.g., Latas, Starcevic, Trajkovic, & Bogajevic 2000; Ozkan & Altindag, 2005). It may be that early abuse leads to the increased likelihood of factors such as hyperarousal, numbing, and re-experiencing of earlier traumatic events, with the experience of these symptoms meeting criteria for PTSD; the experience, misinterpretation, and fear of them leading to a diagnosis of panic disorder; and the experience and maladaptive behavioral reaction to them leading to a diagnosis of BPD (e.g., Kaplow, Dodge, Amaya-Jackson, & Saxe, 2005; Lilienfeld & Penna, 2001; Pollack et al., 1996; Yen et al., 2002).

Although the etiology of mental disorders is quite difficult to determine in even the most methodologically rigorous research studies, let alone in uncontrolled clinical settings, the careful assessment and development of a working case conceptualization about the relations between anxiety and personality disorders is strongly advised in treating clients who may display features of both classes of disorders. The following case vignette highlights the importance of having a case formulation that includes an understanding of the ways personality disorders and anxiety disorders may have a synergistic effect on each other:

*Christie presented for treatment with two significant complaints. First, she reported having a number of failed relationships with men that often led to her withdraw for weeks at a time from the expectations of her daily life. Second, she complained that her anxiety was seemingly ever present but worsened when she withdrew. On several occasions her withdrawal led to her losing friendships*

and jobs. A series of analyses of antecedents and consequences of Christie's maladaptive behaviors in relationships elucidated a pattern in which she would quickly become dependent on the men with whom she was with, and then would begin to worry excessively that they would leave her. Her worry often caused her to be overly demanding and needy, and she would often become furious when she felt that the man was pushing her away. Her angry responses to her boyfriends led to the dissolution of the relationship. This ineffective interpersonal style is often characteristic of people with BPD. Christie reported that as she began to get over her failed relationship her anxiety would begin to rise, just when she started to imagine re-engaging in relationships and other social activities. By description, her anxiety had the quality of SAD in which she dreaded making phone calls and applying to jobs and attending social functions. Examinations of her anxiety in the context of her more global behavioral patterns led her to discover several dysfunctional core beliefs, including that she perceived she was doomed to this pattern of relating and that she was defective and weak.

## **Complications Associated with the Presence of a Comorbid Personality Disorder**

*Greater clinical severity.* The first potential complication to consider in treating clients with an anxiety disorder who have a comorbid personality disorder is that the presence of the latter has been associated in some studies with significantly greater clinical severity and chronicity of the anxiety disorder and with great functional impairment (e.g., Ozkan & Altindag, 2005; Skodol et al., 1995). These findings suggest that the identification of a personality disorder among a client with anxiety should signal to the clinician that such a client may have greater impairment than typically seen among anxiety disorders clients, and conversely that the observation of more extreme symptom presentation and impairment in a client with an anxiety disorder should prompt an assessment of personality disorders if not already completed.

On balance, several studies have failed to find a difference in the severity of anxiety disorders between those with and without a personality disorder (e.g., Grant et al., 2005; Sanderson, Wetzler, Beck, & Betz, 1994) and also have reported that much of the impairment observed among those with personality disorders is largely accounted for by the presence of an Axis I disorder. Thus, the data appear to be somewhat mixed on whether those with a comorbid personality disorder will present with greater clinical severity. However, what is perhaps most important to consider is whether a comorbid personality disorder will interfere with or complicate treatment in some way.

*Poorer response to treatment.* A second important consideration is that those with a comorbid personality disorder have been reported to have a poorer response to the treatment of their Axis I disorder. For instance, Mennin and Heimberg (2000) reviewed nine studies of the treatment of panic disorder in which some participants

had a comorbid personality disorder and reported that two-thirds of these studies showed that the presence of a personality disorder was associated with a worse clinical outcome. Similarly, Pollack and colleagues (1996) reported that in the cognitive-behavioral treatment of 100 outpatients with panic disorder, 59% of those without a comorbid personality disorder achieved at least a two-month period of remission, while this was true for only 29% of those with a personality disorder. The relation between a comorbid personality disorder and poorer treatment outcome has been found in other anxiety disorders beyond panic disorder. For instance, Berger and colleagues (2004) found that the presence of a comorbid personality disorder was associated with a poorer and slower response to treatment. More specifically, the likelihood of a favorable response to cognitive group therapy and/or paroxetine was twice as high among those without a comorbid personality disorder. Poor response also has been shown in other studies in the context of SAD and generalized anxiety disorder (e.g., Massion et al., 2002).

Here too, however, other studies suggest the issue is slightly more complex. Some have reported that although clients with a comorbid personality disorder have greater clinical severity to begin with, they experience as much clinical change over the course of treatment as those without a comorbid personality disorder (Dreessen & Arntz, 1998). An important direction for researchers to focus on is determining *why* individuals with an anxiety disorder might have less favorable treatment outcomes in the presence of a comorbid personality disorder. Some research suggests it is not the presence of a specific personality disorder per se, but the presence of maladaptive, avoidant, and paranoid beliefs that may lead to poor outcomes (Kuyken, Kurzer, DeRubeis, Beck, & Brown, 2001). Also, it is common for people with a personality disorder to conceptualize their problems as being indicative of a character flaw or weakness rather than as a psychological problem to be solved. This belief may make people with personality disorders skeptical that psychotherapy can be beneficial. With a better understanding of how and why personality disorders might limit treatment effects, clinicians can aim their treatment at these factors in an attempt to maximize treatment effects.

*Premature termination of treatment.* Another potential complication is that clients with a personality disorder may be more likely than others to terminate treatment prematurely; however, those remaining in treatment may improve just as much as others (Persons, Burns, & Perloff, 1988; Sanderson et al., 1994). The specific reason for premature termination among people with a personality disorder may vary depending on the disorder present. For instance, people with a comorbid avoidant personality disorder may have an excessive fear of criticism and ridicule from others, including their clinician, which may increase the likelihood of treatment termination (Greenberg & Stravynski, 1985). Those with BPD also have shown rates of treatment attrition that are twice as high as those without BPD. This may be due to BPD-related problems such as conflicts that can arise in the client's relationship with the clinician, concerns about abandonment by the clinician, or concerns about treatment being too intense or inflexible (Chiesa, Drahorad, & Longo, 2000). When treating a client with comorbid anxiety and BPD or dependent personality disorder, it is critical that the clinician be alert to issues pertaining to problems with

dependency. Treatments are often ended precipitously either because the client cannot tolerate their dependency on the clinician or because the clinician finds the client too demanding. These are only a few of the ways in which a comorbid personality disorder may lead to premature termination, and this possibility is one the clinician should keep in mind over the course of the intervention.

*Greater likelihood of self-injurious thoughts and behaviors.* The most important potential complication to consider in treating those with a comorbid personality disorder is that the presence of personality disorders is associated with a significantly higher likelihood of self-injurious thoughts and behaviors (SITB; Duberstein & Conwell, 1997; Isometsa et al., 1996; Linehan, Rizvi, Shaw Welch, & Page, 2000). This is true of personality disorders in general and also is true in the context of Axis I anxiety disorders. Anxiety disorders are independently and significantly associated with both suicidal thoughts and suicide attempts (e.g., Kessler, Borges, & Walters, 1999). Further, prior research has shown that among those with an anxiety disorder, the presence of a personality disorder is associated with significantly higher impulsiveness and suicidal thoughts and behaviors (Starevic, Bogojevic, Marinkovic, & Klien, 1999; Zlotnick et al., 2003).

Anxiety disorders and SITB have both been conceptualized as attempts to escape from aversive cognitive and emotional states (Barlow & Craske, 2000; Hawton, Cole, O'Grady, & Osborn, 1982; Nock & Prinstein, 2004, 2005), and the presence of a personality disorder may signal the existence of especially strong emotion reactivity and increased impulsiveness or irritability (depending on the personality disorder in question), which could together significantly increase the likelihood of SITB. In addition, non-suicidal self-injury (NSSI) can have the effect of being a very potent negative reinforcer (e.g., Nock & Prinstein, 2004). It can function as an effective, but maladaptive, short-term solution to the experience of aversive internal states. Consequently, the presence of this behavior may interfere in the between-session practice components of a behavioral intervention.

## **Resolving Treatment Complications**

*Assessment of personality disorders.* The first thing one must consider clinically in attempting to resolve treatment complications related to the presence of a comorbid personality disorder is whether such a disorder is present at all. There are a number of interview-based and self-report measures of personality disorders, some of which also assess non-diagnostic personality traits that may be of interest clinically. Although a comprehensive review of these measures is beyond the scope of this brief chapter, we review some of the most commonly used measures here. Those interested in more detailed information on the assessment of personality disorders should consult sources dedicated to this topic (e.g., Widiger, in press).

Perhaps the most commonly used interview-based measure of personality disorders is the Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II; First, Gibbon, Spitzer, Williams, & Benjamin, 1995). The SCID-II is a 119-item semi-structured interview that assesses the presence of each of the 10 personality

disorders one symptom at a time. It takes approximately a half hour to an hour to administer and with a trained interviewer yields reliable Axis II diagnoses. The strengths of the SCID-II are that it is based directly on the DSM-IV, it is fairly straightforward to use, and it can be easily and flexibly modified for clinical purposes. For instance, if a single personality disorder is detected at pre-treatment, the clinician can re-administer only that module repeatedly over the course of treatment, or at the end of treatment, rather than the entire SCID-II interview. Some things to consider, though, are that the SCID-II requires fairly extensive training to be administered and scored correctly, and this training may not be accessible or desirable for all clinicians.

Other interview-based measures to consider are the Personality Disorder Examination (PDE-R; Loranger, Susman, Oldham, & Russakoff, 1987) and the Structured Interview for DSM-IV Personality Disorders (SIDP-R; Pfohl, Blum, Zimmerman, & Stangl, 1989). The PED-R takes approximately two and a half hours to administer and the SIDP-R takes anywhere from an hour to an hour and a half to administer. Although the PED-R takes a relatively longer period of time, it is especially accurate in deriving dimensional scores (Beck, Freeman, Davis, & Associates 2004). Both interviews have demonstrated reliability; however, training for these interviews is imperative.

Where semi-structured diagnostic interviews of personality disorders are not feasible or are less desirable, diagnostic measures can be administered in a self-report format. Many clinicians and researchers find this approach most practical because training to administer the questionnaire is not required and questionnaires take less time to complete than interviews. Although evidence suggests that self-report measures of personality disorders result in over-diagnosis (Zimmerman, 1994), there is insufficient evidence supporting that one format, interview or self-report, should be preferred over the other. Questionnaires such as the Millon Clinical Multiaxial Inventory (MCMI-III; Millon, Millon, & Davis, 1994) and the Personality Diagnostic Questionnaire-Revised (PDQ-R; Hyler & Reder, 1988) are just a few of the most widely used self-report measures of personality disorders.

Several self-report measures of personality traits also may be of great clinical use, such as the Schedule for Non-adaptive and Adaptive Personality (SNAP; Clark, 1993) and the Dimensional Assessment of Personality Pathology-Basic Questionnaire (DAPP-BQ; Livesley, 1990). Clinician-rated assessments also are available, such as the Shedler-Westen Assessment Procedure (Westen & Shedler, 2007), which are slightly more time-intensive but provide a unique means of quantifying clinicians' experience of clients' normal and pathological personality traits. In addition, measures such as the Personality Belief Questionnaire (PBQ; Beck & Beck, 1991) and the Schema Questionnaire (SQ; Young & Brown, 1994) have been created specifically to assess cognitive dimensions present in specific personality disorders and thus may be particularly useful in the context of CBT (Beck et al., 2004).

*Assessment of self-injurious thoughts and behaviors (SITB).* Another important issue for the clinician to consider is the assessment of SITB among clients with a

comorbid personality disorder. Comprehensive guidelines for assessing suicide risk have been outlined in detail (AACAP, 2001; APA, 2003; Jacobs, 1999), and several recent papers describe the evidence-based assessment of SITB (Nock, Wedig, Janis, & Deliberto, 2008) as well as practical recommendations for conducting risk assessments (Bryan & Rudd, 2006; Joiner, Walker, Rudd, & Jobes, 1999; Kleespies & Dettmer, 2000). Given both the likelihood that clinicians will encounter potentially self-injurious individuals and the limited training in the assessment of SITB currently provided in most training programs (Kleespies et al., 2000), we strongly suggest consulting these sources on suicide assessment. For the current purposes, we will outline some of the key points to consider below.

First, it is essential to carefully assess the presence of SITB, which can include suicide ideation, suicide plans, suicide attempts, and suicide death – all involving an intent to die on the part of the individual. SITB also include non-suicidal SITB – in which intent to die is not present (e.g., repetitive self-cutting, suicide gestures). It is important to distinguish between suicidal and NSSI, as well as between self-injurious thoughts and behaviors, as research has demonstrated that these different constructs have different base rates and correlates (Kessler, Berglund, Borges, Nock, & Wang, 2005; Nock & Kazdin, 2002; Nock & Kessler, 2006), as well as different responsiveness to treatment (G. K. Brown et al., 2005; Linehan, Armstrong, Suarez, Allmon, & Heard, 1991a). In clinical practice, ascertaining the differences between suicidal and non-suicidal behaviors is often a matter of asking the right questions. Clients often know when their self-injurious behavior is in the service of affect regulation (helping them to calm down from painful emotional experiences) and when the behavior is specifically designed to end their lives. A direct inquiry often helps the clinician make this determination, and of course further assessment of the behavior is always in order to confirm this finding.

Second, the assessment of SITB can best inform one's case conceptualization if it goes beyond the measurement of the presence of SITB to assess the factors that may have led to the initiation and maintenance of SITB. SITB are multi-determined behaviors and the range of factors that may influence their occurrence is extremely broad; however, the strongest risk factors are the presence of prior SITB and the presence and accumulation of mental disorders. Beyond these factors, it is often very useful to assess the specific antecedents and consequences of SITB episodes in order to better understand the specific factors or processes influencing their occurrence (e.g., Nock & Prinstein, 2004, 2005). Several reliable and valid measures are freely available that can guide the assessment of the presence of a wide range of SITB, as well as the factors that influence them, such as the Suicide Attempt Self-Injury Interview (SASII; Linehan, Comtois, Brown, Heard, & Wagner, 2006) and the Self-Injurious Thoughts and Behaviors Interview (Nock, Holmberg, Photos, & Michel, 2007). These and a broader range of measures of SITB are described in great detail elsewhere (G. K. Brown, 2000; Goldston, 2000; Nock, Wedig, Janis, & Deliberto, 2008).

Third, the treatment of SITB should be guided by the information obtained from the assessment of the presence and determinants of the SITB. Prior studies have failed to demonstrate that treating mental disorders (e.g., major depressive disorder)

decreases SITB. However, several treatments targeting SITB directly have proven efficacious at decreasing the occurrence of these behaviors (e.g., G. K. Brown et al., 2005; Linehan, Armstrong, Suarez, Allmon, & Heard, 1991b; Linehan, Comtois, Murray et al., 2006). In addition to targeting SITB directly, it is important to continue to assess SITB over the course of treatment in order to measure treatment effects, as well as to manage the risk of SITB over the duration of the intervention.

*Case formulation.* One of the most important steps in resolving treatment complications associated with the presence of a comorbid personality disorder is determining how that personality disorder fits into the clinician's conceptualization of the development and maintenance of the client's mental disorders and functional impairment. Many valuable sources are available that provide great detail about a cognitive therapy conceptualization of personality disorders (e.g., Beck et al., 2004; Young, 1994) and we can only scratch the surface of such issues here. It is assumed that the pervasive and persistent thoughts, feelings, and behaviors that characterize personality disorders are developed through the interaction of genetic/biological predispositions and significant environmental events (see Goodman, New, & Siever, 2004; Siever, 2005). Through these interactions, individuals may learn to process information and view themselves, others, and the world in inaccurate and dysfunctional ways. More specifically from a cognitive perspective, a person can develop core beliefs that influence the way they subsequently attend to, encode, and process information, as well as how they respond behaviorally in different situations. For instance, a person who develops a core belief of "I will be hurt by others" or "Others are harmful and cannot be trusted" may ultimately behave in ways that are consistent with a diagnosis of avoidant or paranoid personality disorder.

Several of the personality disorder measures reviewed above, such as the PBQ and SQ, may be particularly useful for this purpose as they provide information about specific dysfunctional beliefs that could be targeted in treatment. Obtaining information about the beliefs a client holds, and how these beliefs may influence their personality disordered behavior, as well as their Axis I disorder, can enhance clinical understanding of this behavior and will be extremely useful in guiding treatment as well as in providing information about treatment effectiveness. For instance, if a client is avoiding others because she firmly believes all others will ultimately hurt her, this information: 1) may help the clinician understand and more accurately predict this client's behaviour, and 2) provides a specific target for cognitive therapy. Furthermore, the intensity and frequency of such beliefs and thoughts offer a baseline that can be used to measure change over the course of treatment.

*Ordering of target behaviors in treatment.* Regardless of the case conceptualization, it is essential that the first target of treatment be decreasing and ultimately eliminating any SITB that are present. This is the case for both ethical and clinical reasons, as it is in direct opposition to the role of the clinician as a healer to focus on less severe behaviors while a client directly and deliberately engages in self-injury. In addition, if self-injurious behavior serves as a means of decreasing anxiety, the treatment will have little traction if this behavior is not the top priority. If SITB are not present, then the clinician should focus on behaviors that might interfere with treatment such as attendance and adherence, followed by those

that are most impairing or believed to be the primary problem that the client is currently experiencing. Such a structure for organizing treatment targets has been proposed in much greater detail by Linehan (1993) in discussing the treatment of BPD and serves as an excellent model for all treatments where SITB and treatment-interfering behaviors may arise. It is notable that the clinician should actively assess each of these areas and make decisions about the ordering of treatment targets as often times when SITB are present they are not the chief complaint of the client, or even desirable treatment targets.

*Treating personality disorders in the context of treating Axis I disorder.* As in CBT focused on treating any disorder, whether an Axis I disorder, Axis II disorder, or a problem not addressed in the DSM-IV, it is important to identify target behaviors and define them as clearly, specifically, and objectively as possible. In addition, applying what is currently known about the principles of behavior change to modify these targets is essential. The cognitive-behavioral principles, strategies, and practices used to treat Axis II pathology are quite similar to those used to treat Axis I disorders. For instance, after a thorough assessment, the clinician provides information about the personality disorder(s) present, describes a cognitive-behavioral model of the disorder(s), and outlines the proposed treatment plan. In addition, the clinician develops a case conceptualization and shares this with the client in an effort to work collaboratively toward the agreed-upon goals of treatment.

In attempting to change cognitions and behaviors associated with a personality disorder, the clinician uses the same cognitive strategies as in the treatment of Axis I disorders, such as identifying and labeling dysfunctional beliefs, examining the evidence for these beliefs, and generating and evaluating more balanced beliefs. Particularly helpful in the treatment of personality disorders, especially given their pervasive and persistent nature, is the identification of the client's core beliefs related to their personality disorder. As an example, consider an exchange between Eva and her clinician in which they attempt to uncover the core beliefs that may be contributing to her paranoid personality disorder:

P: I did only part of my homework assignment for this week. I attended the pottery class in an attempt to meet other people. . .but I didn't introduce myself to anyone or start-up any conversations, so I guess I completely blew this one.

T: That's great that you went to the class! That is really wonderful! So let's talk a bit about what happened at the class. Did you remember that your goal for this assignment was to introduce yourself to one person in the class?

P: I'm not an idiot. Of course I did.

T: OK, great. So thinking back to when you were actually in the class. . .did you try to introduce yourself to anyone?

P: No.

T: What thought did you have when you remembered that that is what you were planning to do?

P: I thought: 'This class is mostly women. This sucks. I don't want to talk to any of these women.'

- T: OK, I see. And why didn't you want to talk to any of them?  
P: Because I knew they would be mean to me.  
T: What would it mean if they were mean to you?  
P: That they are just like all other women. . .ALL women are mean to me.  
T: And what does it mean if all women are mean to you?  
P: That I can't trust ANY women. . .they are NOT to be trusted!  
T: And what does it mean if no women are to be trusted?  
P: Everyone is trying to hurt me and they always will.

In addition to purely cognitive strategies, the clinician should incorporate behavioral exposures and experiments in which the validity of the client's dysfunctional beliefs can be tested. For instance, drawing from the exchange described above, the clinician might ask Eva to complete a homework assignment for the following week in which she makes predictions about how others are likely to behave if her dysfunctional beliefs are correct (and write these down on her homework sheet or diary card). The client then should test these predictions by following through with her behavioral exposures and recording the actual behaviors of others, ultimately comparing what she observed to what she expected. In this way, the clinician uses the same cognitive and behavioral strategies to simultaneously target the Axis I (SAD) and Axis II (paranoid personality) disorders. While doing this, the clinician should of course be carefully measuring change in all of the target problems over the course of treatment in order to evaluate the effectiveness of the treatment provided. This is especially important given that although there is evidence supporting the cognitive-behavioral treatment of personality disorders, in many cases the use of these techniques to treat comorbid personality disorders is akin to providing "off-label" treatment of the evidence-based approach. The provision of truly evidence-based treatment requires careful measurement of the target behavior in each and every case (Nock, Goldman, Wang, & Albano, 2004).

*Addressing additional complications.* As mentioned above, the presence of comorbid personality disorders may be associated with problems with poorer treatment attendance, adherence, and the therapeutic relationship. Such problems can arise for many different reasons, and the clinician working with clients with comorbid personality disorders should be prepared for this possibility and should attempt to determine the potential source of the difficulties. One way to conceptualize such complications is to consider whether they may be generated primarily by the client, the treatment itself, or the clinician.

Clients may experience treatment as being too demanding or not appropriate for the problem for which they are presenting. Similarly, many clients have low expectations for treatment or are not particularly motivated to participate in treatment. Each of these problems may be especially pronounced among clients with a comorbid personality disorder (Chiesa, Drahorad, & Longo, 2000) and should be assessed directly by the clinician. Measures have been developed specifically for assessing potential barriers to treatment participation that are freely available to clinicians (e.g., Kazdin, Holland, & Crowley, 1997; Kazdin, Holland, Crowley, & Breton, 1997). Moreover, brief interventions have been developed that are designed

to enhance clients' treatment motivation and participation in treatment, many of which are also freely available to clinicians (e.g., Nock & Kazdin, 2005). These assessments and interventions have been designed for quick and easy use (e.g., 5–10 min of assessment and 5–45 min of intervention in total) and we would encourage clinicians working with clients with a comorbid personality disorder to incorporate these into their regular practices.

Treatments for anxiety disorders can be quite distressing (e.g., “You’re afraid of germs? Let’s take a look at this road kill I have in my desk!”), and this may be especially problematic for those with a comorbid personality disorder who may have problems regulating one’s emotional and behavioral experiences. These issues can be infinitely complex; however, we believe the best advice is for the clinician to (a) think carefully about how the client’s personality disorder may influence their experience of the treatment, (b) assess this directly either by raising it with the client or through behavioral observation in sessions, and (c) modify the intervention to account for these difficulties. For instance, we described above how Eva’s symptoms of borderline and paranoid personality disorder interfered with her ability to engage in exposure exercises both in and out of sessions. If left unchecked, these factors could have continued and ultimately led to treatment being persistently ineffective (and unimplemented in the case of her failure to complete homework exposures), to her dropping out of treatment prematurely, or to the clinician becoming confused and frustrated with her ongoing failure to complete treatment assignments.

Clinicians may experience clients with a personality disorder as being hostile, uncooperative, and manipulative, as mentioned earlier (Bowers & Allan, 2006; Potter, 2006). The presence of a comorbid personality disorder clearly can introduce many treatment complications and can arouse negative thoughts and feelings in the clinician, and it is important for the clinician to address these thoughts and feelings using the same strategies that they teach their clients. For instance, Linehan’s (1993) treatment for BPD requires consultation team meetings in which clinicians meet on a weekly basis and among other things provide each other with objective feedback, direction, and support. Similarly, Beck and colleagues (2004) propose using a “Clinician’s Dysfunctional Thought Record” to identify, evaluate, and correct dysfunctional thoughts that can arise about the client, the treatment, or the clinicians themselves. The same practices and techniques that can be used to change clients’ behaviors often work just as well when directed at the clinician.

## Conclusion

As we have discussed, personality disorders often co-occur with anxiety disorders, and their presence can introduce complications including greater clinical severity, poorer attendance and adherence to treatment, increased likelihood of SITB, and potentially worse clinical outcomes. Given the clinical and methodological complexities involved in studying the treatment of anxiety disorders in the context of comorbid personality disorders, clinical research has not, will not, and should not

aim at studying the treatment of the seemingly infinite number of combinations of anxiety and personality disorder constellations. Instead, the clinician should use what is currently known about the evidence-based assessment of anxiety and personality disorders, cognitive-behavioral case conceptualization, and evidence-based treatment (involving careful and continuous assessment of key outcomes) in order to resolve treatment complications associated with the presence of comorbid personality disorders.

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# Resolving Treatment Complications Associated with Comorbid Anxiety and Substance Use Disorders

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The associations between anxiety disorders and substance use disorders (SUDs) have received significant attention in recent years. This is appropriate both because of the growing literature suggesting the presence of comorbidity and possible causal and functional linkages across these disorders, and because this attention is encouraged by clinical demand for treatment options for these patients. Indeed, the comorbidity of these conditions creates a wealth of challenges for practitioners. According to McGovern, Xie, Segal, Siembab, and Drake (2006) “when it comes to providing services to persons with co-occurring disorders, addiction treatment providers may find themselves lost between the vague and the overly particular. This makes research-to-practice translations even more difficult than usual” (p. 267). Accordingly, the goal of this chapter is to identify and discuss complications in the treatment of an anxiety disorder with a comorbid SUD<sup>1</sup>. Following a review of the links between anxiety disorders and SUDs, we report key issues from assessment through treatment including the emergence of integrated treatments that focus on concurrently addressing a comorbid anxiety disorder and SUD. Finally, we provide two illustrative case examples. Throughout this chapter, we acknowledge that the lack of available research on this topic often prevents specificity within either anxiety disorders or SUDs. As such, we will focus more on broad connections across disorders, but also attempt to address specificity where appropriate.

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<sup>1</sup>It is of note that we will limit our review to alcohol and illicit drugs, to the exclusion of cigarette smoking. Clearly, anxiety and cigarette smoking are highly comorbid. Although there is a clear need to understand the role of anxiety in the development of smoking and the exacerbation of anxiety that may result from smoking, the current chapter is focused more on complications that an SUD might cause in the treatment of anxiety beyond merely exacerbating the symptoms.

## Link Between Anxiety Disorders and Substance Use Disorders

Studies consistently find that individuals with an anxiety disorder diagnosis are at heightened risk for a comorbid SUD. Reiger, Rae, Narrow, Kaelber, and Schatzberg (1998), in analyzing cross-sectional data from the Epidemiological Catchment Area (ECA) study, found that the 12-month prevalence of a SUD among individuals with an anxiety disorder was 15%. Although epidemiological studies tend to find higher prevalence rates for alcohol use disorders (alcohol abuse and/or dependence) than for drug use disorders, prevalence rates for both types of disorders are elevated in the context of an anxiety disorder when compared to individuals without an anxiety disorder diagnosis (Conway, Compton, Stinson, & Grant, 2006; Grant et al., 2004). In regard to alcohol use disorders specifically, Grant et al., 2004 in examining the National Institute on Alcohol Abuse and Alcoholism's National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) Wave 1 data obtained from a nationally representative sample of 43,093 individuals, found that among those with any 12-month DSM-IV anxiety disorder, 13.02% had an alcohol use disorder. Panic disorder (PD) with and without agoraphobia was associated with the highest 21-month alcohol use disorder prevalence rates (18.81% and 15.29%, respectively). Broken down by whether or not participants met criteria for alcohol abuse or dependence, 4.96% of individuals with an anxiety disorder diagnosis also met criteria for alcohol abuse in the past year and 8.06% for alcohol dependence in the past year. In examining the self-reported temporal ordering of anxiety and alcohol use disorders within the National Comorbidity Study, Kessler et al. (1997) found that 21.8% of men and 49.7% of women with alcohol dependence reported that their alcohol dependence was secondary to an anxiety disorder diagnosis. Lower rates, especially for men, were found for alcohol abuse (16.6% for men and 39.7% for women). In a review of epidemiologic surveys, family studies, and field studies, Kushner, Sher, and Beitman (1990) found that the relationship between alcohol problems and anxiety appeared to be variable among the anxiety disorders. Specifically, in agoraphobia and social anxiety disorder (SAD), alcohol problems were linked to attempts at self-medication of anxiety symptoms, whereas PD and generalized anxiety disorder (GAD) were found to more commonly occur following pathological alcohol consumption. Further, simple phobia was not found to be related to alcohol in any meaningful way.

Whereas the relationship between alcohol use and anxiety disorders is well-documented (Grant et al., 2004; Kessler et al., 1997; Morris, Stewart, & Ham, 2005), fewer studies have examined prevalence rates of illicit drug use among individuals with anxiety disorders. However, of the studies that have been conducted, all have found significant relationships between the presence of an anxiety disorder and illicit drug use. For example, Merikangas et al. (1998) analyzed data from six international epidemiological study sites (Germany, Mexico, Netherlands, Ontario, and two sites in the United States). They found that, across all sites, approximately 24.9% of individuals with any lifetime anxiety disorder diagnosis also exhibited lifetime drug use (e.g., cannabis, opioids, stimulants, sedatives, or inhalants, but not alcohol), 35.8% exhibited lifetime drug problems (met at least one DSM-III-R abuse criteria for any drug), and 45.8% met criteria for lifetime DSM-III-R

drug dependence. Lopez, Turner, and Saavedra (2005) collected data from 1,747 individuals from the Southeastern United States and examined drug dependence among individuals with pure anxiety disorder diagnoses (i.e., the occurrence of one or more anxiety disorders that is not accompanied by any additional psychiatric disorder) or an anxiety disorder that was comorbid with some other psychiatric disorder (depression, dysthymia, conduct disorder, or attention deficit/hyperactivity disorder). Of the 4.9% with a pure anxiety disorder diagnosis, 13.9% also exhibited drug dependence. Further, more men (20.8%) than women (10.9%) with a pure anxiety disorder diagnosis also met criteria for drug dependence. Among the 10.2% with a comorbid anxiety disorder, 29.2% met criteria for drug dependence. Again, more men (33.3%) than women (26.9%) with a comorbid anxiety disorder diagnosis also met criteria for drug dependence.

In regard to the relationship between specific anxiety disorders and the use of illicit substances, posttraumatic stress disorder (PTSD) has received particular attention (for a review, see Chilcoat & Menard, 2003). Data from the ECA found that compared to men and women without a diagnosis of PTSD, men with PTSD were 5 times more likely to also exhibit a drug use disorder and women with PTSD were 1.4 times as likely to exhibit a drug use disorder (overall odds ratio of 2.2; Helzer, Robins, & McEvoy, 1987). In the NCS, Kessler, Sonnega, Bromet, Hughes, and Nelson (1995) found that compared to men and women without a diagnosis of PTSD, men with PTSD were approximately 2.97 times as likely and women 4.46 times as likely to exhibit a drug use disorder. Giaconia et al. (1995, 2000) collected data from 384 18-year-olds as part of The Early Adulthood Research Project (EARP) and found that compared to individuals without a history of traumatic exposure, individuals with a lifetime diagnosis of PTSD were 8.8 times as likely to also meet criteria for a lifetime drug dependence diagnosis and 14.14 times as likely to meet criteria for past year drug dependence.

The NESARC provides extensive data on illicit drug use across additional anxiety disorders, including PD, SAD, specific phobia, and GAD. Specifically, data from the NESARC indicate that criteria for lifetime drug abuse is met among 14.7% with PD with agoraphobia, 13.2% with PD without agoraphobia, 12.5% with SAD, 11.4% with specific phobia, and 11.9% with GAD. Criteria for lifetime drug dependence is met among 19.5% with PD with agoraphobia, 9.9% with PD without agoraphobia, 9.8% with SAD, 7.3% with specific phobia, and 11.1% with GAD. In regard to the specific types of illicit drugs used, highest rates (across all anxiety disorders examined) were found for comorbid cannabis use disorder. In addition, individuals with a diagnosis of PD with agoraphobia exhibited the highest lifetime rates of sedative, tranquilizer, opioid, amphetamine, hallucinogen, cannabis, and cocaine use disorders, compared to all other anxiety disorders examined (Conway et al., 2006).

## **Recommendations from Assessment through Treatment**

These aforementioned results highlight the need for all individuals in treatment for anxiety to be fully assessed for the presence or absence of a SUD and conversely, individuals receiving substance abuse treatment should be assessed for a range of

psychiatric disorders, including anxiety disorders. This is especially important given that comorbidity has been found to have a negative impact on the course, treatment outcome, and prognosis of both disorders (e.g., Brown, Stout, & Mueller 1999; Charney, Palacios-Boix, Negrete, Dobkin, & Gill, 2005; Hien, Nunes, Levin, & Fraser, 2000; Ouimette, Ahrens, Moos, & Finney, 1998; Ouimette, Moos, & Finney, 2003). Unfortunately, research on and treatment for patients with co-occurring disorders in substance abuse treatment have been slowly evolving (Hunter et al., 2005), and specific guidelines for assessing and treating the co-occurrence of anxiety and SUDs are only recently becoming more available, with much of these guidelines lacking empirical support (Watkins, Hunter, Burnam, Pincus, & Nicholson, 2005). Nevertheless, there are a few key recommendations that can be developed based on theory and the available empirical literature from assessment through treatment.

### ***Assessment Issues***

Given the complex issues related to the treatment of the comorbidity between anxiety disorders and SUDs, it is especially important to accurately diagnose each disorder and develop an understanding of the complex interplay across disorders. Accurate diagnosis and differentiation between substance-induced states and primary affective illnesses is one of the more difficult tasks in assessing patients with co-occurring mood symptoms and substance use. However, it is also extremely important, as early and accurate diagnosis is likely to improve treatment outcomes (Myrick, Culver, Swavely, & Peters, 2004). Key issues to address include (a) the temporal and functional relationship between anxiety and SUDs, (b) specificity across disorder categories, and (c) selection of assessment measures.

### **Temporal and Functional Relationship Between Anxiety Disorders and SUDs**

At times, the complex and bi-directional relationships between anxiety disorders and SUDs can lead to diagnostic uncertainty. It can be extremely difficult to differentiate between substance-induced states and primary psychiatric diagnoses. The majority of the studies on the comorbidity of SUDs and anxiety disorders are cross-sectional in nature, and therefore, it is impossible to determine the temporal progression of the disorders from these studies. In regard to the specific relationship between anxiety and SUDs, three temporal paths are possible. First, the use of specific drugs may increase the risk for the onset of an anxiety disorder. It is thought that chronic and excessive use of some substances may unmask a genetic predisposition to psychiatric illness (Rabinowitz et al., 1998; Volkow, 2006; Zammit, Allebeck, Andreasson, Lundberg, & Lewis, 2002). Second, illicit drug use may follow the development of an anxiety disorder diagnosis, consistent with a self-medication model of illicit drug use (e.g., Preisig, Fenton, Stevens, & Merikangas, 2001; Quitkin, Rifkin, Kaplan, & Klein, 1972; Swendsen et al., 1998, 2000). Thus, the anxiety disorder precedes the SUD, and substances have been used as a coping

mechanism for the anxiety symptoms. In some individuals there is likely to be a cyclic interaction: depressants, such as alcohol and opiates, may be used in an attempt to decrease anxiety, but anxiety may increase during withdrawal states leading to an exacerbation of the anxiety disorder and making relapse to substance use more likely. Finally, there may be an underlying third factor that increases the risk for both anxiety disorders and drug use; that is, there may be a common underlying mechanism for the comorbid development of these disorders (Goodwin et al., 2002).

To further complicate the clinical picture, it is also clear that intoxication and withdrawal from substances of abuse can mimic symptoms of anxiety disorders. For example, alcohol withdrawal may result in symptoms of sweating, tachycardia, and anxiety (APA, 2004), all of which may be viewed or interpreted as panic attack-related symptoms. While the best way to differentiate substance-induced temporary symptoms from psychiatric problems is through observation during a period of abstinence, the duration of abstinence necessary for accurate diagnosis is controversial. Some have suggested postponing the diagnosis of anxiety disorders for 1 month following treatment entry (Schuckit, 1996). This would potentially decrease the number of false-positive diagnoses, but there are drawbacks associated with waiting a month to diagnosis the anxiety disorder; if a person does have an anxiety disorder, the symptoms of anxiety may be contributing to immediate impairment and may be impeding treatment progress. A family history of the particular anxiety disorder, the onset of anxiety symptoms before the onset of substance abuse and dependence, and sustained anxiety symptoms during prolonged periods of abstinence all suggest a primary anxiety disorder (Myrick & Brady, 2003). In addition, primary anxiety disorders may become more severe after cessation of substance use (as the substance use may have been masking or medicating some of the symptoms of the anxiety disorder), whereas substance-induced anxiety symptoms will slowly remit after cessation of substance use (Brown & Schuckit, 1988; Thevos, Roberts, Thomas, & Randall 2000).

### **Specificity Across Disorder Categories**

Given that research on the links between anxiety disorders and SUDs is only recently emerging, it is often difficult to move beyond more general impressions. However, when attempting to create a clear clinical picture of symptoms some special considerations should be taken into account.

*Generalized Anxiety Disorder:* The “free-floating” anxiety characteristic of GAD has considerable overlap with stimulant intoxication and withdrawal from alcohol, sedative/hypnotics, and opiates. Although many substance-abusing individuals report anxiety symptoms consistent with GAD, they may not meet diagnostic criteria for GAD because of difficulty determining the etiology of these symptoms.

*Panic Disorder:* Many substances of abuse (cocaine/marijuana/other stimulants) may actually induce panic attacks or PD during periods of acute intoxication (Aronson & Craig, 1986; Moran, 1986) or withdrawal. Several reports have noted that cocaine can precipitate panic attacks in individuals without previous PD (Aronson & Craig, 1986; Louie, Lannon, & Ketter, 1989; Rosen & Kosten, 1992).

Finally, panic symptoms can be misinterpreted as withdrawal symptoms, which may be especially relevant for those using substances with a more severe withdrawal profile such as opiates or alcohol.

*Social Anxiety Disorder:* A protracted period of abstinence may not be needed to make a diagnosis of SAD, as the fear of interaction in social situations is not a specific feature of substance use or withdrawal. The social fears that occur only during periods of intoxication with marijuana or stimulants, however, should not be considered sufficient to meet diagnostic criteria for SAD, and a reduction in social fears during periods of chronic substance use also should not be taken to imply remission of social anxiety.

*Obsessive Compulsive Disorder (OCD):* Diagnosing OCD in substance abusers is somewhat less problematic than other anxiety disorders because substance use withdrawal and OCD have fewer overlapping features and the characteristic symptoms of OCD are distinctive, although some of the rituals associated with cocaine use may appear related to compulsive symptoms of OCD. In addition, individuals with OCD may find relief in the rituals associated with substance use and therefore not display other compulsive symptoms characteristic of OCD (e.g., hand washing, ordering, counting). There may be some link between substance use and OCD. In a study conducted by Crum and Anthony (1993), subjects actively using cocaine and also marijuana were found to be at increased risk for OCD.

*PTSD:* It is likely that substance use (in particular, cocaine use) and repeated withdrawal (in particular alcohol, sedative hypnotic, and opiate withdrawal) will exacerbate symptoms of PTSD. Withdrawal from opiates, benzodiazepines, and ethanol is associated with central noradrenergic activation, elevation of vital signs, and subjective states similar to those seen during exacerbations of PTSD symptoms. As such, conditioned withdrawal symptoms may exacerbate PTSD symptoms, leading to self-medication as individuals attempt to alleviate symptoms (Brady, 2001; Kosten & Krystal, 1988). For example, cocaine use is associated with paranoia, hypervigilance, sleep disturbance, and autonomic arousal, all of which may increase the severity of hyperarousal symptoms of PTSD (Brady, 2001). Given these issues, it is crucial to understand that although current research and theory rests more so at a more general picture of anxiety disorders and SUDs, assessment always should carefully consider unique issues specific to specific SUDs and anxiety disorders. Further, given more recent attention to these questions, a close eye on new research is of utmost importance.

### **Selection of Assessment Measures**

Because of the high rate of co-occurrence of anxiety and SUDs, screening patients presenting at either substance use or mental health treatment settings is critical. As mentioned above, this is especially important considering that early diagnosis and treatment can improve treatment outcomes. Brief screening tools for SUDs that have been found useful in mental health settings include the Alcohol Use Disorders Identification Test (AUDIT; Bohn, Babor, & Kranzler, 1995), the Michigan Alcohol Screening Test (MAST; Teitelbaum & Carey, 2000), and the Drug Abuse Screening Test (DAST; Cocco & Carey, 1998; Maisto, Carey, Carey,

Gordon, & Gleason, 2000). The Symptom Checklist (SCL-90; Derogatis, 1994), Anxiety Disorders Interview Schedule (ADIS-IV<sup>L</sup>; Di Nardo, Brown, & Barlow, 1994), and Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1997) are widely used instruments for psychiatric screening and diagnoses and can be useful for diagnosis when both anxiety disorders and SUDs are present. The Addiction Severity Inventory is a semi-structured interview designed to address seven potential problem areas in substance-abusing patients: medical status, employment and support, drug use, alcohol use, legal status, family/social status, and psychiatric status (McLellan, Luborsky, O'Brien, & Woody, 1980; Leonhard, Mulvey, Gastfriend, & Shwartz, 2000).

Despite the value of these measures, there has been wide critique. For example, the MAST is not designed specifically to measure complex co-morbid psychiatric states and only includes one question on treatment in a psychiatric hospital or the psychiatric ward of a general hospital due to drinking problems. Moreover, the reason for hospitalization is not obtained. The MAST is really not designed to measure complex psychiatric states. Further, research on the SCID's differentiation of substance-induced and primary psychiatric diagnoses in patients with substance abuse has found little cross-sectional or predictive validity (Kadden, Kranzler, & Rounsaville, 1995; Kranzler, Kadden, Babor, Tennen, & Rounsaville, 1996); similar concerns are evident for the ADIS, despite its specific utility for assessing anxiety disorders. The Addiction Severity Inventory may be useful to obtain information about consequences of substance use when an anxiety disorder diagnosis is made using another instrument or to suggest potential need for further anxiety screening, but the lack of depth in its assessment of anxiety limits its utility for the purposes at hand here. In line with these problems, the Psychiatric Research Interview for Substance and Mental Disorders (PRISM) (Hasin et al., 1996; 2006) was developed to address the lack of a diagnostic interview that is suitable for diagnosing the comorbidity of substance use and psychiatric disorders.

There are three important characteristics of the PRISM that are specific to comorbidity. First the PRISM adds specific rating guidelines throughout the interview, including frequency and duration requirements for symptoms, explicit exclusion criteria, and decision rules for frequent sources of uncertainty such as temporal relationship of psychiatric symptoms and substance use. The PRISM differentiates between substance-induced symptoms, primary symptoms, and symptoms that are "expected effects" of intoxication and withdrawal. Second, the PRISM positions the alcohol and drug sections near the beginning of the interview, before the mental disorder sections, so that the history of alcohol and drug use is available at the time of beginning the assessment of mental disorders. Third, the PRISM adds more structured alcohol and drug histories to provide a context for assessing comorbid psychiatric disorders. In sum, the PRISM does an excellent job of establishing chronological relationships between the psychiatric symptom and substance use for the purpose of diagnostic clarity and treatment planning.

In addition to using measures such as the PRISM which provide excellent information for determining differential diagnosis and temporal specificity, one also may use a functional analytic approach (Haynes & O'Brien, 1990). In this case, two useful questions to begin with are the following: (1) to what extent is the function of

substance use related specifically back to anxiety? and (2) to what extent do anxiety symptoms tie directly back to substance use? One benefit of this approach is that it is based on current functional relationships and therefore may proceed without a clear understanding of temporal sequencing which simply may not be available or may no longer be relevant as the synergistic effects of the two conditions may lead to a different presentation of each as time progresses. Using a functional analytic approach clearly aids in identifying the problem behavior both in terms of anxiety and substance use. Beginning with substance use resulting from anxiety, this might include an examination of the antecedents that precede substance use. In addressing triggers as well as thoughts and feelings preceding substance use, it can be determined to what extent substance use is being used to address anxiety-related issues.

One caveat is that the pattern of substance use can become so automatic that it may be difficult to establish the role of anxiety because avoidance has become so proficient (resulting in the complete avoidance of anxiety symptoms). In this case, it may be useful to assess what might happen if substance use as a means of avoidance was not a viable option in a time when the person wanted to use, examining the resulting exacerbation of anxiety symptoms across the domains of thoughts, feelings, and behavior. Additionally, it is important to consider that the function of substance abuse in the context of an anxiety disorder may change over time; that is, an individual may initially begin to use substances in an attempt to alleviate or escape anxiety disorder-related symptoms (i.e., self-medication or avoidant function). However, as substance dependence develops, the individual's substance use may no longer be in response to anxiety-related symptoms but instead to substance withdrawal symptoms and other psychopathology such as depression resulting from the substance use consequences. In this case, basing treatment purely on the chronological order of development of the disorders could be misleading based on the current inter-relationship of the disorders. Consequently, in doing a functional analysis of the patient's substance use, it is important to examine the past and current antecedents of the individual's substance use behavior in order to determine current function and its relationship to changes in that function over time.

Moving to a focus on substance use as the factor underlying anxiety, a similar approach is taken with a few specifics regarding pharmacological effects to be considered. For example, one may examine the extent to which anxiety symptoms change in response to acute drug administration. Do the symptoms increase as might be the case with a substance that results in CNS arousal such as cocaine or does it decrease as might be the case with a drug associated with a dampening effect such as heroin? Further, do the anxiety symptoms increase in response to more short-term abstinence with a drug such as heroin or alcohol with a fairly severe withdrawal profile? Finally, do the anxiety symptoms improve or worsen as more long-term abstinence is achieved? In asking such questions, a functional analysis will help identify feedback loops that may provide particularly valuable information for understanding the complex interplay between SUDs and anxiety disorders, often with some specific understanding of how this might differ across particular SUDs and anxiety disorders.

## ***Treatment***

Once the co-existing anxiety disorder and SUD are identified, several issues must be considered in terms of implementing the most effective treatment strategy. In this section, we discuss issues to be considered when utilizing state-of-the-art treatments for anxiety disorders and SUDs, with specific attention to the role of (a) willingness to change, (b) combining stand-alone treatments for anxiety disorders and SUDs, and (c) the emergence of specialized integrative treatments. Finally, we close with two hypothetical illustrative case studies.

### **Willingness to Change**

In considering how one might address comorbid SUD for those with an anxiety disorder, it is crucial to first determine the client's willingness to make changes in their SUD. Motivation has been found to predict both dropout and engagement in community-based treatment of substance abuse (De Leon, Hawke, Jainchill, & Melnick, 2000; De Leon & Jainchill, 1986; Simpson, Joe, & Rowan-Szal, 1997) across treatment settings (Joe, Simpson, & Broome, 1998). One theory that attempts to explain the relationship between an individual's intent to change and their subsequent behavior is Prochaska and DiClemente's (1982, 1983) transtheoretical model of change (TTM). In this model, behavior change is conceptualized as a process that unfolds over time and involves progression through a series of five stages of change: precontemplation, contemplation, preparation, action, and maintenance. They argue that at each stage of change, different processes of change optimally produce progress. Thus, matching change processes to the respective stages requires that the therapeutic relationship be matched to the client's stage of change.

According to Prochaska and DiClemente (1983), individuals in the precontemplation stage are the most resistant to change and are characterized as processing less information about their problems, engaging in less personal evaluation, and experiencing fewer emotional reactions to their substance use. Individuals who are aware of their problem and weigh the positive and negative consequences of their actions are in the contemplation stage. Individuals in the preparation stage have made a decision to take action within the next month, while individuals in the action stage are currently taking steps such as changing their behavior, environment, or experiences. Finally, individuals in the maintenance stage have established their recovery and are learning and engaging in behaviors that will prevent relapse.

Information regarding stage of change is important because if level of motivation is identified prior to treatment, an opportunity is provided to (1) select a treatment that is most in line with an individual's current level of motivation or (2) target motivation level prior to treatment if more advanced treatment approaches are utilized. This work has largely been applied to SUDs and is likely most relevant for SUDs here as well, yet value certainly could be gained from its application to anxiety disorders especially when comorbid with SUDs. Of course one may not consider anxiety symptoms such as bodily arousal to be under the control of the individuals in the same way as drinking or injection drug use. However, motivation and

the stages of change can be easily linked to other symptoms such as avoidance as well as willingness to engage in treatment components such as exposure. We now consider potential recommendations across the stages for treating comorbid anxiety disorders and SUDs, again focused primarily on SUDs but with attention to anxiety disorders specifically as relevant.

*Precontemplation and Contemplation.* For individuals for whom their motivation to change a SUD falls in these two stages, it may be useful to focus treatment more directly on anxiety, with the large part of the substance use related components focused on brief motivational strategies. As anxiety treatment is initiated, it may be useful to tie aspects of how sobriety might be especially useful for anxiety-related gains, especially for drugs which may exacerbate anxiety symptoms such as cocaine. Similarly, for individuals more motivated to address their SUD with less willingness to address anxiety problems, treatment might be targeted more toward SUDs at the onset. At this point it would be useful to address the factors underlying the resistance to anxiety treatment. Three fairly obvious possibilities in this regard are a lack of knowledge of what anxiety is and how it relates to one's substance use, stigma regarding other aspects of mental illness beyond SUDs, and/or an unwillingness to engage in typical anxiety disorders treatment such as exposure. Indeed, an understanding of the factors underlying an inability to commit to anxiety treatment could be thoughtfully addressed over the course of several weeks at the onset of SUD treatment including psychoeducation focused on what anxiety disorders are, how they may impact one's ability to get and stay sober, and how treating the anxiety disorder at the same time might actually facilitate sobriety.

*Preparation and Action.* If the individual is in preparation or action for both conditions, a more integrated treatment may be useful given the clear identification of the problems associated with both disorders as well as a commitment to treatment for both. Indeed, preparation and action indicate that the individual has identified the problem (and may very well have identified how substance use affects anxiety, and vice versa). If the individual is in the preparation or action stage for only one disorder and the other disorder is in the precontemplation or contemplation stages, recommendations from the previous section should be considered.

*Maintenance.* If one condition is in the maintenance stage, treatment for the other condition is less likely to be impaired. However, it is also true that if one condition is in maintenance, treatment may focus so exclusively on the other condition that some threats to the condition in maintenance may become evident: for example, a client with SAD who has essentially covered up the anxiety disorder through chronic substance use. In this case, assessment might miss the SAD, but upon successful alcohol treatment, these symptoms may return. If the client and therapist are not vigilant about the SAD and how the addiction treatment might affect it, improvement in one condition could actually increase the likelihood of relapse in the other. Alternatively, consider a client that has struggled to overcome an addiction to opiates including heroin and pain medication. Over time the client may develop PTSD due to a traumatic event and seek treatment. At this point they may not relapse back to substance use but might feel tempted to do so if the first few sessions of exposure therapy are extremely upsetting. Thus one could say that exposure, although

certainly appropriate for the PTSD symptoms, might actually increase craving for opiates. If the therapist is not aware of this substance use issue it might not even be addressed, with the client given social reinforcement for completing assignments, despite an undetected increase in opiate use. As such, awareness of problems associated with substances and anxiety, even if these problems are in remission, may be necessary on the part of both the therapist and client to prevent a relapse of this condition as their other condition begins to improve.

### **Issues to Consider in Combining Stand-Alone Treatments for Anxiety Disorders and SUDs**

Beyond motivation, there are a number of factors to take into account for the treatment of comorbid anxiety and SUDs when using standard treatments. In doing so we consider factors that are specific to both treating only one disorder at a time and treating both disorders simultaneously, as well as collateral benefits of treating one disorder in the prognosis of the other disorder.

*Issues with treating one disorder at a time.* When treating disorders sequentially, treatment of one disorder could be impaired by the symptoms/consequences of a second untreated disorder. For example, in the treatment of PTSD, acute intoxication, withdrawal symptoms, and other short-term consequences of substance use may interfere with exposure. Alternatively, if PTSD is left untreated until substance use issues can be addressed, symptoms of PTSD (e.g., sleep disturbance, intrusive thoughts, hyperarousal) may increase risk for relapse back to substance use (stemming from a desire to escape, avoid, or somehow alleviate these symptoms). Also relevant to untreated anxiety, many traditional substance use treatments including Narcotics Anonymous and Alcoholics Anonymous heavily utilize group formats (Myrick & Brady, 1996) which may be difficult to engage in due to anxiety symptoms across a variety of disorders such as impaired concentration, agitation, and fear of evaluation. Further, these clients may be unduly judged by the staff as not invested in treatment due to their avoidance of group activities.

*Issues with treating both disorders simultaneously.* When treating both disorders together, two factors should be considered. The first factor is the extent to which symptoms from one disorder worsen as a result of treatment for the other disorder. This may include typical anxiety treatments such as exposure producing a greater desire for substances due to initial elevations in anxiety associated with exposure before exposure begins to have its positive effects on anxiety over time. For example, a patient receiving behavior therapy for SAD who also is struggling with alcohol use may experience an increase in alcohol cravings as a result of engaging in staged exposure exercises in-session and homework exercises involving social contact outside of session. To the extent that an individual has used alcohol (or some other substance) to cope with anxiety-related symptoms, a patient instructed to take part in in vivo exposure exercises in and outside of session may begin to experience increased cravings as a result of being placed in an anxiety-provoking situation where all other avoidance efforts are prevented. In this case, particular attention in

treatment may need to be placed on helping the patient understand the function of substance use and increase awareness of situations that may evoke substance cravings. Further, combining strategies for coping with cravings with those for anxiety may assist the patient in fully engaging in exposure exercises. In addition to cases in which actual treatment components may place improvement in a second disorder at risk, there are risks of positive changes associated with treatment for one disorder affecting improvement of the second disorder. Keeping with the SAD and alcohol example, if sobriety is achieved early, the removal of that coping strategy may lead to a strong spike in anxiety symptoms if the alcohol was serving a self-medication role. Again, attention in session to the functional relationship of the disorders cannot be stressed enough.

The second factor is the extent to which treatment for one disorder actually is contraindicated for the treatment of the other disorder. This is most likely going to become an issue in the case of psychopharmacological interventions for anxiety disorders. For example, it would be problematic to prescribe benzodiazepines for a patient with PD who previously used substances to reduce anxiety-related symptoms, as there is an increased risk for abuse and dependence on the medication. Another problem may arise when a patient's substance withdrawal symptoms are so severe that certain anxiety-focused treatment approaches may pose a health risk. For example, a patient experiencing intense physiological withdrawal symptoms (e.g., increased heart rate, shortness of breath) may have their well-being put at risk by engaging in an intense exposure exercise (e.g., flooding). Of course, this speaks to the previously discussed importance of assessing the current state of a patient's withdrawal symptoms before progressing with anxiety-focused treatment, and in some cases, detoxification may first be required.

*Collateral benefits of treating one disorder in the prognosis of the other disorder.* In addition to risks, however, it should be noted that some strengths exist in treating this comorbidity, as treatment for one disorder may have benefits for the other disorder. For example, it is possible that existing empirically supported substance abuse treatments (e.g., relapse prevention) may include active ingredients (e.g., stress reduction coping skills, discussion of negative affect situations) that contribute to the reduction of anxiety symptoms. In addition, learning strategies to self-regulate anxiety symptoms in anxiety disorder-focused treatment may help patients to break out of the mindset of using substances to fight subjective states and obtain alternative coping strategies (Myrick & Brady, 2003).

### **Specialized Integrative Treatments**

Moving beyond the combination of treatments, recent efforts have begun to move toward more explicitly integrative efforts. As Watkins et al. (2005) point out, there are unified treatment programs with cross-trained staff, co-location of mental health and substance abuse services, and integration of services at a broader system level through interorganizational linkages and referrals. In addition to integration at the infrastructure level, several specialized integrative treatments have been developed

to simultaneously address issues relevant to both anxiety disorders and SUDs in one comprehensive treatment approach. Although the use of these approaches requires additional training to staff and have been developed for specific disorders, they provide an extra level of support and guidance in addressing the interplay and functional similarities across the conditions to provide a more comprehensive and targeted treatment approach.

*Seeking Safety.* Given the high rates of comorbidity between anxiety disorders and SUDs, specialized treatments designed to specifically target this comorbidity are beginning to be developed. Of those treatments that are available, the majority are focused on the comorbidity between PTSD and SUDs. Najavits, Weiss, and Liese, (1996) developed *Seeking Safety* to specifically target comorbid PTSD and SUDs. Seeking Safety is a 24-session cognitive behavioral group therapy protocol treatment that teaches individuals with this comorbid symptom presentation a variety of cognitive, behavioral, and interpersonal skills particularly applicable to individuals with both PTSD and substance use difficulties, all of which are designed with the idea that safety is the top priority in recovery from each disorder (Najavits et al.); that is, coping skills are focused on maintaining abstinence, reducing self-destructive and high-risk behavior, and establishing support. *Seeking Safety* has been found to be effective, with patients exhibiting significant reductions in substance use behavior, trauma-related symptoms, suicide risk, suicidal thoughts, depression, and thoughts about substance use, and improvements in social adjustment, family functioning, and problem solving (e.g., Hien, Cohen, Miele, Litt, & Capstick, 2004; Najavits et al.; Zlotnick, Najavits, Rohsenow, & Johnson, 2003).

*Concurrent Treatment of PTSD and Cocaine Dependence.* Another treatment specifically designed for individuals with comorbid PTSD and SUDs is Back, Dansky, Carroll, Foa, and Brady's (2001) Concurrent Treatment of PTSD and Cocaine Dependence (CTPCD). This treatment consists of 16 individual 90-min sessions. The treatment was designed by integrating previously validated cognitive behavioral treatments for substance dependence (Carroll, 1998) and PTSD (Foa & Rothbaum, 1998; Foa, Rothbaum, Riggs, & Murdock, 1991). CTPCD involves psychoeducation on the link between PTSD and cocaine dependence, coping skills training, relapse prevention skills, and cognitive restructuring. Further, patients undergo in-vivo and imaginal exposure in order to address their PTSD symptoms. In an initial examination of CTPCD, Brady, Dansky, Back, Foa, and Carroll (2001) found that individuals who completed treatment evidenced significant reductions in depressive symptoms, PTSD symptoms, and cocaine use severity.

*Anxiety Sensitivity Treatment for Heroin Users.* Targeting anxiety sensitivity (an underlying vulnerability factor that may increase the risk for both anxiety disorders and drug use), as opposed to any specific disorder within a residential drug treatment setting, Tull, Schulzinger, Schmidt, Zvolensky, and Lejuez (2007) recently developed a behavioral treatment (the Anxiety Sensitivity Treatment for Heroin Users; AST-H) meant to be used in conjunction with standard substance abuse treatment. This treatment was designed to have specific relevance for heightened heroin users with heightened anxiety sensitivity. Previous research (Lejuez, Paulson, Daughters,

Bornovalova, & Zvolensky, 2006) has demonstrated that heightened anxiety sensitivity may increase risk for substance use treatment drop-out among heroin users. In particular, a tendency to fear and unwillingness to have anxiety-related sensations may prompt individuals to attempt to avoid these sensations through the use of heroin. Therefore, we developed a six-session adjunctive treatment where individuals engage in interceptive exposure exercises in order to facilitate acceptance of, and tolerance for, aversive internal sensations, with the goal of preventing the use of heroin for self-medication of these sensations. This treatment is currently under development; however, initial pilot evidence suggests reductions in drug craving for heroin and a significant reduction in anxiety sensitivity and panic symptom severity. In an initial examination of this treatment's effectiveness, the AST-H was found to result in reductions in anxiety sensitivity, heroin cravings, avoidance behavior, and emotion dysregulation (Tull et al., 2007). Improvements were maintained when measured over 1 month post-treatment. Current efforts are underway to replicate this finding within a randomized controlled trial.

On a final note, other treatments are available that may be beneficial for comorbid anxiety and SUDs that were not designed to specifically target this comorbidity. For example, Acceptance and Commitment Therapy (ACT) has been found to be successful in treating both anxiety disorders (e.g., Twohig, Hayes, & Masuda, 2006) and SUDs (Heffner, Georg, Parker, Hernandez, & Sperry, 2003), as well as the co-occurrence of anxiety and substance use (Batten & Hayes, 2005). In addition, the utility of Dialectical Behavior Therapy (DBT) in the treatment of substance use (Rosenthal, Lynch, & Linehan, 2005) and anxiety disorders (Gratz, Tull, & Wagner, 2005) has also recently been suggested.

### Case Illustrations

*Client #1: Presenting problem of Obsessive Compulsive Disorder.* Helen was a 28-year-old woman who presented to a psychology clinic with both obsessions and compulsions that had begun about 1 year ago and had become worse over the past 3 months to the point of severe life impairment. She also reported that she had currently begun to use alcohol on a daily basis as a maladaptive attempt to manage these symptoms; she reported no previous history of substance use problems. The ADIS was used to take advantage of its strength in assessing anxiety disorders and the decision was made not to use a more elaborate assessment to determine the link between the substance use and OCD such as a functional analysis or the PRISM because there was little question about the temporal sequencing and function of the alcohol use. The therapist considered beginning treatment focused on only one condition to limit the burdens associated with treatment, but also had concerns about her ability to engage in exposure if she continued regular alcohol use, as well as the continued functional value of her substance use if her OCD went untreated. Because no integrative treatment was available and data indicate no contraindications of treating these conditions simultaneously once withdrawal has been overcome, exposure with response prevention was planned including a component for relapse prevention. However, prior to beginning either treatment component, it

was necessary to determine if detoxification was necessary. Because her likelihood of severe withdrawal symptoms needing medical attention was low, given her consumption level and dependence profile, it was decided that a plan for immediate sobriety (including an emergency plan if more severe withdrawal symptoms were experienced) would be implemented in the initial session following assessment. The extent to which she was able to abstain by the session, treatment would be set to focus on exposure with response prevention for OCD supplemented with more secondary attention to relapse prevention (given its clear functional link to her OCD symptoms). This emphasis would shift if a slip occurred, when high-risk situations were encountered, or if exposure treatment for anxiety increased her urge to drink alcohol. As expected, withdrawal symptoms were minimal and within a few weeks exposure showed some effectiveness in treating her OCD, with a corresponding reduction in alcohol cravings. Helen initially was somewhat unwilling to continue to plan out strategies for high-risk alcohol situations once she was able to achieve sobriety, yet a discussion of how the interaction between alcohol use and OCD symptoms was sufficient to keep her invested in this aspect of treatment.

*Client #2: Presenting problem of heroin dependence.* Mark was a 43-year-old daily heroin user who was court-ordered to a residential drug treatment facility after being arrested for possession of heroin with intent to distribute. Initial assessment with the PRISM indicated that he met diagnostic criteria for heroin dependence and PD. Follow-up screening with the Anxiety Sensitivity Index also confirmed heightened anxiety sensitivity. Of note, when queried about anxiety and panic specifically, Mark indicated that he was not really sure what was meant by anxiety and that he has never heard of the phrase “panic attack” before. However, in providing further details on what anxiety and panic attacks are comprised of, he reported “nerves” being a problem for him and that he has experienced episodes like the described panic attack. When going through his drug history he also noted a long history of substance use starting in his childhood and that his longest period of abstinence was 3 months about 2 years ago. He also reported “nerves” being a problem for him from childhood. He cited problems with withdrawal symptoms as the key issue in returning to heroin use after 3 months of being sober. Further querying indicated that what he considered to be withdrawal symptoms, long after the typical 1–2 weeks for acute withdrawal, were actually panic symptoms. Given that he already was slotted to receive relapse prevention and other treatment targeting his heroin at the residential treatment center, the decision was made to undertake the Anxiety Sensitivity Treatment for Heroin Users (AST-H) to provide a more comprehensive and integrative approach to address his comorbidity. Given his unfamiliarity with anxiety, treatment began with extensive psychoeducation. Mark initially was skeptical about any treatment other than those targeted directly at his heroin use. However, following psychoeducation about the nature of anxiety, he was relieved to learn that the bodily symptoms he continued to experience in abstinence were actually harmless and not continued heroin withdrawal. The psychoeducation made him more willing to approach and experience anxiety as part of the recovery process and to limit the functional need for heroin use. Further, the psychoeducation and exposure exercises reduced the frequency, severity, and believability of negative cognitions

(e.g., beliefs about the harmfulness of anxiety symptoms) linked to his anxiety. He showed some hesitation about exposure immediately into treatment as his anxiety symptoms worsened through his initial abstinence from heroin. Because he was in a residential treatment where the risk for heroin relapse was minimal, a more aggressive exposure regimen was established after a discussion of how any anxiety-related gains actually would support his heroin abstinence attempt. Within a few weeks, Mark had remained abstinent and reported a reduced frequency of panic attacks and a sense of alternative strategies for coping with panic attacks when they did occur.

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# Resolving Treatment Complications Associated with Comorbid Eating Disorders

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## Introduction

Eating disorders (EDs) represent an interesting challenge for the anxiety clinician. ED treatment often is viewed as a distinct specialty; thus, many anxiety clinicians have minimal background in the treatment of EDs. In addition, EDs often are difficult to treat even with extensive experience, and the substantial medical comorbidity and high mortality rate associated with them can make ED patients anxiety-provoking and ethically challenging for providers. Finally, individuals with EDs often are reluctant to disclose or change their ED behaviors. In this chapter we will (a) summarize findings on the co-occurrence of EDs in anxiety patients; (b) discuss how to assess for EDs in anxiety patients; (c) review empirically supported and promising treatments for EDs; (d) describe a case formulation approach for treating anxiety patients with comorbid EDs; (e) provide illustrative case examples using this approach; and (f) explore the issue of ordering of treatments for this population.

## Likelihood of Encountering Anxiety Patients with Co-occurring EDs

Research examining the co-occurrence of EDs and anxiety disorders suggests that anxiety clinicians will encounter patients, particularly female patients, with comorbid EDs. Researchers typically examine ED and anxiety comorbidity either by assessing the frequency of EDs in samples diagnosed with anxiety disorders or by assessing the presence of anxiety disorders in ED populations. Although the former method likely is of greater interest to readers of this book, the majority of studies adopt the latter approach. Thus, we briefly review the literature from both perspectives and then highlight the implications for anxiety clinicians.

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A recent review by Godart, Flament, Perdereau, and Jeammet (2002) found that prevalence rates of at least one anxiety disorder among ED participants ranged from 23% to 75%. As noted by Godart et al. substantial methodological variation makes this literature challenging to interpret. For example, studies vary markedly in population (e.g., inpatient, outpatient, community), types of EDs included (e.g., anorexia nervosa [AN], bulimia nervosa [BN], current, lifetime), range of anxiety disorders included, and diagnostic criteria and assessment instrument used. Despite this, several relatively consistent findings emerge. First, OCD is the anxiety disorder most consistently associated with EDs (Kaye et al., 2004; Wonderlich & Mitchell, 1997), particularly AN. Lifetime rates of OCD among AN patients range from 10% to 66% (Lilenfeld, 2004). Among BN patients, rates range from 3% to 73% (Bulik, 1995).

Social phobia is the next most commonly identified anxiety disorder in ED populations, although the frequent exclusion of PTSD from most studies is a problem (Kaye et al., 2004). Estimates of the lifetime occurrence of social phobia range from 17% to 59% for patients with AN or BN (Lilenfeld, 2004). Fortunately, Kaye et al., in one of the largest studies to date, included lifetime assessment of the full-range of anxiety disorders, which were assessed with the Structured Clinical Interview for DSM-IV. Among the 672 participants, each of whom met lifetime criteria for AN and/or BN, 20% met criteria for social phobia. GAD, panic disorder, PTSD, and specific phobia were diagnosed in 10%, 11%, 13%, and 15% of this non-clinical sample, respectively. Notably, the rate of PTSD in this study was somewhat lower than in other studies. For example, in another large population-based study, Dansky, Brewerton, Kilpatrick, and O'Neil (1997) found current and lifetime PTSD rates of 21% and 37%, respectively, among women diagnosed with BN. Similarly, Striegel-Moore, Garvin, Dohm, and Rosenheck (1999) found that 25% of female VA patients diagnosed with an ED also met criteria for PTSD. It should be noted, however, that Schwalberg, Barlow, Alger, and Howard (1992) found a lifetime PTSD rate of 10% in a clinical BN sample, a rate consistent with that found by Kaye et al.

Of greater interest to anxiety clinicians is ED prevalence in anxiety populations, particularly clinical samples. As noted above, fewer researchers have investigated this, and most studies target OCD populations. These studies indicate that between 11% and 42% of female OCD patients meet criteria for an ED at some point during their lives. Most studies, however, do not assess eating disorder not otherwise specified (EDNOS), the most common ED encountered in clinical practice (Fairburn & Bohn, 2005). EDNOS consists of atypical, clinically significant EDs that do not meet the specific diagnostic criteria of AN or BN (e.g., a patient who purges five times per week but does not meet binge criteria for BN or weight criteria for AN). EDNOS is frequently as severe as AN and BN and should not be viewed as a less significant disorder (Fairburn & Bohn, 2005). In sum, available data may underestimate the actual co-occurrence of clinically significant EDs with OCD. Finally, a recent factor analytic study of OCD found that comorbid EDs were associated with a symptom dimension that is dominated by contamination obsessions and cleaning compulsions (Hasler et al., 2005).

Few studies have examined the presence of EDs in other anxiety populations. Brewerton, Lydiard, Ballenger, and Herzog (1993) calculated that approximately 20% of female social phobia participants in a study by Van Ameringen, Mancini, Styan, and Donnison (1991) met criteria for AN or BN. In a study of adolescent mixed-gender PTSD inpatients, researchers found that 25% met criteria for an ED (Lipschitz, Winegar, Hartnick, Foote, & Southwick, 1999). Similarly, Pratt, Brief, and Orsillo (2005) found that 14% of female veterans with PTSD met criteria for a current ED (i.e., AN, BN, or binge eating disorder [BED]), and 19% for a past ED.

To our knowledge only one study investigated ED comorbidity across a range of anxiety disorders. Becker, DeViva, & Zayfert (2004) examined the rate of EDs in a sample of female patients presenting to an anxiety clinic. EDs (including EDNOS) were diagnosed using a validated self-report measure. Between 10% and 12% of patients who met criteria for panic disorder, OCD, or GAD appeared to have an ED, and 20% and 16% of social phobia and PTSD patients, respectively, met ED criteria. Because this was a highly comorbid sample (i.e., many patients met criteria for multiple anxiety disorders), Becker et al. used hierarchical multiple-regression analyses to investigate the unique contribution of specific anxiety disorders to eating pathology. Both PTSD and social phobia accounted for significant unique variance in eating pathology.

The combined estimated prevalence of AN and BN among young females is between 1.5% and 4% (American Psychiatric Association, 1994). Thus, available data suggest that EDs may be substantially more prevalent in anxiety populations than in the general population. We concur with other researchers (e.g., Brewerton et al., 1993) who argue that anxiety clinicians should be vigilant for comorbid EDs, particularly in female patients. Results from Becker et al. also raise the concern that EDs may go undetected in some anxiety clinics. More specifically, as is common in many anxiety clinics, Becker et al. relied on the Anxiety Disorders Interview Schedule (ADIS-IV; Brown, DiNardo, & Barlow, 1994) as the primary diagnostic instrument. The ADIS is arguably the best structured interview for the assessment of anxiety disorders, and it also assesses many common comorbid Axis I disorders, such as somatoform, mood, and substance disorders. Notably, however, the ADIS does not include assessment of EDs. Because Becker et al. used a self-report instrument to assess EDs, they investigated how many ED cases were missed when the ADIS was used as the primary diagnostic instrument. Results indicated that interviewers missed 80% of probable ED cases, suggesting that clinicians who rely on the leading anxiety disorders interview may miss eating pathology because patients do not necessarily spontaneously volunteer information about eating when it is not assessed.

In summary, research indicates substantial co-occurrence of EDs and anxiety disorders, and female anxiety patients presenting appear to exhibit substantial ED comorbidity. Given the potential medical complications and elevated risk for mortality associated with EDs (Powers & Bannon, 2004), it is imperative that anxiety clinicians do not assume that they will adequately detect comorbid EDs

if they are not explicitly screening for such disorders. Although anxiety clinicians should remain alert for the presence of EDs in all their patients, clinicians should be particularly vigilant when treating women with OCD, social phobia, and PTSD.

## Assessment of EDs in Anxiety Patients

Depending on the time constraints, setting, and clinician preference, a variety of assessment approaches may be used to detect EDs in anxiety populations. One possible strategy that appeals to some clinicians involves simply having patients fill out a valid self-report questionnaire to ascertain their degree of ED symptomatology. There are several such assessments that have been widely used in the ED field. They include the Eating Attitudes Test (EAT; Garner, Olmsted, Bohr, & Garfinkel, 1982), Bulimia Test-Revised (BULIT-R; Thelen, Farmer, Wonderlich, & Smith, 1991), The Bulimic Investigatory Test, Edinburgh (BITE, Henderson & Freeman, 1987), and the SCOFF (Luck et al., 2002). A recent review (Peterson & Mitchell, 2005) of these instruments, however, highlighted some of the shortcomings of these measures including absence of screening for specific ED diagnostic criteria (for EAT, BULIT-R, BITE, and SCOFF), lack of adequate sensitivity and specificity (for the EAT), a high false-positive rate (for the EAT), and/or lack of validation in US samples (for the SCOFF).

Two other questionnaires have been developed that demonstrate an advantage over the measures described above in that they directly assess DSM-IV (American Psychiatric Association, 1994) ED symptoms. The first, the Eating Disorder Examination-Questionnaire (EDE-Q; Fairburn & Beglin, 1994), is the self-report version of the Eating Disorders Examination (EDE; Fairburn & Cooper, 1993). The EDE is a semi-structured interview considered to be the “gold standard” in the assessment of EDs (Garner, 1995). The EDE-Q is a 36-item measure that assesses both behavioral and attitudinal features of EDs over the past 28 days. Items are rated on a seven-point scale in terms of frequency or intensity. In addition to generating a variety of subscale and total scale scores, responses to the specific diagnostic questions can be used to determine ED case status and a “skip-out” condition can be used to reduce patient burden (Becker et al., 2004). The second questionnaire is the Eating Disorder Diagnostic Scale (Stice, Fisher, & Martinez, 2004; Stice, Telch, & Rizvi, 2000) which contains 22 items assessing the DSM-IV diagnostic criteria for AN, BN, and BED. Responses can be used to generate DSM-IV diagnoses or standardized and summed to create a total ED composite score. Although it is well known that questionnaires are not the ideal method for assessing EDs, they do offer clinicians a method for screening ED symptomatology and may be appropriate in some clinical settings. Information obtained via self-report should be followed up with a clinical interview.

In terms of structured clinical interviews, the Eating Disorder Examination (Fairburn & Cooper, 1993) is the most comprehensive ED-specific interview and

## Screening Questions for Detecting Possible Eating Disorders

(Adapted from Fairburn &amp; Cooper, 1993)

1. Over the past 4 weeks have you been consciously trying to restrict what you eat, whether or not you have succeeded? Has this been to influence your shape or weight?
  - a. *If yes ask: How many days in the past 28 days did you consciously restrict what you eat to influence your shape and weight?*
2. Over the past 4 weeks have you gone for periods of 8 or more waking hours without eating anything? Has this been to influence your shape or weight?
  - a. *If yes ask: How many days in the past 28 days did you go for 8 or more waking hours without eating to influence your shape and weight?*
3. I would like to ask you about any episodes of overeating that you may have had over the past 4 weeks. Different people mean different things by overeating. First, I would like you to describe any times when you have felt that you have eaten too much in one sitting. *Get description of a particular episode of overeating; if not a large amount of food ask if the person has had situations where they eat even more and get description).*
  - a. Did you experience a loss of control during these episodes?
  - b. Have there been times when you felt that you have eaten too much, but others might not agree?
    - i. Did you feel or experience a loss of control during these episodes?
  - c. Optional additional probe questions
    - i. Typically, what have you eaten at these times?
    - ii. What were others eating at the time?
  - d. *In this section, identify the frequency of objective bulimic episodes (i.e., objective binges) in which the patient ate a large amount of food and felt out of control; objective overeating episodes in which the patient ate a large amount of food but did not feel out of control; and subjective bulimic episodes in which the patient ate a normal or small amount of food and felt out of control.*
4. Over the past 4 weeks have you made yourself sick as a means of controlling your shape or weight? *Identify frequency of the behavior.*
5. Over the past 4 weeks have you taken laxatives as a means of controlling your shape or weight? *Identify frequency of the behavior.*

**Fig. 1** Screening questions for detecting possible eating disorders (Adapted from Fairburn & Cooper, 1993)

6. Over the past 4 weeks have you taken diuretics as a means of controlling your shape or weight? *Identify frequency of the behavior.*
7. Over the past 4 weeks have you exercised as a means of controlling your weight, altering your shape or amount of fat, or burning off calories? *Goal is to identify extreme exercise designed to control weight and shape. Ask about form, frequency, and duration of exercise.*
8. Over the past 28 days has your shape been important in influencing how you feel about (judge, think, evaluate) yourself as a person? If you imagine the things that influence how you feel about yourself – such as (your performance at work, being a parent, being a student, your marriage, how you get along with other people) and put these things in order of importance, where does your shape fit in.
  - a. *Optional:* If, over the past four weeks, your shape had changed in any way, would this have affected how you feel about yourself? Is it important that your shape NOT change?
9. Over the past 28 days has your weight been important in influencing how you feel about (judge, think, evaluate) yourself as a person? If you imagine the things that influence how you feel about yourself – such as (your performance at work, being a parent, being a student, your marriage, how you get along with other people) and put these things in order of importance, where does your weight fit in.
  - a. *Optional:* If, over the past four weeks, your weight had changed in any way, would this have affected how you feel about yourself? Is it important that your weight NOT change?
10. Over the past 28 days have you felt fat? (*Do not ask this question if patient is obviously overweight*)
11. Over the past 3 months have you been trying to lose weight?
  - a. *If no:* Have you been trying to make sure you do not gain weight?
12. Have you missed any menstrual periods over the past few months
  - a. *If no:* ask about use of oral contraceptives.

**Fig. 1** (continued)

can be used to obtain a thorough assessment of ED pathology. Although the EDE is a very useful tool for in-depth assessment and highly recommended, most anxiety clinicians likely will find it burdensome to administer on a regular basis. Another clinical alternative is to have a repertoire of questions that can be added to a standard anxiety interview, such as the ADIS. Clinically, we find it useful to rely on the wording developed for the EDE. In Fig. 1, we highlight some questions drawn from the EDE that can be used to screen for ED pathology. It is important to note that assessment of overeating can be challenging because overeating patterns vary wildly. Some ED patients may eat small amounts of food (e.g., one cookie) and report strong feelings of loss of control, whereas others consume very large amounts of food during out-of-control eating episodes. Patients also may experience strong feelings of shame when discussing overeating or purging behaviors. Thus, it is important to appear non-judgmental when discussing these behaviors and to be very comfortable discussing behaviors such as vomiting and the consumption of large amounts of food. We encourage clinicians to read through the directions for the EDE, which can conveniently be located in the same book (Fairburn & Wilson, 1993) that contains a copy of the CBT manual for BN, another important resource discussed below.

Because many patients with EDs display no overt signs of an ED, we encourage anxiety clinicians to add screening questions to their regular clinical interview, particularly when working with female patients. We also recognize, however, that many ED patients are evasive about their behaviors, and it can be helpful to be aware of some classic signs of EDs (see Mitchell, 1995 and Goldbloom & Kennedy, 1995 for further discussion; also see case examples below), while recognizing that a lack of these signs does not mean the patient is ED free. For instance, although many ED patients are normal weight, one obvious indicator of a possible ED is weight. A significantly underweight or overweight patient *may* suffer from AN or BED, respectively. Individuals with AN often deny having a problem with eating and may instead argue that they are naturally thin. Another helpful sign in such cases is the presence of lanugo, which is fine body hair that may accompany low weight. Also, AN patients may report significant problems with feeling cold or wear very heavy clothing to keep warm. Other clinical indicators of patients who purge regularly include puffiness in the cheeks, which is a result of enlarged salivary glands, and significant problems with dental enamel erosion (often detected first by a dentist). Although abrasions on the fingers, resulting from stimulating the gag reflex during vomiting, are a commonly mentioned ED sign in the literature, in our clinical experience few patients present with this sign, either because they use an implement such as a toothbrush or are simply able to vomit on demand. In athletes or patients who exercise extensively, stress fractures, repeated injuries, and prolonged recovery periods may indicate the presence of an ED (see the case of Emily below for further discussion). Dizziness also may accompany EDs. Anxiety clinicians typically assess caffeine use because caffeine may increase anxiety; this also is important with ED patients. Individuals with EDs often use caffeine to increase energy and as a diuretic.

In addition to administering any of the measures listed above, clinicians must assess the patient's current height and weight (either by self-report or by weighing at the clinic). This is important not only for the diagnosis of AN but also to get a sense of whether the patient's body mass index (BMI) falls within the underweight, normal, or overweight ranges. Although self-report height and weight are not ideal, research has shown that self-report weights are reasonably accurate (Stunkard & Albaum, 1981).

Finally, any ED patient should have a complete medical examination, preferably conducted by a physician who is experienced in treating ED patients, although these physicians can be hard to find. This typically includes a general medical exam, blood tests to detect any electrolyte abnormalities, and assessment of the various physical consequences of the ED (e.g., bradycardia, low bone mass). This information is important in guiding decisions about when to address the ED. Although it is beyond the scope of this chapter to review all of the medical complications associated with EDs (e.g., cardiac, endocrine, gastrointestinal, and/or renal complications), clinicians should bear in mind that EDs can affect all organ systems and lead to permanent changes if not death. Many anxiety clinicians may not have adequate familiarity with health risks associated with EDs and the critical importance of multi-disciplinary care for ED patients. Mental health clinicians should always involve a medical practitioner when treating a patient with an ED. We refer clinicians to Powers & Bannon (2004) for a more comprehensive review of medical comorbidity associated with EDs.

## **Overview of Empirically Supported and Promising Treatments for EDs**

As is the case with anxiety disorders, varying amounts of empirical support exist for specific ED treatments. For example, whereas CBT for BN (CBT-BN) is supported by extensive research (Wilson, 2005), treatment for AN has garnered less support, in large part due to a shortage in the number of controlled trials evaluating specific interventions. Complicating the picture is the fact that EDNOS is excluded from most trials. As noted above, EDNOS is more prevalent in clinical practice than AN and BN; it also has much in common with these disorders (Fairburn & Bohn, 2005). Because CBT-BN has been extensively researched and includes many of the techniques commonly employed in ED treatment, we first discuss CBT-BN and the model that underpins it. We then briefly discuss a new intervention, CBT-Enhanced (CBT-E) and its associated model because this model builds on the CBT-BN model and is very useful in developing case formulations for comorbid anxiety and ED patients. Also, preliminary reports of an ongoing trial of CBT-E have been quite promising (Fairburn, 2004). Next, we highlight some cognitive-behavioral strategies that may be of particular use for anxiety clinicians who encounter patients with comorbid EDs, and discuss motivational issues associated with comorbid

EDs. Finally, we briefly review the research supporting several other treatments for EDs.

### ***CBT-BN and CBT-E***

According to recent meta-analyses (Whittal, Agras, & Gould, 1999) and reviews (Wilson, 2005), the treatment for BN with the most empirical support is manual-based CBT-BN (Fairburn, Marcus, & Wilson, 1993). In fact, after conducting one of the most comprehensive reviews of the literature, the National Institute for Clinical Excellence (NICE), an independent organization charged with developing evidence-based guidelines on healthcare in the United Kingdom, recently recommended that CBT be the first-line treatment for adults with BN (Wilson, 2005)

CBT-BN is based on a cognitive model of BN (Fairburn, Marcus, et al., 1993), which proposes that over-concern with shape and weight drives extreme and rigid dietary restriction, which in turn leads to binge eating. Binge eating results secondary to inadequate caloric intake, and to inevitable violation of strict eating rules (e.g., “now that I’ve blown it by eating this cookie, I might as well binge”). Compensatory behaviors, such as vomiting, start as attempts to counteract episodes of overeating, but soon lead to an increase in overeating, in large part because cognitive restraint is reduced when individuals believe they can “get rid of” excess calories. Moreover, some compensatory behaviors, such as vomiting, are easier to initiate when one has consumed a large amount of food (i.e., it often is easier to vomit a gallon of ice cream than two spoonfuls). Binge eating also increases over-concern with shape and weight and fears of gaining weight. Thus, once the disorder has started, it becomes self-perpetuating.

CBT-BN typically consists of 15–20 sessions over approximately 5 months with the initial phase of treatment focused on helping patients institute a regular eating pattern of consistent meals and snacks, learn about the consequences of their behaviors (e.g., extreme dieting, vomiting, and laxative and diuretic misuse), and employ alternatives to bingeing and purging when in high-risk situations (Fairburn, Marcus, et al., 1993). In the subsequent phases of treatment, patients are encouraged to resume consumption of forbidden foods. They also learn to decrease their weight and shape concerns (core ED cognitions), and prevent relapse of future episodes of their ED. Between 40% and 50% of treatment completers cease binge eating and purging (Fairburn et al., 1995). The CBT-BN manual is extremely helpful for any clinician wanting to gain knowledge about the treatment of EDs. As noted above, it is conveniently published in the same book as the EDE (Fairburn & Wilson, 1993).

Despite its efficacy, manual-based CBT does not help all patients with BN. In an effort to maximize treatment outcomes for patients receiving CBT-BN and to better treat the full range of EDs, including EDNOS, Fairburn, Cooper, and Shafran (2003) built on the model described above and proposed a new transdiagnostic model of the maintenance of all EDs that includes four additional maintaining processes that

affect the core psychopathology of EDs. These include clinical perfectionism, core low self-esteem, mood intolerance, and interpersonal difficulties. The transdiagnostic model offers greater flexibility in creating a case formulation for complicated patients, and highlights many factors (e.g., perfectionism, high achievement standards, mood intolerance/avoidance) that are relevant in conceptualizing anxiety disorders. A more prominent role also is given to the well-known observation that many ED patients use binge eating and or purging behaviors to regulate mood (including anxiety).

In addition to describing a new model of EDs, Fairburn et al. (2003) described a new transdiagnostic treatment for EDs. Importantly, this treatment can be used with all forms of clinical EDs that can be managed on an outpatient basis. A patient's specific ED diagnosis is not as important as the idiographic model of maintaining psychopathological features and processes. The four stages of treatment include an intensive initial stage of twice-weekly sessions for 4 weeks which focuses on engagement of the patient, education about EDs, creation of a personalized formulation of their ED, and early behavioral change (Fairburn et al.). Stage two, which lasts 1–3 sessions, includes a review of progress, identification of any barriers to change, and revision of the formulation to include the most relevant of the four additional maintaining factors described above. In the third and most lengthy stage, the revised formulation guides the interventions such that a focus remains on the patient's ED pathology along with the additional processes that have been identified. Fairburn et al. provide treatment "modules" to be used in each case. As with the CBT-BN manual, we find the article describing the transdiagnostic model and treatment (Fairburn et al.; see also Fairburn, 2008) to be very useful in both conceptualizing and treating comorbid anxiety disorder and ED patients.

### ***Commonly Used CBT Strategies for Treating EDs and General Treatment Issues***

Both CBT-BN and CBT-E share many features, not surprisingly given that CBT-E was designed to build upon the success of CBT-BN. More specifically, both treatments are anchored by the traditional CBT reliance on self-monitoring. Patients in ED treatment self-monitor all episodes of food consumption (including exactly what was eaten) and all compensatory behaviors (e.g., vomiting, laxative use, excessive exercise), along with circumstances that surround the episodes (time, location, other related factors). Many ED patients find self-monitoring quite aversive; thus, it is imperative that clinicians educate themselves about how to encourage, respond to, and reinforce self-monitoring in ED patients. More specifically, ED patients need a very clear rationale for self-monitoring along with an explanation of how the monitoring will be used to help them. When they complete assigned monitoring, they need to be reinforced and shaped so that the monitoring continues and becomes optimally useful. In addition, it is critical to avoid appearing judgmental about the quantity or types of foods consumed. ED diets can be quite unusual and patients

are very sensitive to perceived judgment (see CBT-BN manual for more detail about assigning self-monitoring).

In our experience, getting ED patients to regularly and reliably self-monitor is somewhat more challenging than getting anxiety patients to self-monitor. Thus, although it can be tempting to simply assign all patients with comorbid EDs self-monitoring of their eating even when the primary disorder being treated is the anxiety disorder, we recommend caution in adopting this approach. If clinicians do not have a clear clinical plan to use the monitoring then patients often rapidly find self-monitoring unrewarding and they may be more reluctant to complete similar assignments in the future. We, thus, recommend using detailed self-monitoring of eating in the following circumstances. First, and obviously, self-monitoring will be implemented if the clinician decides to start CBT-BN or CBT-E. Second, in some cases it may make sense to briefly use self-monitoring to better assess current patterns of eating. Finally, at times, self-monitoring of eating can be used to “keep an eye” on the ED while completing treatment of the anxiety disorder. If self-monitoring is to be used in this third situation, the clinician should keep two factors in mind. First, if the primary objective is simply to track the frequency of binge eating and purging, many patients find it easier simply to keep a tally of these behaviors as opposed to completing detailed food monitoring. Second, if the patient is going to be asked to complete detailed food records, then some portion of each session should be spent carefully reviewing and using these records so that the patient is reinforced for completing this task.

Other common strategies employed in CBT include psychoeducation (e.g., about dietary restriction, purging behaviors), weekly weighing (e.g., to teach patients to view weight as useful data and to reduce over- or under-weighing), stimulus control strategies (e.g., to facilitate consumption of regular meals and to decrease binge eating), cognitive restructuring (e.g., to address maladaptive cognitions about the importance of weight and shape), and relapse prevention (e.g., to identify situations in which the patient might be at risk to restrict, overeat, or purge). It is beyond the scope of this chapter to review the ways in which each of these techniques are used to treat EDs; we refer readers to the original materials described above for additional important detail. It is important to note, however, that ED patients are frequently less motivated for treatment than anxiety disorder patients and that standard CBT techniques for increasing motivation may be useful in this population. More specifically, many ED patients view their ED as a solution, not a problem, and this may be particularly the case for individuals with EDs who present for treatment for another disorder, such as an anxiety disorder. In fact, as demonstrated in the case studies described below, many anxiety patients with a comorbid ED may come to treatment never intending to discuss their ED with their anxiety therapist. Thus, in addition to assessing EDs, anxiety clinicians need to be prepared to address motivational issues with respect to the ED, even if the plan is to refer patients for specialty ED treatment. Although not a part of the formal CBT-BN manual, we find decision analysis a very useful CBT strategy for increasing motivation for ED treatment. Decision analysis (Janis & Mann, 1977) is a strategy developed to enhance motivation in substance abusers, and it can be helpful in increasing motivation for

ED treatment. In decision analysis, patients explore both the positive and negative consequences of continuing their behaviors and stopping their behaviors, both in the short and long term. We also refer clinicians to Vitousek, Watson and Wilson (1998) for an excellent discussion of other strategies for increasing motivation for ED treatment.

### ***Other ED Treatments with Empirical Support***

Due to the dearth of controlled trials for the treatment of AN (attributable to the low incidence of AN), there is no current treatment of choice for this potentially lethal disorder. Several treatments appear promising, however, including manualized family-based treatment (FBT) for adolescents (Lock, Le Grange, & Agras, 2001) and CBT-E (Fairburn et al., 2003; Fairburn, 2008). FBT, also known as the Maudsley Approach, has garnered some empirical support and further trials are currently underway (le Grange & Lock, 2005; Wilson, 2005). Although there is debate about the strength of the current evidence for FBT (Fairburn, 2005), it remains the case that FBT is commonly recommended and utilized with this population (le Grange & Lock, 2005). As noted above, CBT-E (Fairburn et al.; Fairburn, 2008) targets all EDs. For example, it includes a focus on several clinical features distinctive to AN, including fear of weight gain and perfectionism. Final results from the ongoing trial of this treatment are eagerly anticipated by the ED community.

It is important to note that the medical issues associated with AN (e.g., refeeding syndrome) raise a number of ethical concerns for ED novice providers. In general, we recommend referring severely underweight patients for ED specialty care. At minimum, care of such patients requires supervision by an ED specialist and collaborative nutritional and medical care. As noted by Vitousek et al. (1998), ED treatment generally requires clinicians to obtain extensive knowledge about a wide range of topics including, but not limited to, nutrition, effects of dietary restriction, exercise, factors contributing to weight including metabolic factors, and recent diet fads so as to help patients disentangle myth from fact. We recognize that many anxiety clinicians may not want to devote significant time to obtaining this knowledge; thus, a good network of ED specialists for referral can be one of the most important tools in working with comorbid anxiety and ED patients. The Academy of Eating Disorders ([www.aedweb.org](http://www.aedweb.org)) can be a useful source for finding ED providers.

The treatment for BED (the only well-categorized EDNOS to date) with the most empirical support is CBT-BED (Wilson, 2005). However, in contrast to CBT-BN, CBT-BED has not been shown to be superior to other forms of psychosocial treatments (Wilson & Fairburn, 2002). Interestingly, CBT and interpersonal psychotherapy (IPT) for BED have shown almost equivalent outcomes in both the short- and long-term (Wilfley et al., 2002; Wilfley et al., 1993). In addition, dialectical behavior therapy (DBT) has been used successfully to treat BED (Telch, Agras, & Linehan, 2000). Research on the treatment of BED is less extensive than that of BN (Wilson, 2005) and several projects are underway in an effort to add to this area of knowledge.

## **Using the Case Formulation Approach to Guide Treatment Planning for Anxiety Patients with Comorbid EDs**

No empirically tested treatment manual is available to address any combination of comorbid anxiety and EDs or the complicating life problems of these patients. Therefore, treatment planning for such patients can be exceedingly challenging for clinicians who want to deliver evidence-based treatment. We have found the evidence-based case formulation approach described by Persons and her colleagues (Persons, 2005; Persons & Tompkins, 2007) to be a powerful method for addressing comorbid presentations and resolving complications and obstacles as they emerge in treatment (see Zayfert & Becker, 2007) for more specific discussion. This approach helps clinicians organize an array of clinical information in a theoretically coherent manner, resulting in a case formulation that serves as a guide for systematic selection and ordering of interventions.

When using the evidence-based case formulation approach, the clinician starts by identifying predisposing, precipitating, and maintaining factors. Next, the clinician generates hypotheses about the interrelationships among the patient's multiple problems. To the degree possible, hypotheses are based on evidence-based nomothetic models of specific disorders and associated problems, so as to reduce clinical judgment errors (Wilson, 1996). By considering a range of evidence-based nomothetic models within a hypothesis-testing approach to the individual case, the clinician can, in effect, apply a scientific method to the treatment of complicated patients, maximizing the likelihood of a positive outcome. Based on the hypotheses, the clinician then selects treatment strategies, drawing as much as possible from treatments with substantial empirical support. Finally, the clinician assesses response on a regular, ongoing basis to determine if the patient is responding as predicted by the hypotheses. If the patient is not responding as predicted, the clinician revisits the hypotheses.

Persons & Tompkins (2007) outline seven steps for constructing a case formulation (see Fig. 2). Following these steps, the clinician aims to construct a formulation that is evidence-based, has clinical utility, and is reasonably parsimonious.

### **OCD Case Example**

#### ***Case Description***

Emily, a 17-year-old student, was referred by her high school counselor for treatment of OCD. Emily reported an extensive history of contamination obsessions, washing and checking compulsions, and avoidance behaviors. Emily feared being contaminated by "AIDS" and cancer, and she recently had been unable to handle her textbooks for fear that someone with AIDS or cancer might have previously handled the books. Emily reported that her contamination fears dated back to childhood, when she engaged in elaborate washing rituals when she thought she had been

given “cooties” by another child. Emily tried to make her thoughts “go away” but was unable to banish them. She currently engaged in extensive showering and hand-washing rituals; the skin on her hands and arms were noticeably red and chapped. In addition, Emily also reported “sterilizing” her food to prevent it from contaminating her. This compulsion began at age 15 when she learned of an *Escherichia coli* outbreak and became highly concerned that food could contaminate her. Emily reported that she only ate foods cooked at very high temperatures for significant periods of time to insure that the food was clean. Emily realized that her concern was irrational in that she was willing to feed her younger sister, who she loved very much, food that had not been sterilized. At intake, Emily reported that her food intake was “somewhat limited” by her contamination fears, but stated that it was more important to overcome some of her other contamination fears first because they impaired her academically.

Emily was a healthy looking, thin young woman (i.e., BMI of 18.5) who enjoyed being on the varsity cross-country team. She noted that she had played soccer as a child, but had given it up secondary to feeling contaminated by her contact with the other players on the field. She also reported significant difficulty showering in public places, and had special showering rituals to wash off the germs from the showers in athletic facilities.

Noting Emily’s thin appearance, her participation in weight-oriented sport, and some evasiveness about her actual diet, Emily’s therapist probed further into Emily’s eating. Emily stated that her eating was the “least of my worries” but ultimately began to disclose further. She noted that after she began sanitizing food, she also started to reduce the number of foods that she ate because some foods were harder to sanitize. For example, she eliminated dairy foods from her diet because sanitizing

#### Steps in Constructing a Case Formulation (Persons & Tompkins, 2007)

1. Obtain a comprehensive Problems List
2. Assign a five-axis DSM diagnosis
3. Select an anchoring diagnosis
4. Select a nomothetic formulation of the anchoring diagnosis to use as a template for the hypothesized psychological mechanisms part of the formulation
5. Individualize the template so that the formulation accounts for the details of the case at hand and for all of the problems on the Problem List and their relationships
6. Propose hypotheses about the origins of the psychological mechanisms
7. Describe the precipitants of the current episode of illness or symptom exacerbation

**Fig. 2** Steps in constructing a case formulation (Persons & Tompkins, 2007)

burned these foods to the point that they were unappetizing. Emily also reported that after developing the food obsession she lost weight, which pleased her from both an appearance perspective and because of positive feedback from her coach that her running was improving due to her weight loss. At intake, Emily limited her diet to two kinds of canned, low-fat soup (chicken noodle and tomato) that she boiled for a minimum of 45 min to sanitize. When asked why the soup had to be low fat, Emily reluctantly admitted that she also was concerned about gaining weight. Further questioning revealed that Emily avoided some foods due to contamination issues, though she avoided other foods due to weight and shape concerns. Questioning also revealed extensive misuse of diuretics and the consumption of large amounts of (highly boiled) coffee for energy and diuretic properties. Emily described her weight as one of the most important things in how she judged herself, even though she felt superficial for considering it important. Her menstrual cycle was highly irregular.

### ***Case Formulation***

#### *1. Problem List:*

- Contamination obsessions
- Checking and cleaning (including sterilizing) rituals which interfere with studying
- Avoidance (of particular foods and of touching objects she believes contaminated) interfering with ability to function (e.g., academically)
- Over-concern with weight and shape
- Malnutrition secondary to inadequate intake: related to both obsessions and over-concern with weight and shape
- Repeated problems with stress fractures during running; possibly related to osteoporosis and irregular menses
- Possible electrolyte imbalance and cardiac abnormalities
- Misuse of diuretics (including caffeine)
- Episodes of dizziness, which are interpreted to indicate possible contraction of disease or contamination by inadequately sterilized food
- Overtraining: related to weight/shape concerns and perfectionist standards about needing to be the best runner on the team

#### *2. Diagnoses:*

- Axis I: OCD, EDNOS<sup>1</sup>
- Axis II: None
- Axis III: Malnutrition, R/O osteoporosis, R/O electrolyte imbalance, R/O cardiac abnormalities

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<sup>1</sup>Although underweight, Emily did not meet the weight criteria for AN. She also did not meet criteria for BN because she did not engage in binge eating.

- Axis IV: Interpersonal problems with teachers secondary to attending classes poorly prepared
- Axis V: 50

3. *Anchoring Diagnosis: OCD*

4. *Relevant Nomothetic Formulations*

- Cognitive-behavioral models of OCD (see Steketee & Barlow, 2002)
- Transdiagnostic CBT model of EDs (Fairburn et al., 2003; Fairburn, 2008)

5. *Individualize the Template –*

*Biological/Somatic factors:* Weight loss, dizziness, and weakness due to malnutrition

*Behavioral factors:* Avoidance: Excessive washing rituals, sterilizing foods, avoiding touching “contaminated objects,” avoiding particular foods

*Cognitive factors:* “Food can be dangerous – it can make me sick.” “I need to be very careful about what I eat or else I will get sick or fat.” “I’m only worthwhile when I’m thin. “Being thin is *the* most important thing in life.” “Being thin makes me a better athlete.”

6. *Working Hypotheses –*

The therapist hypothesized that, as a result of both biological and psychological vulnerabilities, Emily viewed her obsessions as dangerous thoughts to be avoided, and also overestimated the likelihood of danger from contamination. In response, she attempted to suppress the thoughts, which led to them occurring more frequently. In addition, she attempted to neutralize her thoughts by engaging in cleaning and checking rituals and by avoiding stimuli that triggered the thoughts in the first place. Cleaning and checking initially reduced Emily’s anxiety; thus, these behaviors became negatively reinforced and increased.

The therapist further hypothesized that a variety of life factors led Emily to become over-concerned with weight and shape as a means of improving how she felt about herself. These factors included a father who teased Emily if she gained weight, learning to overvalue the importance of achieving (i.e., perfectionism), and core low self-esteem. When Emily lost weight, many people reinforced her weight loss, including her coach and her father, who both actively praised her for becoming thinner. Emily reported that, for the first time, these two important people regularly told her she was doing well. Emily also reported that she enjoyed the weight loss because she looked better (i.e., was closer to both the thin-ideal standard of female beauty and the cross-country runner’s thin-ideal standard of appearance). In order to maintain and even increase her weight loss, Emily engaged in more drastic restriction. She began using diuretics after her boyfriend, who wrestled, mentioned using them to make weight. Diuretic misuse can result in hypokalemia (low serum potassium). Thus, the therapist hypothesized that Emily’s dizzy spells might be related to both general malnutrition and hypokalemia, which put Emily at risk for cardiac problems. Low protein intake also raised risk of cardiac abnormalities. Emily’s dizzy spells served to increase her anxiety in that she interpreted dizziness as a sign of illness, due

either to contamination from touching an object or inadequate sterilizing of food. The therapist also hypothesized that the female athlete triad (Nattiv, Agostini, & Yeager, 1994), which consists of disordered eating, irregular or cessation of menses, and osteoporosis, was a potential problem for Emily. The female athlete triad recognizes that many female athletes (even those without clinical EDs) do not consume diets adequate to maintain regular menses given the excess calorie expenditure associated with competitive sports. Regular menses are needed for bone health; Emily's stress fractures suggested that she might be experiencing early (i.e., osteopenia), or not so early, stages of osteoporosis. Because most people cannot build bone mass after age 25, determining Emily's bone status was important.

Finally, the therapist hypothesized that Emily's reluctance to discuss her eating indicated that, like many individuals with EDs, Emily was not motivated to address her ED. Thus, as long as medical issues were addressed, Emily might respond better to an approach that first targeted her OCD. After Emily experienced progress in this area, and after she and her therapist had built a more solid therapeutic alliance, Emily might be more willing to tackle her ED behavior.

7. *Precipitants of current episode:* *E. coli* outbreak triggered food sanitizing; weight loss triggered further dieting and diuretic use.

### ***Treatment Plan***

After reviewing the case formulation hypotheses, Emily and her therapist agreed to start relatively straightforward exposure and response prevention (ERP) for Emily's OCD because Emily was motivated for OCD treatment and it appeared that she could make progress faster in this area. Emily and her therapist also agreed that it would be difficult to proceed with ED treatment as long as she felt compelled to sanitize her food. In contrast, the ED would not necessarily interfere with exposure to unsanitized foods, as long as they were relatively low calorie. Because of the medical concerns associated with Emily's ED, the therapist made treatment contingent upon Emily meeting with a physician; explaining her nutritional status, diuretic use, and exercise level; having a complete physical (including cardiac) assessment; having her electrolyte levels checked; arranging for a bone scan; and agreeing to allow contact between physician and therapist. Emily also agreed that she would take supplements (e.g., potassium and/or calcium) if recommended by her physician. The therapist explained that exposure could be used to help Emily consume the supplements if contamination fears arose, as predicted by Emily. In addition, Emily agreed to decrease caffeine use (in consultation with the physician), which often exceeded a pot of coffee per day and likely exacerbated her anxiety, and to attempt to decrease diuretic use. Finally, Emily agreed to emphasize nutritious foods on her exposure hierarchy. For example, Emily had no calorie concerns about adding spinach, bananas, or skim milk to her diet, but had contamination fears about these foods. After 15 sessions of twice-weekly ERP with ongoing assessment, Emily and

her therapist would review her progress and reassess her ED. At that time, if indicated, Emily would either start CBT for her ED or begin decision analysis aimed at increasing her motivation to engage in ED treatment. ERP consisted of exposure to a variety of objects (e.g., books, tables at school, unsanitized food) and prevention of compulsions (e.g., washing after contact with books, extensive showering). With regard to food exposure, Emily agreed to bring a designated unsanitized food to therapy, and she and the therapist would eat the food together, with Emily attending to her anxiety for the remainder of the session. Emily then continued with home practice of exposure.

## **Social Phobia Case Example**

### ***Case Description***

Patricia was a 24-year-old female who sought treatment for social anxiety. Patricia, an only child, had “always been shy.” She reported having just a few friends in high school, although she developed a more active social life in college, primarily by drinking alcohol to reduce her anxiety. Patricia recently started graduate school in a new city and was intent on making friends at student social gatherings. Her busy schedule motivated her to drink less, and as a result, she found her anxiety elevated at social events.

Patricia’s anxiety also increased abruptly after she was asked to deliver a 5-min presentation to her class summarizing a homework assignment. She recalled that she froze, her heart pounded, her hands shook, her face flushed, and her mind “went blank.” “Nothing came out of my mouth – everyone stared at me and I looked like a complete idiot.” When she finally spoke, her voice was shaking but she delivered her presentation reading from her prepared notes. She spent the remainder of the day “obsessing” about her performance and anticipating negative reactions from peers. In the coming weeks her anxiety increased as she realized that her graduate classes would require more frequent class presentations. Overtime, she became preoccupied with wondering when the next presentation would be and she became more uncomfortable with raising her hand to speak in class. In addition, she became more self-conscious at social gatherings, fearing that her peers would ask her about what happened in class. Increasingly, she began to dread social gatherings expecting that she would “make a fool of herself again” and she often made excuses to avoid them, or else endured them by “keeping a low profile.” Her ever-present dread of speaking in class caused her to avoid classes and her concern about her academic performance led her to seek treatment.

Unbeknownst to her therapist, Patricia also had suffered from an ED intermittently since age 15. Patricia did not disclose this at intake because she did not see it as part of the problem that was interfering with daily life. On the contrary, Patricia thought the behavior was helpful to her in maintaining her weight; moreover, it reduced her anxiety because she could reassure herself at social situations that she was one of the thinnest women in the room.

At intake, the therapist focused on Patricia's social anxiety, its effects on her life, and how she was coping; her eating habits were not discussed. Several weeks into treatment, however, Patricia alluded to discomfort eating with others in restaurants. When the therapist probed further Patricia acknowledged that this was not solely due to concerns about others watching, but also because the food choices were "fattening." Patricia was concerned both that the foods would cause weight gain and that others would think her gluttonous for eating such foods. At this point, the therapist began to suspect an ED and decided that further assessment and a more comprehensive case formulation would be helpful. Upon further probing, Patricia confessed that she "sometimes" vomited after large meals or after "gorging" while she was home studying. She had begun purging in high school. She had vomited less frequently in college and noted that living in the dorm made it hard to conceal. Instead, she restricted her intake to a limited range of low-calorie foods. With the increased stress of graduate school, the escalating social anxiety, lower alcohol consumption, and living alone, she began bingeing more when highly stressed. Within a few weeks she became very concerned about her weight, which had increased slightly, and increased the frequency of her vomiting from occasional to several times per week. She also started vomiting after fairly moderate meals. In addition, Patricia began to look forward to evenings alone when she could binge to escape the stress of the day knowing that she would have the release of vomiting afterward. Despite her efforts, Patricia was slowly gaining weight due to the increasing binges and this led to increased self-consciousness in public and increasing avoidance of social gatherings. In addition, her fears of weight gain led her to further limit her food intake during the day as she ruminated about the effects of her previous night's bingeing and anticipated an upcoming social event.

1. *Problem List:*

- Fear of public speaking
- Anxiety and avoidance of social gatherings
- Avoidance of classes and declining academic performance
- Social isolation
- Binge eating
- Weight gain
- Vomiting after binges and forbidden foods
- Excessive concern with shape and weight
- Food restriction (including low protein intake)
- Possible electrolyte imbalance and cardiac abnormalities

2. *Diagnoses:*

- Axis I: Social phobia, BN
- Axis II: None
- Axis III: R/O electrolyte imbalance; R/O cardiac abnormalities; R/O osteoporosis
- Axis IV: None
- Axis V: 50

3. *Anchoring Diagnosis*: Social phobia

4. *Relevant Nomothetic Formulations*:

- *Cognitive-behavioral model of social phobia* (Hofmann & Scepkowski, 2006).
- *Cognitive-behavioral model of Bulimia Nervosa* (Fairburn, Marcus et al., 1993)
- *Transdiagnostic CBT model of EDs* (Fairburn et al., 2003; Fairburn, 2008)

5. *Individualize the Template*:

*Biological/Somatic factors*: Blushing, heart pounding, shaking when speaking to others. Also, evening fatigue from high anxiety, and hunger/lightheadedness from food restriction.

*Behavioral factors*: Avoidance: public speaking, social gatherings, eating in restaurants, daytime food consumption. Safety behaviors: wearing loose clothing to conceal weight gain.

*Cognitive factors*: “I’ll never measure up.” “People will think I’m an idiot.” “If they knew about my vomiting they would be disgusted and not talk to me.” “They’ll see how fat I am and think I’m disgusting.” “I’ll never make friends here.” “I don’t fit in.”

6. *Working Hypotheses*. The nomothetic model of social anxiety proposes that social anxiety is maintained by an interaction of cognitive (high standards for social performance, low perceived ability to meet standards, and high perceived social costs of not meeting standards; negative self-perception, low perceived emotional control) and behavioral factors (increased self-focused attention, avoidance and/or safety behaviors in social situations). The therapist hypothesized that the social phobia was the core disorder and that the same factors (e.g., perfectionism, avoidant coping) that helped to maintain the social phobia also maintained the ED, along with the self-perpetuating cycle of restriction, binge eating, and purging. The therapist presumed that the ED also contributed to maintenance of her social anxiety but that this influence was less powerful. Patricia’s therapist hypothesized that Patricia had set very high standards for her performance in social situations. After the speaking incident, Patricia believed that she was incapable of performing to her standards for public speaking. This led to increased self-focused attention as she scanned her body and behavior for indications that she might perform poorly by displaying her anxiety while speaking either to a group or in a social interaction. The more she attended to herself, the more anxious she became about signs of anxiety she detected, which further increased her anxiety. Her growing anxious apprehension in anticipation of possible public speaking tasks or other social situations then led to elevated levels of arousal. Without her old coping strategy (alcohol), Patricia resorted to bingeing as a means of reducing anxiety. Although bingeing resulted in short-term anxiety reduction by distracting her from social fears, it triggered anxiety about weight gain, which resulted in increased vomiting. Purging reinforced Patricia’s use of bingeing to cope with anxiety. Bingeing also, however, resulted in slight weight gain. Moreover, it led to an increase in negative self-perceptions due to weight

gain and strengthened her perception of herself as being unable to meet her high social standards. Patricia also began to feel ashamed of her binge/vomit behavior which she believed was “disgusting” and fears of rejection by others if they found out compounded her anxious apprehension of social encounters. Her therapist hypothesized that her weight gain and her reliance on binge/purge behaviors to cope with her social anxiety, together, were increasing her avoidance. Her avoidance of public speaking and social gatherings was, in turn, interfering with habituation of her fears of these situations and preventing her from testing the validity of her beliefs about her social performance.

7. *Precipitants of current episodes:* Starting graduate school, decreasing alcohol use, living alone, the “speaking incident,” weight gain.

### ***Treatment Plan***

Patricia was not motivated to address her ED, in part because it was her only strategy for coping with her anxiety as well as the overall stress of graduate school. She remarked to her therapist on several occasions how stressed she felt during the day and the remarkable relief she felt when she came home knowing that the comfort of food awaited her. At the same time, she felt consumed by her preoccupation and dread of public speaking and wanted to rid her mind of her “obsession” with this fear so that she could focus on her studies. Her therapist hypothesized that it would be difficult to reduce her bingeing while her anxiety levels were high. Likewise, if her social anxiety were diminished it would result in less reliance on bingeing as a distraction and escape from her anxiety. Thus, the initial treatment plan focused primarily on direct treatment of social anxiety using an exposure-based protocol while monitoring her binge–purge behaviors. Patricia agreed to keep daily records of her bingeing and vomiting. She also agreed to meet with a physician to assess her medical status. The therapist anticipated that teaching Patricia to consume regular daytime meals (rather than restricting daytime food intake) would decrease her propensity to binge, but to ensure that the strategy would succeed, she decided to postpone implementing it, and more complete self-monitoring of food intake, until later in treatment – approximately 8 weeks into the social anxiety component of treatment. At this point she expected that Patricia’s overall anxiety might be reduced to the point that adding this intervention (regular meals per CBT-BN) might result in a decrease in binge frequency before directly addressing her purging behaviors and over-valuing of the thin-ideal.

### **Ordering of Treatment: Factors to Consider**

Several approaches can be taken to address comorbid conditions. First, treatment can be administered sequentially (i.e., treat one disorder, then reassess and treat the second disorder). This is often the most parsimonious course of action. One other advantage to this approach is that treatment may be closer to that employed

in clinical trials thus increasing the likelihood that research results will generalize to the individual case. In addition, emerging anxiety disorder research indicates that targeting one disorder (e.g., panic disorder) may bring about reductions in comorbid anxiety conditions (e.g., GAD; Tsao, Mystkowski, & Zucker, 2005). Also, as noted by Wilson (1998), research suggests that effective treatment of BN with CBT results in generalized improvement in depression and self-esteem (Fairburn, Jones, Peveler, Hope, & O'Connor, 1993). Thus, it is possible that effective treatment of one disorder may lead to improvement in the comorbid condition, and that a single intervention is sufficient.

One challenge in using a sequential approach – either with the hope that the comorbid disorder will resolve or with the plan to address the second disorder at a later date – is deciding which disorder to address at the outset. In addition, given the relative paucity of research clearly demonstrating whether treating an anxiety disorder leads to a reduction in ED symptoms, clinical decisions about which disorder to target first must be made on the basis of the case formulation. Although each patient is unique, we use the following rules of thumb when considering a sequential approach. First, in our experience, compared to EDs anxiety disorders are usually somewhat easier to treat. Thus, rapid improvement may be more readily obtained by focused anxiety disorder treatment, which may strengthen the therapist's credibility in the eyes of the patient. A second advantage of targeting the anxiety disorder first is that patients are often less ambivalent about receiving treatment for their anxiety disorder, particularly if they are presenting to an anxiety clinic or therapist. In addition, because ED treatment increases anxiety, patients with comorbid EDs and anxiety disorders sometimes find the increase in anxiety difficult to tolerate, thus stalling progress during ED treatment. Finally, the ED may function as a coping or avoidance strategy, particularly for patients with a comorbid diagnosis of PTSD. ED behaviors commonly have an anxiety-reducing function – bingeing, purging, and/or preoccupation with food and weight/shape can serve to distract from anxiety and anxiety-provoking stimuli. Thus many patients understandably are reluctant to give up their ED. In such cases, it may be difficult to resolve the ED without resolving at least some of the need for the coping strategy.

Some factors, however, may point to addressing the ED first. For example, if the anxiety disorder is not severe and/or the patient admits that the ED is a problem, it may make sense to begin with ED treatment. A sizeable number of patients with BN respond to CBT-BN, and Axis I comorbidity has not been a solid predictor of outcome in clinical trials (Wilson, 2005). Also, at times, the medical implications of the ED may necessitate immediate intervention.

In many cases, a true sequential approach may not be feasible, as highlighted by the cases described above. In such cases another option is what we refer to as a simultaneous monitoring approach (Zayfert & Becker, 2007). In this approach, the patient and clinician agree to predominantly target one disorder while simultaneously monitoring the other disorder. Often the “monitoring” may also include some low-level intervention. For example, in Patricia's case, the therapist planned to start with social phobia treatment while monitoring binge eating and vomiting. She then planned to introduce food monitoring and a regular pattern of eating (i.e., a schedule of three meals and two snacks per day) once some reduction of overall anxiety

had been achieved from the social anxiety treatment. The therapist expected that the lower anxiety level would potentiate the effect of this intervention and result in reduced binge frequency.

In yet other cases, a completely blended (simultaneous) approach may be warranted. In our experience, these cases often present to ED clinics because of the severity of the ED. They also, however, may present to trauma treatment programs. Thus, clinicians who treat PTSD may encounter such cases. Unfortunately, it is beyond the scope of this chapter to fully detail a simultaneous approach. We refer readers to Zayfert and Becker (2007) for more detailed discussion of the simultaneous treatment of PTSD and comorbid conditions, such as EDs.

## Conclusion

EDs can present a challenge for anxiety disorder clinicians. Although they co-occur with anxiety disorders more often than many clinicians realize, anxiety patients rarely disclose ED symptoms to clinicians from whom they are seeking anxiety treatment. Such patients often are highly distressed by their anxiety and less motivated to address their ED. They may see troublesome eating as a legitimate and normal (and not unhealthy) weight management strategy and/or as essential to coping with their anxiety. They may not even recognize anything unusual. Yet, as the above cases illustrate, the interactions between EDs and anxiety problems can be complex and the problems can become mutually maintaining. ED behaviors can be precipitated by anxiety (such as when Patricia used bingeing to cope with high anxiety or when Emily's food avoidance took on a weight-loss function in addition to neutralizing her contamination obsessions). They can also precipitate anxiety (as when Emily interpreted dizziness due to electrolyte imbalance as a sign of illness and thus increased her food sanitizing and avoidance, and when Patricia's bingeing and purging exacerbated her social anxiety because she feared others would notice her weight gain or discover her vomiting). EDs also present a challenge for anxiety clinicians because many clinicians well-versed in anxiety disorders do not have specialized training in evidenced-based treatment for EDs. Nonetheless, clinicians can manage these co-occurring problems by first being prepared to detect them, and secondly incorporating them into the evidence-based case formulation. Formulating hypotheses about how the ED may reinforce the anxiety disorder and vice versa is often essential to treatment success. Often the formulation will lead to a treatment plan that begins with anxiety treatment and aims to build motivation to eventually address the ED, either with the anxiety clinician, or accepting a referral to a specialized ED treatment provider.

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# Resolving Treatment Complications Associated with Comorbid Medical Conditions

Joseph Greer, Jessica Graham, and Steven Safren

## Introduction

Anxiety disorders, the most prevalent psychiatric morbidity diagnosed within the United States (Kessler, Berglund, et al., 2005; Kessler, Chiu, Demler, Merikangas, & Walters, 2005), frequently co-occur with serious medical conditions. Indeed, recent epidemiological studies confirm that individuals with anxiety disorders are at substantially greater risk for experiencing comorbid medical illnesses, such as cancer, cardiac, and respiratory diseases, even when adjusting for patient demographic characteristics, depression, and substance use disorders (Honda & Goodwin, 2004; Sareen, Cox, Clara, & Asmundson, 2005; Sareen et al., 2006). Not surprisingly, individuals with anxiety and medical conditions are more likely to report functional disability and poorer quality of life, compared to patients without anxiety (Kessler, Ormel, Demler, & Stang, 2003; Sareen et al., 2006; Sherbourne, Wells, Meredith, Jackson, & Camp, 1996; Stein et al., 2005). Moreover, the comorbidity between anxiety and various physical disorders substantially increases mean total annual medical costs, with a notable incremental expense of \$7,378 for patients with acute myocardial infarction (Marciniak et al., 2005).

Despite the emergence of several large-scale studies examining the strong association between anxiety disorders and medical illness, the exact nature and direction of this complex relationship remains unclear. As Sareen et al. (2006) note, one potential explanation is that medical illness leads to an increase in symptoms of anxiety by provoking the stress response, particularly when the physical condition is life threatening and involves invasive medical procedures. Beyond simply triggering autonomic arousal, medical illness can also cause anxiety symptoms directly through physiological mechanisms, such as dyspnea associated with pulmonary embolus or tachycardia in hyperthyroidism. Conversely, anxiety may potentially lead to the development or exacerbation of certain medical conditions through chronic activation of the hypothalamic-pituitary axis and related changes in immune function (Sareen et al., 2006). Moreover, treatment adherence and other health behaviors, genetics, and socio-environmental circumstances play salient

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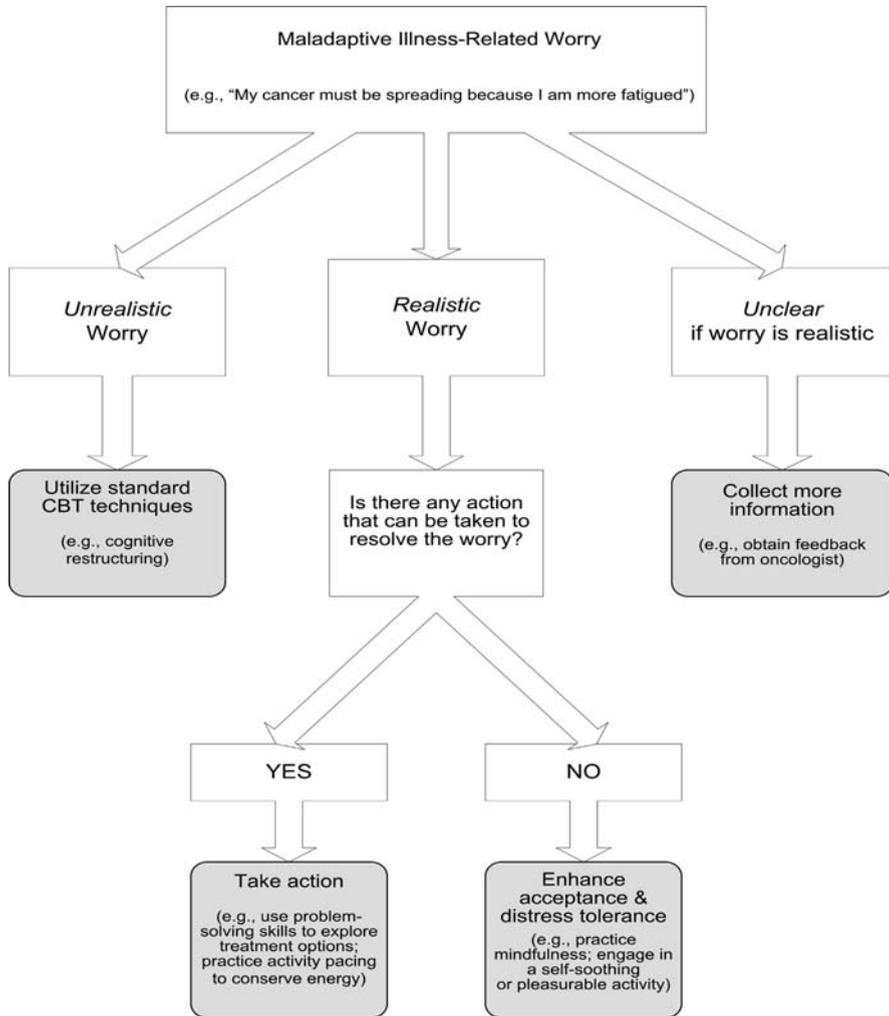
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roles in the association between anxiety and physical illness. For example, anxious patients with coronary artery disease are more likely to continue smoking than non-anxious patients (Benninghoven et al., 2006). Further complicating the assessment of anxiety within the context of medical conditions, some physical illnesses (e.g., cancer) involve toxic medical treatments, which may lead to symptoms that are often mistaken as anxiety. Close collaboration between the medical team and mental health clinician is essential for accurate diagnosis and management of anxiety, given the multiplicity of etiologies and overlap in disease presentation.

Complications are likely to arise not only in the assessment but also in the treatment of anxiety comorbid with medical conditions. Although cognitive-behavioral therapy (CBT) is an effective first-line treatment for anxiety disorders (Otto, Smits, & Reese, 2004), it has been tested primarily in medically healthy populations and focuses on the elimination of unrealistic fears as well as behavioral avoidance and other maladaptive coping. By restructuring distortions in perceptions, particularly the overestimation of negative outcomes, and by confronting feared stimuli through exposure-based therapies, CBT helps individuals to challenge catastrophic thoughts and to extinguish their anxiety (Barlow, 2002). In contrast, individuals with medical conditions, who frequently experience disease-related symptoms and treatment side effects, report realistic concerns about pain, functional disability, and in some cases, even death. Within this context, traditional CBT for restructuring fears about mortality or symptoms of disease progression is less useful, depending on the patient's prognosis. Cognitive restructuring plus exposure may not be sufficient to reduce symptoms of anxious preoccupation and worry in patients who are physically ill and coping with medical symptoms, tests, procedures, and an uncertain future.

In addition, medical patients often report an escalation of anxiety when experiencing somatic concerns like fatigue, pain, or nausea, which are functionally limiting and at times difficult to interpret. This anxiety, in turn, through the physiologic changes associated with the stress response (e.g., muscle tension, rapid respirations, digestive slowing), can heighten the severity of disease and treatment-related symptoms, perpetuating the cycle and causing further disability. Comprehensive cognitive-behavioral approaches to the management of anxiety comorbid with physical disease must therefore address both psychological and medical illness concerns. Figure 1 depicts a treatment approach for evaluating and managing the maladaptive worry and functional limitations associated with medical illnesses (partially adapted from Moorey & Greer, 2002).

To highlight methods for resolving treatment complications for anxiety comorbid with medical conditions, we discuss three disease states that have a well-documented association with anxiety disorders. Although anxiety is related to numerous other medical conditions, we have chosen to narrow our focus to these three illnesses in order to illustrate a comprehensive approach for adapting standard cognitive-behavioral techniques in resolving treatment complications. Specifically, we review the literature on the symptoms, epidemiology, correlates, and psychological treatment of anxiety associated with cancer, coronary heart disease, and asthma. Many of the challenges and recommendations for treating anxiety comorbid with medical illness naturally can be applied across various diseases, though we



**Fig. 1** Algorithm for addressing illness-related worry

have decided to highlight methods for adapting cognitive-behavioral therapy when discussing each of the specific conditions throughout the chapter.

### **Anxiety Comorbid with Cancer**

Cancer, or malignant neoplasm, is characterized by unregulated cell growth, resulting from chromosomal mutation. Malignancy can develop in nearly every cell type of the body, and these cancer cells may invade organs locally or spread to distant sites via the blood stream or lymphatic system. More than 1.4 million Americans

received a new cancer diagnosis in 2008, the most of which were prostate, breast, and lung (American Cancer Society, 2008). Cancer is responsible for one in every four deaths in the United States, making it the second leading cause of mortality behind cardiovascular disease (Kung, Hoyert, Xu, & Murphy, 2008). Yet, ongoing advances in screening procedures and improvements in medical treatment regimens have led to significant increases in cancer survival over the last two decades, with approximately 10.8 million Americans living with cancer today (National Cancer Institute, 2007; Ries et al., 2008).

A generation ago, the diagnosis of cancer carried with it the stigma of poor prognosis and expectation for an early and painful death. Today, while some people still experience rapid decline, particularly those with certain types of lung or pancreatic cancers, many more individuals are either cured of their disease or receive ongoing treatment (American Cancer Society, 2008). As cancer care continues to evolve, allowing for longer survival and transforming cancer into a chronic medical condition, even for those with advance-stage malignancy, many individuals will experience concomitant anxiety that is debilitating and at times exacerbating of the disease-related symptoms and treatment side effects.

Approximately 10–25% of individuals with cancer have a diagnosable anxiety disorder (Stark & House, 2000). In some cases, this anxiety reflects an exacerbation of a premorbid sub-clinical or clinical condition, while in others, anxiety symptoms emerge in response to stressors from the cancer diagnosis and treatment. Indeed, the experience of anxiety is a natural reaction to news of a life-threatening diagnosis and abates for many patients with cancer as the prognosis and treatment plan are clarified. Thus, differentiating persistent, maladaptive anxiety symptoms from more transient, self-limited distress is often challenging. Although as many as 40% of individuals with cancer screen positive for psychological distress (Fulton, 1998; Zabora, Brintzenhofesoc, Curbow, Hooker, & Piantadosi, 2001), Stark et al. (2002) found that approximately 18% qualify for an ICD-9 anxiety disorder. Such variation in prevalence may be due to differences not only in the methods and timing of anxiety assessment but also in the types and stages of cancers sampled as well as medical treatment factors.

While some degree of anxiety is protective and useful, ideally facilitating effective coping behaviors, the acute cognitive and behavioral changes associated with the stress response may become maladaptive, particularly among individuals with medical illness. Common features of dysfunctional anxiety among individuals with cancer include intrusive negative thoughts (Devine, Parker, Fouladi, & Cohen, 2003), excessive worry (Nordin, Glimelius, Pahlman, & Sjoden, 1996), and deficient coping (Wasteson, Nordin, Hoffman, Glimelius, & Sjoden, 2002). Patients experiencing such symptoms report disruptions in concentration, decision-making, sleep, and social/occupational functioning (Stark & House, 2000). This impairing anxiety is also associated with ineffective behavioral responses, such as decreased medical adherence, reassurance seeking, longer hospital stays, social withdrawal, and other avoidance behaviors (Prieto et al., 2002; Ristvedt & Trinkaus, 2005; Shapiro, 1987; Stark et al., 2004; Thomas, Glynne-Jones, Chait, & Marks, 1997; Weinmann et al., 2005). For example, patients with specific phobias such as claustrophobia or needle

phobias may compromise their cancer care by avoiding necessary medical procedures and scans. Recent findings from our own work (Greer, Pirl, Park, Lynch, & Temel, 2008) in patients with advanced non-small-cell lung cancer revealed that heightened anxiety symptoms within 2 months of diagnosis predicted poorer treatment adherence, including more chemotherapy dose reductions and dose delays, compared to those with less anxiety.

### ***Cancer-Related Symptoms, Treatment Side Effects, and Quality of Life***

In addition to precipitating maladaptive changes in thought and behavior, anxiety symptoms aggravate the expected somatic symptoms and treatment side effects seen in patients undergoing cancer care. Fatigue, insomnia, and pain are among the most common complaints in patients with cancer diagnoses. Anxiety may intensify the experience of these symptoms, given that it strongly relates to patients' perception of cancer-related pain (Theobald, 2004; Thielking, 2003; Velikova, Selby, Snaith, & Kirby, 1995; Zimmerman, Story, Gaston-Johansson, & Rowles, 1996) as well as fatigue and insomnia (Barnes & Bruera, 2002; Fossa, Dahl, & Loge, 2003; Redeker, Lev, & Ruggiero, 2000; Stark et al., 2002). Additionally, compared to non-anxious patients, individuals with cancer and comorbid anxiety report more complications with chemotherapeutic treatments, including side effects of medications, such as tamoxifen (Cameron, Leventhal, & Love, 1998), worse nausea and vomiting (Andrykowski, 1990; Chin, Kucuk, Peterson, & Ezdinli, 1992; Watson, Meyer, Thomson, & Osofsky, 1998), and poorer response to anti-emetic drugs (Fujii et al., 2001). The relationships among anxiety, disease, and treatment-related symptoms are complex and, as Theobald (2004) notes, each may contribute to the maintenance of the other, resulting in significant impairments in quality of life.

Cancer patients with higher levels of anxiety also report having poorer quality of life than less anxious individuals. In one investigation of women receiving treatment for breast cancer, for example, higher levels of anxiety at the start of treatment were negatively correlated with quality of life at initiation of chemo-radiotherapy and 1 year later (Schreier & Williams, 2004). The authors advocated further research into interventions targeting anxiety symptoms at the onset of treatment. Similarly, Stark et al. (2002) reported that, in a sample of 178 oncology patients with diverse primary tumor sites, anxiety disorders measured by screening questionnaires and diagnostic interviews were present in 18% of participants and associated with poorer quality of life, especially insomnia. Two other studies confirm that impaired quality of life for cancer patients correlates strongly with anxiety and that cancer-related symptoms such as global health status, fatigue, pain, and illness severity do not explain observed quality-of-life deficits (Redeker et al., 2000; Smith, Gomm, & Dickens, 2003).

## ***Psychological Interventions for Anxiety Comorbid with Cancer***

Psychosocial interventions to treat patients with cancer ranged in format (e.g., group versus individual), technique (e.g., supportive versus psychoeducational), target population (e.g., patient versus provider), and method of delivery (e.g., face-to-face versus telephone versus web-based) (Andersen, 2002; Fawzy, Fawzy, Arndt, & Pasnau, 1995). Given the large number of studies in this area, several investigators have conducted meta-analyses of the literature to discern the benefit of psychosocial interventions for a variety of mental health and cancer outcomes, including anxiety, quality of life, and survival (Chow, Tsao, & Harth, 2004; Devine & Westlake, 1995; Meyer & Mark, 1995; Newell, Sanson-Fisher, & Savolainen, 2002; Sheard & Maguire, 1999). Employing rigorous methodology to evaluate the efficacy of psychological therapies for cancer, Newell et al. (2002) found 1 “good-quality” trial and 24 “fair-quality” trials that evaluated interventions aimed at reducing oncology patients’ anxiety. Of these, only 11 included cognitive-behavioral therapy, with 3 demonstrating statistically significant results. The authors therefore recommended that CBT for anxiety among cancer patients warrants further exploration since published investigations to date have yielded inconsistent findings due to poor methodological quality (e.g., inadequate specification of randomization strategy, poor monitoring or protocol adherence), small sample sizes, and short-term follow-up periods. However, a more recent meta-analysis of randomized controlled trials of cognitive-behavioral therapy revealed a large effect size ( $ES = 1.99$ ) for decreasing anxiety among cancer survivors (Osborn, Demoncada, & Feuerstein, 2006). Whereas these results provide some encouraging evidence for the utility of CBT to alleviate anxiety in medically complex patients, further high-quality trials testing the efficacy of CBT tailored to the needs of patients with cancer may help overcome the limitations of previous research.

## ***Complications in Treating Anxiety Comorbid with Cancer***

The difficulties of treating anxiety comorbid with cancer often begin with the referral for psychosocial services and the challenges in scheduling sessions (Curry, Cossich, Matthews, Beresford, & McLachlan, 2002). Upon receiving a cancer diagnosis, patients need to attend numerous medical appointments for diagnostic procedures, treatment planning, surgery, and chemo-radiotherapy. As a result of anti-cancer treatment, patients may experience functional limitations and side effects such as nausea, pain, and severe fatigue, frequently lasting for days. These symptoms not only interfere with quality of life but also result in family role disruptions, work absenteeism, and considerable financial cost (Fortner et al., 2003; Hassett, O’Malley, Pakes, Newhouse, & Earle, 2006; Kim, 2007). Scheduling psychotherapy appointments in addition to those for primary cancer care may at times feel overly burdensome to patients.

In addition to barriers to providing care, clinicians may struggle with the differential diagnosis of patients’ somatic symptoms (Ryan et al., 2005). For

example, while some patients present with complaints of feeling anxious and worried, others simply describe problems with dyspnea, nausea, palpitations, or difficulty concentrating. These somatic complaints may indicate a primary anxiety disorder but also could be due to serious medical complications related to the disease process (e.g., acute shortness of breath from pulmonary embolus), medical treatment (e.g., nausea from chemotherapy), or medication side effects (e.g., agitation and restlessness from use of corticosteroids and phenothiazines; Massie & Greenberg, 2005). Of course complicating the diagnostic considerations further is that the associations between patients' anxiety and illness-related symptoms are likely bidirectional and complex in nature.

After considering the medical reasons for anxiety symptoms, clinicians must then discern a treatment plan and the optimal approach for adapting empirically based psychotherapies to address the individual needs of the cancer patient. Given that researchers have predominantly tested and validated cognitive-behavioral therapy for anxiety disorders in relatively homogenous samples, often excluding patients with multiple conditions, the application of these techniques to patients with comorbidities poses another challenge to the mental health clinician (Ruscio & Holohan, 2006). In the case of cancer, of primary concern is how to help anxious patients cope with fears about an uncertain future, whether due to disease progression, cancer recurrence, or imminent death; that is, what methods should be employed when trying to intervene and challenge maladaptive thoughts that are based on realistic concerns? How does the mental health clinician within the oncology setting help to reduce anxiety and life stress when a patient's functioning is compromised and triggers of disease worries (e.g., pain) are chronic and recurrent? In response to these issues, investigators have begun to publish clinical guides that address the tailoring of cognitive-behavioral therapy for individuals coping with various chronic illnesses, including cancer (Sperry, 2006; Taylor, 2006; White, 2001).

### ***Resolving Treatment Complications for Anxiety Comorbid with Cancer***

To minimize barriers for patients with cancer to access care, mental health professionals would ideally be integrated into the ambulatory cancer center setting. While this is generally not the case at present (Greenberg, 2004), clinicians who are co-located at a tertiary care hospital may schedule psychotherapy sessions in tandem with the patient's existing medical appointments in order to decrease the burden of multiple hospital visits. When not located on-site, however, clinicians would want to consider the patient's current medical treatment regimen and determine the appropriate timing for scheduling outpatient psychotherapy sessions. For example, patients who experience extreme fatigue secondary to chemotherapy should schedule their counseling sessions only after sufficient time has passed following infusions.

To address assessment issues regarding anxiety comorbid with cancer, clinicians need to stay abreast of the most common organic etiological factors, such as those

discussed earlier, which potentially underlie patients' anxiety symptoms. However, oncologic care will continue to evolve at a rapid pace, and multidisciplinary team-based approaches, encouraging regular communication among medical and mental health colleagues, facilitate effective diagnosis and targeted treatment of anxiety comorbid with cancer. Such models are being tested with notable benefits for patients in the ambulatory oncology settings (Bruera et al., 2001; Graves et al., 2007; Strasser et al., 2004). Recognizing the importance of identifying and alleviating psychological distress associated with cancer, the National Comprehensive Cancer Network, a non-profit alliance of leading cancer centers worldwide, has recommended routine screening for psychological distress and developed specific guidelines for the evaluation and treatment of anxiety in this patient population (National Comprehensive Cancer Network, 2006).

As detailed in the treatment algorithm at the beginning of the chapter, the therapist and patient must evaluate disease-related worries in order to establish which are realistic when providing cognitive-behavioral therapy. Standard cognitive-restructuring practices are applicable to those thoughts that represent likely distortions. For example, during a follow-up medical visit, a patient treated for breast cancer could receive encouraging news about her health and prognosis but misinterpret the oncologist's nonverbal cues (e.g., poor eye contact, not smiling) and make negative predictions about her risk of cancer recurrence. The role of the therapist in this case would be to identify these thoughts as forms of "mind-reading" and "catastrophizing" while working with the patient to develop more rational responses by considering disconfirming evidence, such as the positive results from recent scans and alternate explanations for the oncologist's behavior.

Of course, however, many cancer-related fears will be valid and rooted in reality, based on the patient's experiences and on available data from imaging procedures and feedback from the oncology team. For example, soon after diagnosis, most patients with lung cancer report major concerns about the illness, prognosis, and effects on family (Hill, Amir, Muers, Connolly, & Round, 2003); many of these worries represent realistic risks given the high mortality rates due to lung cancer (Surveillance, Epidemiology, & End Results Program, 2006). Under such circumstances, mental health clinicians should not challenge such thoughts as cognitive distortions, even though they may cause maladaptive responses including disturbances in concentration, sleep, and well-being (Taylor, 2006; White, 2001). Rather, as per the guidelines of the aforementioned treatment algorithm, a more effective approach would include helping patients discern how and when to apply problem versus emotion-focused coping strategies to their realistic worries. More specifically, Moorey and Greer (2002) suggest that clinicians utilize a decision tree to determine the extent to which patients are able to take some action in resolving their concerns. If action is possible or needed, such as seeking further medical consultation, clinicians may then employ problem-solving skills, developing a list of possible solutions, evaluating the pros and cons for each, choosing the best option, and creating a step-by-step action plan to address the worry (Nezu, Nezu, Friedman, Faddis, & Houts, 1998).

However, there will be times when patients correctly note that no action can be taken to resolve their worry, as in the case of a patient with advanced pancreatic malignancy who is anxious about poor prognosis. In such moments, clinicians may encourage patients to tolerate their fears, drawing on alternate interventions aimed at coping with the emotional distress such as engaging in pleasurable activities, mindfulness meditation, distraction, and self-soothing techniques (Moorey & Greer, 2002; Taylor, 2006). Rather than focusing on the maladaptive thought, a patient may find solace by directing attention toward an enjoyable hobby, listening to music, preparing a favorite meal, or receiving a massage. Of growing interest among researchers is the benefit of mindfulness-based meditation for relieving stress and worry among cancer patients (Smith, Richardson, Hoffman, & Pilkington, 2005). Mindfulness meditation involves raising conscious awareness by intentionally bringing attention to internal and external experiences in the present moment and observing thoughts, feelings, and physical sensations in a non-judgmental manner (Kabat-Zinn, 1994). For patients who are at risk of having catastrophic thoughts related to unfavorable prognosis, recurrent pain symptoms, and functional disability, mindfulness may be a useful method for regulating attention, enhancing focus on the present moment, habituating to the experience of negative affect and unpleasant sensations, and increasing acceptance of current physical status (Hamilton, Kitzman, & Guyotte, 2006).

## **Anxiety Comorbid with Coronary Heart Disease**

Coronary heart disease, also known as coronary artery disease or atherosclerotic heart disease, is the leading cause of death for men and women in the United States despite impressive advances in cardiac risk reduction, development of high-tech interventions in cardiac care, and increasing knowledge about the benefits of drug treatment after heart attack. Sixteen million Americans have coronary heart disease, causing as many as 450,000 deaths annually in recent years. In 2008, the estimated cost of coronary heart disease was \$156.4 billion, which included not only direct medical expenses but also indirect costs, such as lost wages and productivity (American Heart Association, 2008). The symptoms of coronary heart disease result from the narrowing of the coronary arteries, which supply blood to the heart. Also called atherosclerosis, the narrowing and hardening of the coronary arteries develops over time from the accumulation and calcification of cholesterol plaques on the inner wall of the blood vessels. Eventually, this progressive narrowing may reduce the flow of blood and oxygen to the heart (called ischemia) either chronically or acutely, causing serious cardiac conditions including angina, arrhythmia, heart attack, and potentially heart failure (Selwyn & Braunwald, 2005).

Few data exist regarding the prevalence of anxiety disorders in patients with coronary heart disease. Based on the extant literature, panic disorder ranged from 10% to 50% in studies of cardiology outpatients and individuals with diagnosed coronary artery disease (Fleet, Lavoie, & Beitman, 2000). Using the Structured

Clinical Interview for the DSM-IV to assess for psychiatric diagnoses, Bankier, Januzzi, and Littman (2004) reported high rates of posttraumatic disorder (29%) and current generalized anxiety disorder (24%) in a sample of 100 stable outpatients with coronary heart disease. However, these results provide no indication as to whether the anxiety disorders preceded coronary heart disease (CHD) diagnosis or were the result of experiencing a significant medical stressor, such as myocardial infarction.

### *Anxiety as Risk Factor for Coronary Heart Disease*

In addition to poor health behaviors (e.g., smoking), investigators have identified anxiety, depression, anger, hostility, personality traits (e.g., Type A & D behavior patterns), and chronic life stressors, such as social isolation and low socioeconomic status, as key risk factors for coronary heart disease (Januzzi, Stern, Pasternak, & DeSanctis, 2000; Kop, 1999; Kubzansky, Davidson, & Rozanski, 2005; Rozanski, Blumenthal, Davidson, Saab, & Kubzansky, 2005; Smith & Ruiz, 2002). Although studies have been limited and somewhat mixed due to methodological weaknesses (Rozanski et al., 2005), early investigations revealed associations between coronary heart disease, especially sudden cardiac death, and anxiety symptoms such as panic disorder (Coryell, Noyes, & House, 1986), phobic anxiety (Haines, Imeson, & Meade, 1987; Kawachi et al., 1994), and worry (Kubzansky et al., 1997). More recently, researchers have questioned the utility of studying psychological attributes in isolation (Suls & Bunde, 2005) and begun to explore the covariation and shared contributions of multiple affective states, such as anxiety, depression, and anger, given that the combination appears to account for greater variability in the incidence of coronary heart disease (Boyle, Michalek, & Suarez, 2006; Kubzansky, Cole, Kawachi, Vokonas, & Sparrow, 2006). Interestingly, even when adjusting for the general distress from the overlap of negative emotions, aspects of anxiety still independently predict the development of non-fatal myocardial infarction (Kubzansky et al., 2006).

Anxiety symptoms increase risk for coronary heart disease primarily through two pathways: unhealthy lifestyle behaviors and direct biological mechanisms. The worldwide INTERHEART study of 52 counties showed that greater than 90% of the risk for acute myocardial infarction is due to modifiable factors, with smoking and elevated cholesterol accounting for two-thirds of the risk (Yusuf et al., 2004). Although the results from this large-scale investigation demonstrated that psychosocial variables exert independent effects on the likelihood of first myocardial infarction (Rosengren et al., 2004), anxiety may simultaneously play an indirect role, via the development and maintenance of other primary cardiac risks, such as smoking (Benninghoven et al., 2006; McKenna & Higgins, 1997) and obesity (Simon et al., 2006). Moreover, researchers have begun to explore the degree to which anxiety and mental stress relate to the development of atherosclerosis and risk for cardiovascular events through a myriad of physiological processes (Rozanski et al., 2005) including impairment in myocardial perfusion (Fleet et al.,

2005), thrombosis (von Kaenel et al., 2005), ventricular arrhythmias (Watkins et al., 2006), and decreased heart-rate variability (Lavoie et al., 2004).

### ***Persistent Effects of Anxiety and Quality of Life in Patients with Coronary Heart Disease***

Anxiety not only precedes the diagnosis of coronary heart disease but also occurs subsequent to major cardiac morbidity such as heart attack, causing worse psychological effects among young patients (Lavie & Milani, 2006). Such anxiety symptoms are common following myocardial infarction (Lane, Carroll, Ring, Beevers, & Lip, 2002) and confer greater risk for recurrent cardiac events as well as increased utilization of healthcare services (Benninghoven et al., 2006; Strik, Denollet, Lousberg, & Honig, 2003). Moser and Dracup (1996) observed that anxious patients were almost five times more likely to have ischemic and arrhythmic complications after acute myocardial infarction compared to patients with less anxiety. Moreover, cardiac treatment providers fail to detect clinically significant anxiety symptoms in at least 50% of distressed patients (Huffman et al., 2006), underscoring the need for enhanced screening procedures both before and after the occurrence of major heart-related events.

In addition to changes in anxiety levels, patient-reported quality of life frequently worsens after myocardial infarction or cardiac revascularization procedures, particularly among women (Westin, Carlsson, Erhardt, Cantor-Graae, & McNeil, 1999). Several prospective studies have shown that anxiety symptoms significantly predict decrements in health-related quality of life and physical functioning not only in the first 12 months after a major cardiac event (Dickens et al., 2006; Hofer, Doering, Rumpold, Oldridge, & Benzer, 2006; Sullivan, LaCroix, Baum, Grothaus, & Katon, 1997) but up to 5 years later as well (Sullivan, LaCroix, Spertus, & Hecht, 2000). Moreover, the persistent negative effects on health-related quality of life and functional status in coronary artery disease are more strongly linked to anxiety and depression than to biomedical factors, such as the number of diseased coronary vessels (Hofer et al., 2005; Sullivan et al., 1997) or type of medical intervention (e.g., percutaneous angioplasty versus coronary artery bypass grafting; Hofer et al., 2006). One potential explanation for this strong association with anxiety is that cardiac events significantly threaten and disturb patients' attainment of higher-order life goals (Boersma, Maes, & Joeke, 2005), and thus is an important target of psychological treatment in this patient population.

### ***Psychological Interventions for Anxiety Comorbid with Coronary Heart Disease***

Non-pharmacological interventions for coronary heart disease include cardiac rehabilitation programs, which may be comprised of monitored aerobic exercise, stress management, nutrition counseling, and smoking cessation, as well

as individual and group psychotherapy, and complementary treatments. Although accumulating evidence demonstrates worse medical outcomes in anxious patients with coronary heart disease, researchers have not observed reduced mortality through treatment of psychological conditions (Rees, Bennett, West, Davey, & Ebrahim, 2004). For example, results of the ENRICHD study, the largest NIH-funded trial to date investigating the utility of empirically supported cognitive-behavioral therapy for depression and social isolation in 2,481 patients with coronary heart disease, showed that rates of mortality and recurrent myocardial infarction were no better in patients receiving CBT versus usual care, despite modest improvements in depression and social support (Berkman et al., 2003). Of note, the ENRICHD study investigators did not address lifestyle behaviors or medical treatment adherence as part of their psychosocial intervention.

The Cochrane Collaboration published a review of psychological interventions for coronary heart disease (Rees et al., 2004) and identified 36 randomized controlled trials that measured clinical events, modifiable risk factors, and/or psychological outcomes. Only 9 of the 36 trials had interventions that targeted anxiety symptoms, which overall yielded a small but statistically significant effect size (weighted mean difference =  $-0.17$ ) for anxiety reduction. Most of these trials included complex stress management interventions with a variety of cognitive-behavioral techniques, such as relaxation training. Although psychological treatment studies for anxiety and depression in patients with coronary heart disease have yet to reveal significant benefit in reducing disease-related morbidity or mortality, the poor quality and heterogeneity of research trials to date limit firm conclusions regarding biomedical outcomes (Rees et al., 2004). Moreover, given the additive impact of anxiety and physical illness on patient's quality of life, mental health clinicians who encounter cardiac patients with psychiatric comorbidities must endeavor to provide and tailor the most efficacious psychological treatments available.

### ***Complications in Treating Anxiety Comorbid with Coronary Heart Disease***

Given the overlap between cardiac and anxiety symptoms, a common challenge for many clinicians and their patients with coronary heart disease is differentiating chest pain and shortness of breath due to cardiac ischemia from symptoms of panic or other anxiety disorders. For example, Fleet et al. (1998) found that in a sample of patients with a history of coronary artery disease presenting to the emergency department with non-cardiac chest pain, 34% met criteria for panic disorder. Further complicating this diagnostic challenge are gastrointestinal disorders, such as gastroesophageal reflux disease, another common cause of chest pain in this patient population that may result from treatment with anti-anginal medications (Ros et al., 1997).

Treatment complications arise for many patients with coronary heart disease after acute myocardial infarction, when they may experience greater somatic monitoring and heightened anxiety associated with symptoms of chest pain, tachycardia, and arrhythmias. This somatic and anxiety sensitivity often leads to avoidance of physical activity, work, and social withdrawal, which may in turn lower anginal thresholds. Standard cognitive-behavioral therapy for panic disorder includes interoceptive exposure treatment in which patients generate and experience feared anxiety symptoms, such as heart palpitations, so that they may habituate to the internal physical sensations and decatastrophize their meaning (Barlow, 2002). However, in the case of coronary heart disease, the warning signs for major cardiac events (i.e., chest tightness, palpitations, diaphoresis, nausea, and shortness of breath) are essentially identical to those of panic and other anxiety disorders. Therefore, exposure-based therapy in this context, while potentially still beneficial for anxiety reduction, must be modified to account for the genuine risks of acute cardiac ischemia.

Although most individuals with coronary heart disease will be able to resume normal functioning relatively soon after an acute myocardial infarction, some will experience Class III or IV angina, characterized as chest discomfort causing marked limitation in physical activity (Selwyn & Braunwald, 2005). In addition, fatigue, or exhaustion, is a common complaint in patients with coronary heart disease (Appels, 2004), which is compounded by use of cardiac medications (Kop, Appels, Mendes de Leon, & Bar, 1996). These functional limitations and side effects may heighten anxiety and reduce quality of life by interfering with patients' abilities to work and achieve their life goals. Anxiety treatment, therefore, must incorporate strategies for adjusting to and managing such disability.

### ***Resolving Treatment Complications for Anxiety Comorbid with Coronary Heart Disease***

As discussed previously with respect to cancer, challenges of differential diagnosis again raise the importance of collaborative care when treating patients with serious chronic medical conditions, such as cancer and coronary heart disease. Communicating with the medical team is essential for the mental health clinician to have an informed understanding of the patient's risk factors, current status, disease course, and prognosis, especially when developing psychotherapy goals and treatment strategies. The biological data, which may be gathered directly from consultation with the cardiologist and from scans and other laboratory tests results, are salient factors when assessing the cognitive, affective, and behavioral targets of care (Belar & Deardorff, 1995). Although western medicine continues to dichotomize patients' symptoms as either medical or psychological in nature, mental health clinicians treating patients with physical illness have perhaps a greater responsibility to bridge these reductionistic approaches in order to develop a com-

prehensive biopsychosocial conceptualization (Engel, 1977) of the relationships and interactions among the patient's psychological disorder, disease, and environment (Belar & Deardorff, 1995). Such models are helpful not only in assessment but for patient education as well. For example, in the case of CHD, clinicians might validate the challenges and burden of managing symptomatic disease, while simultaneously encouraging patients to appreciate the complex ways in which anxiety and stress are both caused by and worsen pain, fatigue, and disability, at times leading to maladaptive coping behaviors, such as avoidance of physical activity.

A primary behavioral target for anxiety treatment in coronary heart disease includes helping patients to generate, experience, and habituate to the somatic sensations of the stress response, which may be achieved through interoceptive exposure and physical activity. As noted, interoceptive exposure, such as conducting an overbreathing exercise or inducing palpitations, often raises alarm for the patient and mental health clinician about stressing the heart given that these symptoms mimic the physical signs of coronary ischemia. However, for a patient with stable heart disease, a clinician may consider employing interoceptive exposure after consultation and clearance with the patient's medical care providers. Additionally, exposure-based therapy could be integrated into structured cardiac rehabilitation programs, which incorporate supervised and graded physical exercises. In such programs, patients are initially encouraged to experience and tolerate unpleasant physical sensations as they exercise at or near their anginal threshold, with the reassurance of continuous electrographic and blood pressure monitoring by a cardiology nurse specialist or exercise physiologist. Over time, patients withdraw monitoring and supervision as they gain confidence in their ability to manage somatic symptoms of the stress response and physically exert themselves safely. In a sample of 588 consecutive patients with coronary artery disease, Lavie and Milani (1997) observed that cardiac rehabilitation with exercise training significantly reduced anxiety symptoms by 40%.

For those individuals who are unable to return to relatively normal physical and role functioning due to impairment from coronary artery disease or its medical treatment, illness management strategies may aid in anxiety reduction (White, 2001). Several validated behavioral techniques derived from the chronic pain literature, such as activity planning and pacing, may assist patients in adjusting their expectations for performance while remaining engaged in social, recreational, and even occupational pursuits (Keefe, Abernethy, & Campbell, 2005). Specifically, for individuals disabled by severe fatigue and recurrent chest pain, clinicians can help guide prioritization and scheduling of daily tasks by alternating physically stressful and less demanding activities, thus enabling the patient to achieve an optimal balance between physical exertion and rest. For many individuals with coronary artery disease, particularly after myocardial infarction, the tendency may be to avoid any physical stress for fear of subsequent heart attack, leading to greater deconditioning. Also, family, friends, and coworkers may inadvertently contribute

to maladaptive illness behavior by being solicitous in taking over responsibilities for the patient. While this avoidance may help to reduce patients' anxiety in the short term, it ultimately reinforces their fear of recurrent cardiac events and perpetuates the cycle of increasing disability. Activity pacing and other adaptive illness management techniques, such as energy conservation strategies, may benefit individuals with chronic physical limitations to reduce symptom burden, overcome their fears, and work toward desired life goals (Caudill, 2001; Turk & Gatchel, 2002).

## **Anxiety Comorbid with Asthma**

Asthma, a chronic respiratory disease which affects approximately 20 million Americans, is a lung disorder characterized by obstruction of airflow. The underlying causes for airflow obstruction are bronchoconstriction, a narrowing of the small airways through contraction of the muscular walls of the bronchioles, and inflammation, which results in swelling of the mucous membranes and an increase in mucous secretion into the airway lumen. Although airway inflammation and bronchoconstriction are normal reactions of the immune system in response to serious infection, the reasons for airway hyperactivity and disproportionate inflammatory response seen in asthmatic patients, even in the absence of infection, remain unclear. Researchers have identified multiple stimuli that incite asthma, including allergens, pharmacologic effects, air pollution, occupational factors, respiratory infections, and perhaps emotional stress (McFadden, 2005). The incidence of asthma appears to be increasing in both the developing and developed world (Eder, Ege, & von Mutius, 2006). The annual economic costs to our nation related to asthma are substantial, with 14.7 billion dollars in direct healthcare costs, much of which is accounted for by prescription drug expenditures. Indirect costs including productivity losses add another \$5 billion (American Lung Association, 2009).

Anxiety disorders, especially panic symptoms, are common among individuals with asthma, with previous research confirming high rates of comorbidity in community and clinical samples (Goodwin, Jacobi, & Thefeld, 2003; Goodwin, Olfson et al., 2003; Heaney, Conway, Kelly, & Gamble, 2005; Katon, Richardson, Lozano, & McCauley, 2004). For example, in a large-scale epidemiological study of 4,181 adults in Germany, 21% of individuals with severe asthma met criteria for a current DSM-IV anxiety disorder, compared to 8.7% of participants without asthma (Goodwin, Jacobi, et al., 2003). Accumulating evidence further suggests that respiratory disease such as asthma significantly increases the likelihood of experiencing panic attacks (Goodwin & Pine, 2002), and there appears to be a bidirectional, dose-response relationship between asthma symptoms and likelihood of having anxiety disorder (Goodwin, Jacobi, et al., 2003; Hasler et al., 2005). These associations are pronounced among smokers and women (Hasler et al., 2005; Tovt-Korshynska, Dew, Chohey, Spivak, & Lemko, 2001).

## ***Hypotheses for Comorbidity Between Asthma and Anxiety Disorders***

Various theories have emerged in the last decade to explain the pathophysiological, environmental, cognitive, and behavioral mechanisms linking asthma to anxiety, and most notably, panic disorder (Katon et al., 2004). Correlational and experimental investigations of biological factors indicate that adult patients suffering from asthma with panic disorder or self-reported anxiety are more likely to perceive worse asthma symptoms such as breathlessness; yet, despite this association, researchers have failed to demonstrate that anxiety relates to pulmonary function indices, including peak-flow variability (Janson, Bjornsson, Hetta, & Boman, 1994), vulnerability to a 35% CO<sub>2</sub> challenge (van Beek et al., 2003), bronchial responsiveness to histamine (PC<sub>20</sub>; Van Peski-Oosterbaan, Spinhoven, Van der Does, Willems, & Sterk, 1996), or forced expiratory volume in one second (FEV<sub>1</sub>; Rimmington, Davies, Lowe, & Pearson, 2001). At the same time, other data suggest that psychosocial variables, and in particular depression, may play a role in pulmonary function (Ritz & Steptoe, 2000; Schmalig, McKnight, & Afari, 2002). Further research is necessary to elucidate these complex relationships and biological pathways.

In their 21-year prospective study, Goodwin, Fergusson, and Horwood (2004) reported that adolescents and young adults with asthma are at increased risk having panic attacks and other anxiety disorders; however, these associations do not remain statistically significant after controlling for confounding factors. According to the authors, asthma and such psychiatric morbidity may not necessarily share a direct causal relationship but rather may be connected through other common factors that need further exploration.

Environmental variables that may influence the development of both asthma and anxiety disorders include low socioeconomic status, urban residence, parental smoking, and stressful life events (Creer, Bender, & Lucas, 2002; Goodwin, 2003). For example, in a study of individuals of diverse socioeconomic status, Rumbak, Kelso, Arheart, and Self (1993) observed that 51% of indigent, asthmatic patients noted that their acute asthma attacks were frequently triggered by anxiety compared to only 19% of patients who were privately insured and not indigent. Similarly, asthmatic patients with less education report greater anxiety symptoms versus those with more years of school attendance (Centanni et al., 2000). Finally, investigators have found a greater prevalence of respiratory disorders in first-degree relatives of individuals with panic, though whether this finding indicates a genetic diathesis or environmental influence remains unclear (van Beek, Schruers, & Griez, 2005).

Previous research also indicates that cognitive attributions, or catastrophic thoughts, are a potential salient mediator in the bidirectional relationship between asthma and anxiety. Ley (1987; 1989) discussed the role that fear of dyspnea and of related somatic symptoms caused by hyperventilation play in the etiology of panic disorder. Considering that the primary symptoms of asthma include unpredictable constriction of the airway and breathlessness, cognitive perceptions of this physiological experience would intuitively predict the likelihood of developing an anxiety

disorder. Indeed, investigators have found that asthmatic patients with greater fear of bodily sensations (as measured by the Anxiety Sensitivity Index) were more likely to qualify for panic disorder (Carr, Lehrer, Rausch, & Hochron, 1994). Catastrophic cognitions also significantly predict illness-specific and generalized panic fear among individuals with asthma, even when controlling for pulmonary function variables (Carr, Lehrer, & Hochron, 1995). Tests of histamine-induced bronchoconstriction show that anxious patients perceive worse symptoms of breathlessness (Spinhoven, van Peski-Oosterbaan, Van der Does, Willems, & Sterk, 1997) and greater discomfort of breathing, increasing the need for use of bronchodilators independent of actual bronchial closing (Nouwen, Freeston, Cournoyer, Deschesnes, & Boulet, 1994). In sum, these findings underscore the need to target oversensitivities to pulmonary sensations with cognitive restructuring and/or modified interoceptive exposure (see below) as part of treatment of anxiety secondary to asthma.

Healthy lifestyle behaviors, such as compliance with taking inhaled medications, monitoring respiratory symptoms, and attending medical appointments regularly, are essential to maintaining effective asthma control. Whereas some researchers have found that anxiety is associated with poor compliance and asthma management (Cluley & Cochrane, 2001; Lavoie et al., 2005; Smith et al., 2005), others have failed to demonstrate such relationships (Bosley, Fosbury, & Cochrane, 1995; Lavoie et al., 2006). Comprehensive understanding of self-care behaviors is another indicator that predicts good asthma control. However, patients with anxiety are less knowledgeable about self-management practices for acute respiratory episodes (Kolbe, Vamos, Fergusson, Elkind, & Garrett, 1996). Noncompliance with treatment regimens and anxiety disorders compromise a variety of asthma outcomes. More specifically, asthmatic individuals with panic disorder and related anxiety symptoms are more likely to visit the emergency department, to be admitted to the hospital and have longer stays, and to receive prescriptions for oral corticosteroids, which may cause side effects, exacerbating the physiological stress response (Adams, Boath, Homan, Campbell, & Ruffin, 2001; Carr, 1998; Dahlen & Janson, 2002; Kolbe, Fergusson, Vamos, & Garrett, 2002). Adapting anxiety treatment to include cognitive-behavioral strategies for improving adherence to disease management protocols may not only reduce psychological distress but also enhance asthma control.

### *Effect of Anxiety on Quality of Life Related to Asthma*

As in the case of cancer and coronary artery disease, adults with asthma report poorer quality of life, which corresponds to frequent mental distress (Strine, Ford, Balluz, Chapman, & Mokdad, 2004), higher dosage of inhaled corticosteroids (Bonala et al., 2003), increased visits to primary care physicians (Feldman, Lehrer, Borson, Hallstrand, & Siddique, 2005), as well as limited ability to work and perceived lack of control over health (Adams et al., 2004). Using the PRIME-MD to diagnose DSM-IV disorders in 504 consecutive patients from an asthma clinic,

Lavoie et al. (2006) found that patients with anxiety disorders are more likely to report greater bronchodilator use and worse asthma-related quality of life across four domains: activity limitation, asthma symptoms, emotional distress, and management of environmental triggers. One potential explanation for these relationships is that individuals with asthma fear situations that may trigger symptoms of asthma and panic, potentially initiating a cycle of avoidance behaviors that lead to greater social isolation, distress, and functional limitations. Therefore, cognitive-behavioral interventions designed to alleviate anxiety for this patient population may be augmented by addressing phobic avoidance and various domains of asthma-related quality of life (Deshmukh, Toelle, Usherwood, O'Grady, & Jenkins, 2007).

### ***Psychological Interventions for Anxiety Comorbid with Asthma***

Psychological interventions for asthma generally include psychotherapy, patient education programs, and self-management interventions (Bender & Creer, 2002). Only one randomized controlled trial to date has tested a cognitive-behavioral therapy intervention within a sample of patients with coexisting asthma and panic disorder (Ross, Davis, & MacDonald, 2005), demonstrating notable acute treatment effects for both conditions but only long-term reduction in anxiety outcomes. A Cochrane review (Yorke, Fleming, & Shuldham, 2006) of psychological interventions for adults with asthma identified 15 randomized controlled trials, 9 of which incorporated a variety of relaxation techniques and 3 of which utilized cognitive-behavioral therapy. Overall, the usefulness of these treatments for anxiety symptoms and control of asthma remains inconclusive due to the methodological weaknesses and lack of consistency in outcome measures and types of interventions. Cognitive-behavioral therapy, however, did significantly improve quality of life in the patient population. These findings draw attention to the marked gap in high-quality efficacy studies aimed at comorbid anxiety and asthma.

### ***Treatment Complications for Anxiety Comorbid with Asthma***

The process of diagnosing anxiety disorders is understandably challenging for clinicians working with asthmatic patients and may lead to inadequate treatment for either or both conditions. Given that the symptoms of panic disorder in particular mimic many medical conditions, including asthma, patients with this psychological condition are likely to undergo multiple diagnostic tests and utilize healthcare services (Roy-Byrne, Craske, & Stein, 2006). When the anxiety and respiratory condition are comorbid, accurate detection becomes even more problematic, yet essential for appropriate treatment planning.

Considering the paucity of data on the use of cognitive-behavioral therapy for anxiety associated with asthma, mental health clinicians have no clear guidelines

and must discern how best to adapt standard CBT treatments for patients with respiratory conditions. Recognizing this deficit in the treatment literature, authors have questioned the utility of empirically validated panic control therapy and interoceptive exposure for patients with asthma (Deshmukh et al., 2007; Feldman, Giardino, & Lehrer, 2000). More specifically, as in the case of coronary heart disease, the optimal method for desensitizing a patient systematically to anxiety-provoking triggers, which often include internal somatic sensations that may aggravate the medical condition, remains unclear (Carr, 1998; Deshmukh et al., 2007).

A third complication in the management of anxiety comorbid with asthma is the lack of attention to medical adherence issues in traditional cognitive-behavioral treatment protocols. Many chronic illnesses, such as asthma, diabetes, and HIV, require lifelong management of medications, diagnostic testing, and follow-up care. Moreover, as noted earlier, poor adherence to medical regimens is often related to psychological morbidity that negatively impacts asthma outcomes. Therefore, mental health clinicians play a pivotal role in the multidisciplinary team for helping patients identify and address obstacles to maintaining good compliance and other salient health behaviors, for the dual purpose of reducing anxiety symptoms and improving asthma control, which interact and influence one another (Bender & Creer, 2002).

### ***Resolving Treatment Complications for Anxiety Comorbid with Asthma***

To aid in the diagnosis of anxiety comorbid with asthma, researchers have identified a number of indicators to consider during evaluation. First, the most reliable method for discriminating anxiety symptoms from asthma include pulmonary function tests, such as measures of peak flow and FEV<sub>1</sub> (Feldman et al., 2000). In addition, as is true with any other psychiatric disorder, conducting a comprehensive assessment of the situational triggers and the presence of a family history of anxiety may clarify diagnosis. Beyond these standard techniques, the types and clusters of symptoms associated with each condition may also assist in the differential diagnosis. For example, panic attacks typically have rapid onset and reach their peak within 10 minutes while this is generally not the case for asthma. Furthermore, in a study comparing asthmatic patients to a cohort-matched group of individuals with panic disorder, Schmaling and Bell (1997) found that reports of panic-fear and hyperventilation were strongly associated with panic disorder, while airway obstruction symptoms, such as wheezing, mucous congestion, and coughing, were more predictive of asthma attacks. These three symptoms demonstrated high sensitivity (> 0.90) and specificity (> 0.70) in distinguishing the patients groups, thereby serving as useful tools for identifying when patients are experiencing one or both types of attacks (Feldman et al., 2000; Schmaling & Bell, 1997).

Again, as discussed previously with cancer and coronary heart disease, the approaches for tailoring cognitive-behavioral therapy for anxiety depend on the

nature of the medical condition. Rather than emphasizing graded in-vivo or interoceptive exposures that potentially place a patient at risk of an asthma attack, Deshmukh et al. (2007) suggest that behavioral interventions for anxiety should address fears of asthma stimuli and related avoidance behaviors through a combination of symptom monitoring, problem-solving techniques, appropriate use of asthma medications, and development of an asthma action plan for managing attacks. The authors argue that these behavioral methods will assist patients with differentiating anxiety from asthma and bolster confidence in the ability to cope effectively with asthma symptoms and dyspnea, thereby minimizing triggers of panic.

Similarly, Feldman et al. (2000) argue that CBT must be adapted for anxiety related to asthma and that teaching patients to discriminate between panic and anxiety symptoms, including the ways that hyperventilating and wheezing are different, is essential for cognitive restructuring and appropriately treating somatic sensations. Considering the potential risks of bronchoconstriction associated with interoceptive exposure techniques (e.g., voluntary hyperventilation, production of chest tightness, and inhaling through a straw), the authors substitute HRV biofeedback, slow breathing exercises, and pursed lips breathing as safe alternatives for treatment. Also, to improve self-management of asthma and panic attacks, the authors developed a decision tree in which they use indicators of peak-flow monitoring, the presence of mucous production or coughing, and rapid onset of the attack to guide decision-making for proceeding with an action plan to resolve the asthma or anxiety symptoms (Feldman et al., 2000).

Finally, cognitive-behavioral therapy needs to be modified to target compliance with medical treatments. Simply treating adherence in isolation or anxiety in isolation is not sufficient, as these factors synergistically affect asthma control. Some of the work of our research team (Safren, Gonzalez, & Soroudi, 2007; Safren et al., 2004; Safren et al., 2009; Soroudi et al., 2008) integrates adherence interventions into standard CBT protocols designed to treat psychological conditions comorbid with medical illness. More specifically, Safren et al. (2004), Safren, Otto, and Worth (1999) developed a brief guide that includes 11 steps for patient education, motivational enhancement, and behavior change to increase medication adherence among people with HIV. Incorporating this intervention as part of several randomized studies testing the efficacy of CBT for depression in patients with HIV (and currently being tested with Type 2 diabetic patients), the authors have demonstrated positive effects for both adherence with medical treatment and mood symptom reduction (Safren et al., 2004, 2009). Rigorous cognitive-behavioral therapy trials testing similar integrated models are needed for anxiety and asthma.

## Summary and Conclusions

In this chapter, we demonstrated an approach to the assessment and treatment of anxiety disorders in medically ill populations. To illustrate these concepts, we first reviewed relevant literature pertaining to the symptoms, prevalence, correlates,

and outcome studies of anxiety comorbid with cancer, coronary heart disease, and asthma. Second, we highlighted the types of treatment difficulties mental health clinicians frequently encounter when providing cognitive-behavioral therapy to patients who have concomitant anxiety and medical conditions; lastly, we provided suggestions for resolving those complications. The following is a summary of the clinical recommendations, which may be applied more broadly to patients coping with a variety of illnesses beyond those presented, such as diabetes, HIV, and irritable bowel syndrome:

- 1) Minimize barriers for patients to access psychotherapy by scheduling sessions either in conjunction with other medical appointments or at the time when patients are less likely to be disabled by the ill-effects of medical treatments (e.g., allow sufficient time after chemotherapy infusion).
- 2) Communicate regularly with medical providers to clarify and confirm patients' diagnoses and to develop an appropriate treatment plan.
- 3) Resist mind–body dualism and employ biopsychosocial models of illness when educating patients about the complex and bidirectional relationships among their anxiety symptoms, disease, and environment.
- 4) Adapt cognitive-behavioral therapy by considering with patients the extent to which their health-related worries and fears reflect realistic concerns and tailor treatment decisions based on this assessment.
- 5) Incorporate illness, symptom, and pain management strategies, such as activity pacing techniques, to enhance physical and role functioning, if needed.
- 6) Utilize interoceptive exposure-based therapy only when patients have appropriate medical clearance and modify techniques so as not to exacerbate symptoms of the medical condition.
- 7) Assess for compliance with medical treatment regimens and integrate cognitive-behavioral strategies for improving medical adherence, when necessary.

While the above recommendations may offer assistance to clinicians working with this complex patient population, further research is needed to test the efficacy and effectiveness of psychosocial interventions with medically ill patients. Few methodologically rigorous, published studies exist regarding the psychological treatment of anxiety disorders in patients with chronic medical conditions. The development of novel therapies that are theoretically driven and empirically based, targeting the clinical features and concomitant psychosocial sequelae of physical illnesses and their treatments, will ideally enhance a number of disease, psychosocial, and healthcare-related outcomes for patients.

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# Resolving Treatment Complications in Children and Adolescents

Jamie A. Micco and Jill T. Ehrenreich

Numerous randomized controlled studies have found that cognitive-behavioral therapy (CBT) is efficacious in treating childhood anxiety disorders, including those evaluating individual or group CBT for children and adolescents with social phobia (Beidel, Turner, & Morris, 2000; Spence, Donovan, & Brechman-Toussaint, 2000), specific phobia (Silverman et al., 1999), school refusal (King et al., 1998), panic disorder (Pincus, Ehrenreich May, Whitton, Mattis & Barlow, 2009), and PTSD (Cohen, Deblinger, Mannarino, & Steer, 2004), as well as for heterogeneous groups of children diagnosed with separation anxiety disorder, generalized anxiety disorder, and/or social anxiety disorder (Kendall, 1994; Kendall et al., 1997).

Kendall's Coping Cat program (Kendall, 1994), designed for anxious children ages 9–13, is perhaps the most well-known CBT protocol for childhood anxiety. Of the children randomized to the individual Coping Cat treatment, 64% no longer met criteria for their primary anxiety disorder at post-treatment, compared to only 5% of the children in the waitlist control group (Kendall, 1994). In a second randomized controlled trial (Kendall et al., 1997), similar results were obtained, with 53% no longer met criteria for their primary anxiety disorder at post-treatment, compared to 6% of the children in the waitlist control group. For children continuing to meet criteria for their primary anxiety disorder, the majority experienced significant improvement in the severity of their disorder. Treatment gains were largely maintained over long-term follow up (Kendall et al., 1997; Kendall, Safford, Flannery-Schroeder, & Webb, 2004).

A recent meta-analysis of 20 randomized controlled trials of CBT for children and adolescents with anxiety disorders found a large pre- to post-treatment effect size ( $d=0.94$ ) when comparing children who received CBT to controls (Ishikawa, Okajima, Matsuoka, & Sakano, 2007). Further, a review of the longer-term treatment outcomes of CBT for childhood anxiety reported that overall, treatment gains tend to be maintained for at least one year post-treatment (Compton et al., 2004).

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However, despite these studies supporting the use of CBT with anxious children, a sizeable minority of children and adolescents with anxiety disorders fail to respond adequately to CBT (see Cartwright-Hatton, Roberts, Chitsabesan, Fothergill, & Harrington, 2004; Velting, Seltzer, & Albano, 2004). Consequently, there is a great need to increase our understanding of why CBT is not effective for a sub-sample of anxious children and how treatment can be tailored to meet the needs of these non-responders.

This chapter will describe the most commonly observed complications that arise in CBT of childhood anxiety disorders. Specifically, we will review the current literature examining the degree to which treatment of children with anxiety is influenced by comorbid disorders, parenting behaviors, parental anxiety, sociodemographic characteristics, and parent-child symptom agreement. We will also provide evidence-based recommendations for how standard CBT for child anxiety can be modified to address these potential complicating factors, followed by detailed case examples drawn from our experience working with children at the Center for Anxiety and Related Disorders at Boston University and Massachusetts General Hospital.

## **Comorbidity**

Clinically anxious children and adolescents who present for CBT often meet criteria for additional disorders, including major depression and dysthymia (Kendall, 1994; Strauss, Last, Hersen, & Kazdin, 1988), attention-deficit/hyperactivity disorder (ADHD; Last, Hersen, Kazdin, Orvaschel, & Perrin, 1991), and oppositional-defiant disorder (ODD; Verduin & Kendall, 2003). Cognitive-behavioral therapists who use empirically supported treatments for childhood anxiety disorders, such as Kendall's (1994) Coping Cat manual, March and Mulle's (1998) CBT for obsessive compulsive disorder, and Beidel and colleague (2000) treatment for social anxiety disorder, may find that these treatments need to be adapted to maximize treatment gains in anxious children with comorbid depressive or externalizing disorders. In this section, we present data on the prevalence of the most common disorders that co-occur with anxiety in children and adolescents, including depression, ADHD, and ODD. For each type of comorbidity, we review common patient characteristics and research showing how comorbidity affects response to CBT. We then describe adaptations that can be made to CBT for anxiety disorders that help to address the needs of children with comorbid conditions.

## **Treating Comorbid Anxiety and Depressive Disorders**

### ***Prevalence***

Depressive disorders are likely to co-occur with anxiety disorders at a rate greater than chance expectations in children and adolescents (Seligman & Ollendick,

1998). In a prospective community study of 785 11-year-olds (Anderson, Williams, McGee, & Silva, 1987), over 15% of the participants who met criteria for an anxiety disorder also met criteria for a depressive disorder. When these children were followed up at age 15, 13% of the anxiety-disordered adolescents had a comorbid depressive disorder (McGee et al., 1990). Another community study found much higher rates of comorbid anxiety and depression in adolescents (Kashani & Orvaschel, 1988). Specifically, 9 out of 13 adolescents (69%) meeting criteria for an anxiety disorder, from a sample of 150 adolescents, also had an affective disorder.

Studies employing clinical samples have found varying rates of comorbidity between anxiety and depressive disorders in children. In a sample of children (ages 8–13) with primary depression, 31.5% also met criteria for an anxiety disorder (Kovacs, Gatsonis, Paulauskas, & Richards, 1989). Meanwhile, two studies of children and adolescents with primary anxiety disorders found that approximately 30% also have clinical depression (Kendall, 1994; Strauss et al., 1988). However, these rates were lower in a study conducted by Verduin and Kendall (2003). Of the 199 children (ages 8–13 years) in this study with primary social anxiety disorder, generalized anxiety disorder, or separation anxiety disorder, 4.5% also met criteria for major depressive disorder, while 8.5% had dysthymic disorder. Similarly, in a study of group treatment for anxiety disorders in children ages 7–16 years (Rapee, 2003), only 6.1% also had a mood disorder.

## Patient Characteristics and Treatment Response

A number of studies have found that children with comorbid anxiety and depressive disorders tend to be older and more functionally impaired than children with anxiety disorders or depressive disorders alone (Bernstein, 1991; Brady & Kendall, 1992; Manassis & Hood, 1998; Manassis & Menna, 1999; Masi, Favilla, Mucci, & Millepiedi, 2000; Strauss et al., 1988). For instance, Strauss et al. (1988) found that children and adolescents (5–17 years old) with comorbid anxiety and major depressive disorders had more severe and interfering anxiety symptoms than anxious participants without depression (Strauss et al., 1988). In another study by this group, anxiety and comorbid depression in school-aged children was associated with decreased popularity with peers (Strauss, Lahey, Frick, Frame, & Hynd, 1988). Research also suggests that depression is more likely to co-occur with generalized anxiety disorder and social anxiety disorder than other anxiety disorders (Last, Strauss, & Francis, 1987; Manassis & Menna, 1999; Verduin & Kendall, 2003). In a study of 58 children and adolescents with primary generalized anxiety, more than half of the sample had a comorbid depressive disorder (Masi, Mucci, Favilla, Romano, & Poli, 1999). In addition, Verduin and Kendall (2003) found that while 17.4% of children with generalized anxiety disorder and 15% with social anxiety disorder also met criteria for either major depression or dysthymia, only 2% of children with separation anxiety disorder also had a depressive disorder.

Despite the common co-occurrence of anxiety and depression in children and adolescents, only a few studies have examined the degree to which this comorbidity affects treatment outcome. Berman et al. (2000) found that symptoms of

depression significantly predicted worse treatment response in anxious children receiving CBT (Berman et al., 2000). On the other hand, Rapee (2003) found that among children (ages 7–16) receiving group CBT for primary anxiety, those with comorbid disorders (including mood and externalizing disorders) had similar levels of post-treatment improvement as those without a comorbid disorder. However, at 1-year follow-up, the children with comorbid disorders showed a slight symptomatic increase on parent-report measures whereas the children without comorbidity continued to maintain their treatment gains (Rapee, 2003). This suggests that anxious children with comorbid disorders may need extra support in maintaining improvements made in CBT.

## **Modifications for Treating Children with Comorbid Depression**

While children with anxiety and depression both experience negative affect, children with depression also have low levels of positive affect (Watson & Clark, 1984; Watson & Kendall, 1989). Depressed children with anhedonia and low energy often avoid activities with peers, which in turn gives them little opportunity to experience positive affect associated with outside activities. In addition, the hopelessness, low self-efficacy, and low energy associated with childhood depression may affect willingness and motivation to engage in a structured treatment like CBT. Thus, anxious children with depression may benefit from the inclusion of behavioral activation and scheduling of pleasant events early on in the treatment. Therapists should help these children make a list of activities that are enjoyable and likely to produce a sense of mastery; then, between sessions, the children engage in at least one activity from the list each day. As children experience greater positive affect, they begin to understand that their actions have an influence on their mood, which may produce a greater willingness to discover new ways to cope with their anxiety.

In addition, cognitive restructuring should be modified to include cognitions associated with depression. While anxious children tend to overestimate threat in situations (Bögels & Zigterman, 2000), children with depression tend to selectively focus on negative aspects of events (Weems, Berman, Silverman, & Saavedra, 2001) and endorse thoughts of personal loss and failure (Schniering & Rapee, 2004). As such, anxious–depressed children benefit from learning ways to challenge their negative self-evaluations about past events in addition to their future-oriented worries. Hypothesis testing, or gathering evidence for or against specific beliefs, can also help children generate adaptive self-talk. For instance, a child who declares, “I never get good grades,” after not doing well on one test would be asked to keep track of all his grades over the course of the week in order to evaluate the veracity of his belief. Assuming that he earns a range of grades, his therapist can help him construct a new coping statement, such as, “I do well in school many times.”

Children with depression (particularly when it is comorbid with social anxiety) may have social skills deficits that need to be addressed in treatment (Segrin, 2000). Social skills training, tailored to the child’s specific areas of difficulty (such as maintaining eye contact, starting conversations, etc.), is a key component of CBT for

child depression (Stark, Sander, Yancy, Bronik, & Hoke, 2000). Children with anxiety and depression also benefit from learning social problem-solving skills that consist of identifying a problem, generating solutions to the problem, and selecting the most effective solution. Problem-solving goals include increasing involvement in pleasurable activities (which addresses the depressive symptoms) and enacting a coping plan for engaging in anxiety-provoking situations (Kendall, Kortlander, Chansky, & Brady, 1992).

### *Case Example*

Jake was a 13-year-old boy with a primary diagnosis of generalized social anxiety disorder and a secondary diagnosis of dysthymic disorder. His parents were divorced, and he lived with his mother and stepfather, although he frequently spent time with his father as well. By the time that he began CBT at our clinic, Jake was avoiding nearly all social situations with peers or unfamiliar adults. He engaged in no extracurricular activities; after school, he walked home and spent the afternoon and evening playing video games or watching TV by himself. He was failing most of his classes in eighth grade. He reported that he had felt depressed “for years,” while his mother described him as “always irritable.”

At the first session of individual CBT, Jake indicated that he did not have much confidence that treatment could help him. He and his therapist discussed the advantages (“I could feel less unhappy,” “My parents will stop asking me what’s wrong”) and disadvantages (“It will be hard and it might not work”) of engaging in treatment, after which Jake decided that, “it’s worth a try, I guess.” The therapist described how his avoidance of social situations led to a short-term decrease in his anxiety, but also resulted in a longer-term lack of confidence in his ability to handle social situations and an increase in depression because he did not have the opportunity to receive positive social reinforcement. The therapist also addressed Jake’s depression by teaching him that his choice about how to respond to difficult situations had a big effect on his mood; thus, he learned that he had some control over his feelings. For instance, if he got a bad grade on an assignment, he could choose to do nothing (and feel sad and angry), or he could ask his teacher for extra help and the opportunity to redo the assignment, which would likely boost his mood and increase his self-efficacy. Jake and the therapist also made a list of activities he most enjoyed, and he was asked to pick one pleasant activity to do each day.

Jake learned to identify his automatic thoughts and his most common cognitive errors, including mind reading (assuming others thought he was “dumb”), black and white thinking (“you’re either popular or a loser”), and mental filter (focusing on the negative at the expense of the positive). He learned to challenge his anxious and depressed thoughts with coping self-statements. For instance, when he was fearful he would “say the wrong thing” during a class presentation, he challenged this thought by reminding himself that, “Everyone makes mistakes sometimes – if it happens to me, I’ll make the best of it.”

Situational exposures were designed to gradually address Jake’s fear of social interactions while maximizing the likelihood that he would experience pleasure and

mastery after completing each exposure. Items on his Fear and Avoidance Hierarchy (FAH) included asking an acquaintance to the movies, going to brunch with his family in a crowded restaurant, talking to a new person at lunch, and signing up for a confidence-building activity (martial arts classes). Jake was taught problem-solving skills to determine ways in which he could handle himself if social interactions did not go as planned. In addition, his therapist worked with him to enhance his social skills in conversations, particularly varying his tone of voice, asking open-ended questions, and maintaining eye contact. He first practiced these skills in role-plays with the therapist, then with “confederates” at the clinic, and finally as situational exposures over the course of the week.

Jake was regularly talking to other boys at school and seeing acquaintances on the weekends by the time he finished eighth grade. However, he experienced a setback in his anxiety and depression when summer vacation began; without regular opportunities to talk with peers at school, he engaged in fewer exposures. In addition, he was not involved in a structured activity for the summer, resulting in him spending a lot of time by himself. Further, he complained that his parents worked long hours with little time to spend with him. Thus, his therapist strongly encouraged Jake’s parents to enroll him in an extracurricular summer activity and to devote more time to one-on-one activities with him to promote positive family interactions. After working on these goals with his parents, Jake again felt less depressed and more motivated to call friends from school.

At the beginning of ninth grade, a reward system was developed with Jake and his parents to increase his motivation to complete schoolwork; he could earn points towards rewards (i.e., paintballing with his friend, a DVD, etc) in exchange for completing homework on time. Jake began to earn As and Bs in his classes, and after two weeks of the reward plan, he felt that he no longer needed extrinsic rewards to spend time on his schoolwork. He reported feeling more confident in his academic skills. By the end of treatment, in the fall of ninth grade, Jake reported few symptoms of depression. Although he continued to experience anxiety about talking to new peers, he had made several good friends and regularly saw them outside of school. He felt more supported by his parents, who set aside time each week to check in with him. At his termination session, he and his therapist reviewed the skills he had learned during his treatment as part of his relapse prevention plan.

## **Treating Comorbid Anxiety and Externalizing Disorders**

### ***Prevalence***

Epidemiological and clinical studies have found a high level of comorbidity between anxiety disorders and ADHD and/or ODD in children and adolescents. Bird et al. (1988) found that in a community sample of 386 children (ages 4–16), 9.3% met DSM-III criteria for an anxiety disorder and ODD or conduct disorder (CD), while

approximately 6% had co-occurring anxiety and attention-deficit disorder (Bird et al., 1988). Among studies using DSM-IV criteria, presence of ADHD in children with anxiety disorders ranged from 0% to 16.7%, and presence of ODD or CD ranged from 7.9% to 33.3% (Angold, Costello, & Eklani, 1999).

In children presenting for treatment for anxiety disorders, comorbidity with externalizing disorders is quite high. Verduin and Kendall (2003) found that in clinically anxious children ages 8–13, 17.6% met criteria for ADHD and 9.5% met criteria for ODD; there was no significant difference in rate of comorbidity among diagnoses of social anxiety disorder, separation anxiety disorder, or generalized anxiety disorder, although there was greater comorbidity between anxiety and externalizing disorders among boys. Comparable rates of comorbidity were found by Last et al. (1987) in a study of 91 children referred to an anxiety disorders clinic. They found that 1% and 9% of the children were diagnosed with an anxiety disorder and ADHD or ODD, respectively (Last, Hersen, Kazdin, Finkelstein, & Strauss, 1987). In another study, they found that 13% of 381 children with primary anxiety disorders also had ADHD (Last et al., 1991).

## **Patient Characteristics and Treatment Response**

By and large, children with comorbid anxiety and externalizing disorders tend to be younger and more impaired than children with anxiety alone (Anderson et al., 1987; Bird, Gould and Staghezza, 1993; Manassis & Hood, 1998; McGee et al., 1990; Strauss, Lease, Last, & Francis, 1988). In a study of children and adolescents with primary DSM-III overanxious disorder, 61% of children aged 5–11 years were also diagnosed with an externalizing disorder, compared to only 15% of those between 12- and 19-years old (Strauss et al., 1988). Children with comorbid anxiety and externalizing disorders tend to have more severe symptomatology, with greater irritability, mood lability, and frequency of emotional outbursts (Kashani, Deuser, & Reid, 1991). In addition, mothers' ratings of conduct problems in clinically anxious children significantly correlate with level of impairment as measured by the Global Assessment of Functioning (GAF; Manassis & Hood, 1998).

Several studies have examined the degree to which comorbid externalizing disorders impact CBT outcome for child anxiety. Kendall, Brady, and Verduin (2001) found that anxious children with pre-treatment externalizing disorders improved to the same degree after manualized CBT as children without externalizing disorders. Indeed, 68.4% of children with anxiety alone were free of their principal anxiety disorder diagnosis at post-treatment, compared to 70.6% of children with anxiety plus ADHD or ODD. In addition, children with pre-treatment comorbid externalizing disorders also had fewer diagnoses of ADHD and ODD at post-treatment, suggesting that treatment gains generalized beyond anxiety. Notably, however, children who continued to meet criteria for a comorbid disorder at post-treatment were less likely to experience remission of their pre-treatment principal anxiety disorder (Kendall et al., 2001). In a follow-up to this study, Flannery-Schroeder et al. (2004) compared

19 anxious children with ADHD or ODD/CD to 19 anxious children without this comorbidity and found that both groups maintained comparable levels of improvement in their principal anxiety disorder 7 years post-treatment, although parents of the anxiety plus externalizing group of children rated their children as having more externalizing symptoms than children with anxiety alone (Flannery-Schroeder et al., 2004).

In contrast, Costin et al. (2002) piloted an eight-session CBT group treatment for anxiety with five boys (ages 9–12) who also were diagnosed with both ADHD and ODD. This group included a separate parent group that focused on psychoeducation and strategies for managing child anxiety, although explicit training for handling oppositional behavior was also included in the parent sessions. At post-treatment, the boys did not experience improvements in self-ratings of anxiety or parent-ratings of internalizing and externalizing symptoms, suggesting that more severe comorbidity may interfere with the efficacy of brief CBT for anxiety (Costin et al., 2002).

## **Modifications for Treating Comorbid ADHD and ODD**

When treating anxious children with comorbid ADHD, we have found that several modifications to standard CBT are typically required to ensure children's engagement in treatment. First, children with ADHD may learn CBT skills more effectively in the context of shorter individual sessions (i.e., dividing one manualized session into two sessions), with brief breaks within sessions. Second, particularly for children with hyperactive or combined types of ADHD, it may be necessary to implement a behavioral plan within sessions to reinforce on-task behavior and discourage inappropriate behavior. For example, children can earn stickers for remaining in their seat for 5- or 10-minute intervals of time; at the end of session, they can pick a small prize for earning a specified number of stickers. Third, breaking CBT principles and skills down into simple "bullet points," and writing down what was learned at the end of each session, can be particularly helpful for inattentive children. Homework assignments may also need to be modified to reduce the amount of written work children need to produce between sessions. Fourth, when teaching problem-solving skills to anxious children with ADHD, therapists may wish to especially focus on problems the children encounter at school as a result of their inattentiveness and/or hyperactivity. In our experience, children with anxiety and ADHD tend to be easily overwhelmed by the demands of school and extracurricular activities, to the point that they worry a great deal about their performance in these areas. When teaching problem-solving skills, these children appear to benefit from learning how to generate and implement solutions to organizational difficulties and problems with time management.

For children with comorbid ODD or conduct problems, it is first important to determine the degree to which their oppositional behavior is triggered by anxiety or fear, as this will guide the therapist in providing affective education to the child.

For example, imagine an 8-year-old girl with separation anxiety disorder who hides in her room and tells her parents she “hates” them as they get ready to take her to school. During treatment, the girl’s therapist would first teach her to distinguish between nervous and angry feelings; in later sessions, the girl would then practice using coping skills for anxiety (i.e., relaxation techniques, deep breathing, positive self-talk) as soon as she noticed she was having nervous feelings, which could then “short-circuit” the pathway between anxiety and subsequent angry feelings.

When teaching anxious and oppositional children ways to manage physiological symptoms of anxiety (i.e., relaxation), the therapist should encourage the application of these skills to times when the children feel angry. Modifications to cognitive components of the treatment are based on findings that children with ODD tend to have difficulty identifying social cues and misattribute hostile intent to their peers or parents (Crick & Dodge, 1994). Consequently, these children will need assistance in challenging their assumptions about others’ intentions, which can be done in the context of cognitive restructuring. The tendency for children with ODD to select aggressive solutions to problems (Lochman & Dodge, 1994) should also be addressed when children learn problem-solving skills or through the addition of a social skills training module.

The inclusion of parents in the treatment of comorbid anxiety and ODD is essential. In particular, parents benefit from learning how to implement rewards and consequences at home in order to reinforce both brave and non-aggressive behaviors. The therapist may model a behavioral plan for parents by setting ground rules for behavior during sessions (i.e., using respectful language) and rewarding children’s adherence to the rules. Selective ignoring skills can be used for both anxious and oppositional behaviors; in other words, parents may be encouraged to practice diverting their attention away from excessive reassurance-seeking or acting-out behavior followed by close attention to the child as soon as he/she engages in appropriate behavior.

### *Case Example*

Daniel, a 9-year-old boy with diagnoses of separation anxiety disorder, specific phobia of vomiting, ADHD (inattentive type), and ODD, was referred for individual CBT after he had been in supportive therapy for over a year without improvement. At his diagnostic interview, Daniel’s parents reported that he had been extremely fearful of vomiting ever since he saw another child in school throw up in his classroom when he was in second grade. He avoided being separated from his parents (especially his mother) because he was intensely afraid that he would feel sick while he was by himself and would not know how to handle it. His parents told the interviewer that Daniel had become increasingly irritable and “touchy.” He “constantly” talked back to his parents when they asked him to complete homework and chores, and recently he had been screaming and throwing objects when his mother was about to leave the house. However, his parents noted that he was typically well behaved in school and had several close friends.

At his first treatment session, Daniel insisted that his mother stay with him in the room, after which he put his jacket over his head and refused to remove it. He answered the therapist's questions about school and friends with the jacket on his head, but soon after, he agreed to the therapist's suggestion that they could play a game if he took the jacket off. As they played the game, Daniel was able to answer more specific questions about his fears and worries. During the second session, he again wanted his mother with him in the room, although this time he was willing for her to wait in the waiting room after 10 minutes in order to earn a reward from the clinic's "treasure box." To maximize Daniel's attention and interest in learning CBT principles of anxiety, the therapist helped Daniel understand the difference between his thoughts, feelings, and actions when he became nervous or angry by having him play a quiz-show game that involved putting examples of each component into their appropriate categories. At the end of psychoeducational sessions, he worked with the therapist to write down three things he learned during the session, after which he earned pencils or stickers.

In addition to learning to distinguish between "nervous thoughts" and "calm thoughts" in response to situations Daniel found to be anxiety provoking, he also practiced identifying his "angry thoughts," which often occurred right after he had an anxious thought. For example, he drew a picture during session that depicted his mother dropping him off at school; the therapist asked him to include "thought bubbles" over his head in the picture. In the thought bubbles, he wrote his usual nervous thought ("If Mom leaves, I'm going to get sick") and angry thought ("It's stupid I have to stay here!"), and he and his therapist generated a new coping thought ("I've never gotten sick in school before, so I probably won't today"). Daniel's therapist also taught him a progressive muscle relaxation exercise, which he was encouraged to use whenever he noticed he was having the nervous or angry thoughts/feelings he had identified with his therapist.

When it became time to construct a fear and avoidance hierarchy (FAH) of feared situations and to begin situational and interoceptive exposures, the therapist met individually with Daniel's parents to discuss how to best implement exposure practices at home with the help of a reward plan. When Daniel completed an exposure (particularly at the beginning of this portion of treatment), he could choose from several rewards, including baseball cards and playing a game with his parents. At the same time, the therapist also worked with Daniel's parents to determine how to apply rewards to appropriate behavior at home. For example, when Daniel was able to get out of the car at school without yelling at his mother, he earned points toward something from his reward menu. After Daniel experienced some success with exposures from his FAH and earning rewards for appropriate behavior, the therapist introduced consequences for acting-out behavior to the behavioral plan. For instance, Daniel's parents removed previously specified privileges (i.e., video game time, ice cream after dinner) when he called them names or threw objects.

As Daniel reached the top of his FAH and experienced a significant decrease in his fear of vomiting and being away from his parents, his parents found that they no longer needed the behavioral plan to manage Daniel's oppositional behavior.

Overall, Daniel became more confident in his ability to handle his fears on his own, and he became more certain that it was not dangerous to feel nervous or sick (with the help of cognitive restructuring and interoceptive exposure). His parents noticed that when Daniel began to feel less nervous being by himself at school, he seemed to have greater “emotional energy” left over at the end of the day to handle minor frustrations or disappointments at home.

## Parenting Behaviors and Parental Anxiety

While comorbid disorders are frequently a challenge in the treatment of primary anxiety disorders in children and adolescents, parenting behaviors and parental anxiety may also need to be addressed during CBT in order for treatment to be maximally effective. Multiple studies demonstrate that a family history of anxiety symptoms is a significant risk factor for the development of anxiety among offspring (e.g., Beidel & Turner, 1997; Kashani & Orvaschel, 1990; Kendler, Neale, Kessler, Heath, & Eaves, 1992). These studies reveal that, in general, anxiety disorders proliferate within families. While this indicates that parental anxiety is likely related to a child’s anxiety through biological mechanisms, the risk conferred genetically seems to be a somewhat generalized one that may be common to many forms of internalizing psychopathology (Andrews, 1996). However, family studies appear to support a rather specific linkage between the type of anxiety disorder observed within families, indicating that environmental factors may interact with biological mechanisms to influence the specific form of anxiety observed between parents and their children (Beidel & Turner, 1997; Eley, 1997; Rapee, 2002).

Few environmental factors in the development of anxiety have received as much research and clinical attention as parenting behavior. Many authors have argued that the manner in which a parent responds to their child’s emotions and directs their behavior is central to the likelihood of that child developing anxiety (Rapee, 2002). Two parenting constructs: *overprotection/control* and *low warmth/high criticism* have dominated the theoretical and empirical parenting literature in terms of demonstrable associations with childhood anxiety. However, other parenting behaviors, including parental modeling of anxiety and a parent’s engagement in catastrophizing or similar communications that enhance a child’s fear (Barrett, Rapee, Dadds, & Ryan, 1996; Beidel & Turner, 1998) may also be relevant to child anxiety and its maintenance over time (Fisak, Negy, & Ehrenreich, 2008).

Some models of anxiety development (Chorpita, Brown, & Barlow, 1998; Ginsburg & Schlossberg, 2002; Rubin & Mills, 1991) suggest that family-related biological and environmental risks may interact when high levels of anxiety in parents interfere with effective parenting, leading to the overuse of strategies that promote child anxiety symptoms. With these models in mind, in this section we will explore the existent literature on the role of parenting behaviors in the development of child anxiety disorders. We will then present some family-based modifications

to CBT for childhood anxiety that address parental and family factors in treatment. Following this, a case example demonstrating the interaction of several parental variables in a child anxiety treatment context will be discussed.

## Parenting Behavior and Child Anxiety

### *Warmth and Control*

Studies of both clinical (Hudson & Rapee, 2001; Moore, Whaley, & Sigman, 2004) and community (Ehrenreich & Gross, 2002; Woodruff-Borden, Morrow, Bourland, & Cambron, 2002) samples support that at least one caregiver, typically mothers, may exhibit a greater frequency of controlling behavior and lower amounts of warmth (or a higher degree of criticism) during interactions with their anxious children. A number of studies have used observational methods to examine these behaviors across various interaction tasks with the families of anxious children (see Wood, McLeod, Sigman, Hwang, & Chu, 2003, for a review). For example, Siqueland, Kendall and Steinberg (1996) analyzed the interrelationships between maternal control, maternal warmth, and childhood anxiety using both self-report and observational techniques. Siqueland et al. (1996) observed parents in dyadic interactions with their child, plus a triadic interaction with both parents and the child, while discussing a "hot topic," identified by the parents as a prevalent and contentious family issue. Across the types of family interaction, parents of children with anxiety disorders were rated as less granting of psychological autonomy (a variable related to parental control that specifically examines the degree to which parents allow their child independent decision-making and action) than parents of non-anxious children. Although anxious mothers were rated as less granting of psychological autonomy than comparison group mothers, no significant differences in maternal warmth were found between groups (Siqueland et al., 1996). However, a replication and extension of these findings by Moore et al. (2004) found that regardless of maternal levels of anxiety, mothers of anxious children were observed to be *both* less warm and less granting of autonomy than mothers of non-anxious children. An interesting twist on such findings may be gleaned from a recent investigation by Ginsburg, Grover, Cord and Ialongo (2006), in which mothers with an anxiety disorder displayed both overcontrolling behavior and heightened criticism in a highly structured task, but only heightened criticism in a low-structure task, suggesting that situational demands may play a role in the use of these anxiety-related parenting strategies.

Parental behavior that is low in warmth and high in control may interact with a child's anxiety through several potential mechanisms. Barlow (2002) has suggested that these parenting behaviors may reinforce anxiety disorder development by diminishing a child's own sense of control. For instance, overcontrolling behavior may minimize a child's sense of control by conveying that the anxious child is incapable of handling novel or difficult situations, a notion that may be reinforced through the presentation of fewer opportunities for the child to learn how to

negotiate such situations independently (Hudson & Rapee, 2004). Parenting behaviors that communicate a low degree of warmth and a high degree of criticism may also indicate to the child that his/her caregiver may not act in a supportive manner toward him/her when difficult or threatening situations arise (Moore et al., 2004). Conversely, children with high levels of anxiety, who frequently display distress and may fail to respond to other more supportive parenting behaviors, may naturally evoke critical and controlling behaviors from their parents over time (Ehrenreich & Gross, 2002). Manassis and Bradley (1994), for example, observed that even non-anxious parents of anxious children may be conditioned to respond to their children's distress through overprotective behavior.

## Parental Modeling and Communications About Threat

Parenting influences on child anxiety have also been examined in terms of direct modeling of anxiety and communications about potential threat between parents and anxious children. Beidel and Turner (1998) suggest that observational learning between a socially phobic parental model and his/her child may contribute to the concordance rates observed for this disorder, acting partly through a process they call *information transfer*. Information transfer refers to caretakers verbally and non-verbally engaging in communications leading to a child's fear acquisition and may be typified by a parent repeatedly avoiding seemingly benign social interactions or discussing such events as anxiety-provoking in the presence of the child (Beidel & Turner, 1998). Similarly, the work of Barrett et al. (Barrett et al., 1996; Dadds, Barrett, & Rapee, 1996) indicates that family communications about the potential for threat in an anxiety-provoking situation may stimulate or encourage anxious thoughts in children with anxiety disorders, resulting in the selection of avoidant behavior more frequently than in families of children without anxiety disorders. Moore et al. (2004) identifies maternal communications that are catastrophizing, or that exaggerate the potential for negative consequences, as particularly likely to occur in parent-child interactions associated with child anxiety, regardless of whether their parent also exhibits an anxiety disorder.

A theoretical model of anxiety development presented by Craske (1999) suggests that parenting styles characterized by such communications and behavior may activate trait anxiety in offspring and subsequently reinforce patterns of increasing anxiety symptoms in children with anxiety over time. Micco and Ehrenreich (2008) indicate that one factor underlying this type of parenting behavior may be a parent's belief that his/her child is unable to cope with the demands of fear-evoking situations, particularly those that are more salient in their fear value to the child. It follows that a negative interaction cycle may persist between parents and anxious children in which a parent develops a negative belief about their child's ability to adequately handle a feared situation (possibly perpetuated by his/her own anxiety, where present; Whaley et al., 1999) and conveys this belief to the child through modeling of anxious/avoidant behavior and communication regarding anxious thoughts about the degree of threat in a given scenario. Since children with anxiety disorders

appear to mirror the negative coping expectations and heightened threat perception of their parents (Micco & Ehrenreich, 2008) and are more likely to select an anxious or avoidant solution following discussions about anxiety-provoking scenarios (Barrett et al., 1996), such avoidant child behavior may perpetuate this cycle by reconfirming parents' negative beliefs about a child's ability to cope with feared stimuli or events.

## **Parenting-Based Modifications and Treatment Response**

Family factors, such as those cited above, are often implicated as a variable in the failure of individual CBT to sufficiently benefit some children and adolescents with anxiety disorders (Ginsburg & Schlossberg, 2002). In response to such concerns, several investigators have augmented individual CBT formats to allow for a greater degree of parental involvement. The resulting treatment approach is often referred to as Family Cognitive Behavior Therapy (FCBT; e.g., Barrett, Dadds, & Rapee, 1996; Cobham, Dadds, & Spence, 1998).

Over the last 10 years, FCBT has been examined using a number of variations regarding the degree and type of parent involvement incorporated into a relatively fixed child anxiety CBT format similar to the one first developed by Kendall et al. (1990). However, evidence for the incremental utility of FCBT over individual CBT for child anxiety remains scant and inconsistent (Wood, Piacentini, Southam-Gerow, Chu, & Sigman, 2006). Seven investigations have examined FCBT in comparison to an individual CBT methodology (Barrett, 1998; Barrett et al., 1996; Cobham et al., 1998; Mendlowitz et al., 1999; Nauta, Scholing, Emmelkamp, & Minderaa, 2003; Spence et al., 2000; Wood et al., 2006), all with children presenting for treatment of generalized anxiety disorder, separation anxiety disorder, or social anxiety disorder. As summarized by Wood et al. (2006), the majority of these studies present findings supportive of FCBT in comparison to individual treatment, but only across a minority of treatment outcome measures, with most measures failing to support significant differences between treatment modalities. Taken together, these results may indicate that the addition of family components to individual CBT is unlikely to improve treatment outcomes for anxious children. However, some have also suggested that FCBT skills are best applied with anxious parents of anxious children and that FCBT usage with non-anxious parents might be unnecessary or mask positive treatment effects for families with a greater degree of psychopathology (Cobham et al., 1998). Yet another compelling explanation may be the possibility of a somewhat poor match between the types of parenting skills utilized in typical FCBT and common parenting factors observed in the families of anxious children (Wood et al., 2006).

In most iterations of FCBT, parents are engaged as treatment "coaches" who are encouraged to serve as models for the effective usage of CBT skills. Parents often receive psychoeducation about the cognitive-behavioral model of anxiety and its intervention components, along with anxiety management or parent training skills. However, as observed by Wood et al. (2006), FCBT rarely includes an

explicit focus on parental intrusiveness, autonomy granting or similar control behaviors developmentally linked to childhood anxiety disorders. To remedy this, these authors created an FCBT protocol that adds to the original approach used for parent involvement (e.g., Barrett et al., 1996) through an explicit focus on altering parent communications that may convey a greater level of intrusiveness or limit the child's autonomy inappropriately. During these modified FCBT sessions, which are part of the *Building Confidence* program (Wood & McLeod, 2008), the child is seen alone for the first 15–20 min of the session, after which parents are seen alone for 25–30 min, followed by a 10–15 min family meeting to conclude the session. The goal of parent-only time in session is to teach parents communication techniques that will enhance the child's self-efficacy and aid in the child's acquisition of new anxiety management skills. Specific communication skills taught include: (1) how to give children choices if they appear indecisive in a feared situation (rather than choosing a solution for them); (2) allowing children to experience distress and learn new solutions through "trial and error", as opposed to taking control of the situation for them; (3) effectively identifying and accepting emotions and emotionally driven responses, rather than criticizing these; and, (4) facilitating the uptake of new self-help skills (Wood et al., 2006). Appropriate responding to anxious child behaviors using the behavioral reward system and planned ignoring skills set forth by Barrett et al. (1996) are also included in the parent-oriented component of the *Building Confidence* program.

Wood et al. (2006) compared the efficacy of the *Building Confidence* FCBT approach to an individual child CBT protocol (Kendall et al., 1990) in a sample of 40 children (aged 6–13 years) with generalized anxiety disorder, separation anxiety disorder, or social anxiety disorder who were randomly assigned to one of the two treatment conditions. Similar to previous controlled trials comparing individual CBT to FCBT, both groups of children improved significantly from pre- to post-treatment on several measures of child anxiety. However, not only did the youth in the *Building Confidence* FCBT condition improve to a significantly greater degree across parent- and independent evaluator-rated child anxiety symptom measures, including measures of disorder severity, child distress, functional impairment, and interpersonal/familial relationships, but they did so at a more rapid rate of change (Wood et al., 2006). These results suggest that the explicit incorporation of relevant parenting factors associated with child anxiety into treatment of childhood anxiety disorders may significantly enhance treatment outcomes while simultaneously averting potential deleterious family-related complications in child treatment.

### ***Case Example***

Ella, an 8-year-old female diagnosed with separation anxiety disorder and several sub-clinical specific phobias (water, darkness, planes) presented for treatment along with her mother, Mrs. A. Ella's father participated in the initial clinical assessment process, but was unable to attend subsequent treatment sessions due to his work schedule. At the start of treatment, Mrs. A reported that Ella was experiencing very

high levels of fear and worry, along with a strong reluctance to separate prior to school several mornings per week and at dance classes, play-dates, birthday parties, and similar activities. The prior summer she reportedly refused to attend summer camp after one half-day of attendance. Although Mrs. A indicated that she and her husband rarely went out without Ella and her younger sister, Grace, she reported being unable to leave Ella with a babysitter on those occasions due to Ella's distress. In the initial assessment, Ella only reported some fears of the dark and swimming in the ocean, and moderate concern about being "taken" from school. In a pre-treatment behavioral observation task, during which Ella and her mother played together briefly followed by a clean-up task, Mrs. A appeared to have difficulty allowing Ella to play independently and made a relatively high frequency of critical comments with regard to Ella's play. For instance, when Ella picked up a doll and attempted to engage in pretend play about her doll running in a race, Mrs. A responded, "Don't be silly. Dolls can't run in races."

Given the observed interaction patterns between Ella and her mother, along with Mrs. A's reported history of generalized anxiety, a treatment plan that maximized both the teaching of parent anxiety management skills and child CBT components was selected. At first, Ella and her mother were typically seen together for the majority of session, with some additional time spent independently with Mrs. A to teach parenting strategies that might help facilitate the use of child anxiety management skills. The first three sessions, which covered basic emotion identification, psychoeducation about how thoughts, feelings, and behaviors perpetuate separation anxiety, and a review of anxiety-related physiological sensations and relaxation skills, proceeded without incident. In parent-alone time, the therapist introduced a behavioral reward system and discussed how to combine planned ignoring skills with labeled praise to encourage Ella's appropriate management of her anxiety symptoms.

In the interval prior to session 4, Mrs. A called the therapist and indicated that she was having increasing difficulty managing Ella's anxiety, particularly in the mornings before school. In fact, Mrs. A had allowed Ella to stay home from school on the day she called following a particularly intense and lengthy tantrum. Mrs. A stated, "I just couldn't talk her into going and she wouldn't stop crying. . . I started worrying that if I didn't give her a break today then I would never get her to school again." Over the next two sessions, it was determined that a stronger focus on both parent communication skills when Ella exhibited fear or reluctance to engage in feared activities and expedited movement toward graduated exposure plans, created in consultation with Ella, were appropriate. Given Ella's young age, the therapist decided to forgo cognitive restructuring and concentrated on the creation of an FAH and discussion of the rationale for exposure instead. Ella and her mother selected the least fear-evoking item from the list (staying at dance class without mom for 10 min) and a contingency contract was developed to ensure that Ella would receive a tangible reinforcer (an inexpensive bracelet from a local store) following completion of this activity, since Mrs. A had been inconsistent in the delivery of rewards previously. During parent-only time, the therapist reviewed the rationale for exposure and

role-played several communication principles for verbal and non-verbal responding to Ella's distress. Among these, an emphasis was placed on Mrs. A tolerating her own distress about Ella experiencing anxiety and allow Ella, wherever possible, to approach feared situations and negotiate solutions to difficult scenarios, without Mrs. A's direct influence.

Sessions 7–12 largely followed the format of the previous two sessions, with a review of exposure homework, followed by the selection of increasingly difficult situations from Ella's FAH and the establishment of a contingency contract to support reinforcer delivery for successful completion of FAH items. In parent-only time, it became clear that Mrs. A had difficulty recognizing when it was appropriate to praise and reward her daughter's increasingly brave behaviors. Therefore, therapist modeling of positive behavior identification and reinforcer delivery was provided, along with extensive role-playing of communications that would support Ella's independent usage of self-help skills. Over time, tangible reinforcers were largely minimized in Ella's treatment plan and replaced by enhanced usage of praise and other reinforcers, such as time spent playing or eating privately with one of her parents. By session 12, Ella was no longer consistently avoiding any of the items on her original FAH and Mrs. A asked to discontinue treatment, owing to difficulties with the scheduling of future sessions. Following a termination "party" celebrating Ella's achievements, a post-treatment assessment was conducted. This assessment found that while Ella's avoidance was now minimal, Mrs. A and Ella both felt that she still experienced sub-clinical fears about separation, particularly when alone at night or going on school trips independently. According to Mrs. A, if these situations were targeted for exposure, Ella complied with requests to engage in these situations independently and her fear decreased significantly following separation. In novel or unexpected fear situations, Mrs. A indicated more difficulty applying treatment techniques and newly acquired communication skills. Following discussion with Ella's therapist, Mrs. A opted to seek CBT for her own generalized anxiety, rather than engaging Ella in further treatment of these concerns. A 6-month follow-up revealed that neither Ella nor her mother was currently experiencing clinically significant anxiety symptoms.

## **Additional Complicating Factors in Child Anxiety Treatment**

The research on additional factors impacting the outcome of child and adolescent anxiety disorder treatment is still at an early stage of development. Some sociodemographic and clinical variables, such as parent-child (dis)agreement about presenting problems, have been identified that may affect the course of treatment for childhood anxiety. However, since the research on these potentially complicating factors in child anxiety treatment is either new or lacks consistent substantiation, we will only briefly review these here in hopes of further stimulating research on these topics.

## Sociodemographic Characteristics

Similar to findings observed in community mental health settings (e.g., Gould, Shaffer, & Kaplan, 1985; Weisz, Weiss, & Langmeyer, 1987), research has supported that children and their families who complete treatment for anxiety disorders differ very little from those who fail to complete treatment (Kendall & Sugarman, 1997; Pina, Silverman, Weems, Kurtines, & Goldman, 2003). For instance, Pina et al. (2003) examined characteristics of 137 children or adolescents (aged 6–16) and their families who were either considered treatment completers or non-completers following administration of a brief (10–12 session), exposure-based cognitive and behavioral treatment for either a phobic or anxiety disorder. Treatment completers ( $n = 106$ ) were considered those participants who completed the entire 10- or 12-session protocol, while non-completers ( $n = 31$ ) began the program but discontinued completely at some point prior to protocol completion (average number of sessions attended = 5). Other than some suggestion that attrition rates may have been higher among Hispanic/Latino participants in this study, few differences between groups were observed. The authors of this study recommended that future investigations concentrate less on sociodemographic characteristics of children receiving anxiety treatment and more on stressors and barriers to treatment attendance, along with therapist, relationship, and context issues or demands that might discourage attendance in treatment (Pina et al., 2003).

## Parent–Child Symptom Agreement

A common problem across domains of child psychopathology treatment is the degree to which parents and their children disagree about the nature and severity of the child's difficulties and the associated need for treatment of such concerns. For instance, in a study by Yeh and Weisz (2001), 381 clinic-referred children and their parents were asked to list the child's primary or target concerns. Of these participants, 63% failed to concur on even a single target problem. Even when grouped by broad problem category, a third of the parent–child pairs still demonstrated no overlap in problems identified (Yeh & Weisz, 2001). While these authors speculate that difficulties or conflict about the nature of a child's difficulties may explain instances of poor outcome for both internalizing and externalizing treatment samples, a child's acceptability of and positive engagement in the treatment context seems to be particularly important to the success of child anxiety treatment (Chu & Kendall, 2004).

To this end, the resolution of significant parent–child conflicts or differences about presenting concerns and intervention needs should be prioritized in child anxiety treatment. Thorough clinical assessment procedures may provide several opportunities to build consensus across any existent areas of shared concern and, as appropriate, emphasizing the fit between anxiety treatment components and shared treatment priorities. While some providers may be tempted to only share clinical

conceptualizations of child problems with parent alone (a process that may provide some opportunity to address areas of disagreement and conflict), it is also vital to identify and validate any significant misgivings or areas of divergence in problem focus with children and adolescents who voice such concerns directly. In addition, prioritizing intervention components that fit best with problem areas identified by both parent and child early in treatment may help build rapport with families and help facilitate subsequent child and parent engagement in therapy.

## Summary and Future Directions

Despite strong support for the efficacy of CBT in treating childhood anxiety disorders, there are some children who fail to benefit from standard CBT approaches. This chapter identified several complicating factors that may contribute to resistance to CBT for anxiety disorders and provided practical recommendations for modifying standard CBT to address these factors. First, children presenting with comorbid anxiety and depressive disorders may be prone to symptomatic relapse following CBT; as such, the addition of treatment components specifically targeting children's depression (including behavioral activation, challenging negative self-evaluation, social skills training, and positive reinforcement) may be beneficial for this subsample of anxious children. Second, studies of the effects of externalizing disorders on anxiety disorder treatment have produced mixed results, with some studies showing that this comorbidity has no effect on treatment outcome (Flannery-Schroeder et al., 2004), and other studies showing that it affects immediate treatment outcome (Costin et al., 2002) or ability to maintain treatment gains (Rapee, 2003). When treating children who present with secondary externalizing disorders (most common in younger boys), it may be necessary to include treatment modifications that specifically target inattention/hyperactivity (i.e., shorter sessions, problem solving) and aggression (i.e., behavioral contingency plans, learning to differentiate between anxious and angry feelings).

Family interactions, characterized by parental criticism/lack of warmth and over-control (behaviors that may, in turn, be reinforced by children's anxious behaviors), are common challenges in CBT for child anxiety, particularly if one or both of the parents is also anxious. Recent advances have been made in developing CBT for child anxiety that effectively address parenting characteristics; family CBT that specifically targets problematic communication patterns, such as overly intrusive comments and reinforcement of anxious behaviors, has been found to be particularly efficacious (Wood et al., 2006). Therapists treating anxious children should also work with children and their parents to achieve consensus regarding important treatment targets in order to maximize children's engagement and motivation in treatment.

Although recent advances have been made in determining factors that contribute to treatment resistance, much work remains in identifying how best to individualize CBT for different sub-populations of anxious children. In particular, future research

should examine the effect of more severe comorbidity (i.e., co-principal anxiety and externalizing disorders; Costin et al., 2002) on CBT for anxiety disorders, followed by a controlled evaluation of a modular CBT approach (see Chorpita, Taylor, Francis, Moffitt, & Austin, 2004) and emotion-focused or “unified” CBT approaches (e.g., Ehrenreich, Goldstein, Wright, & Barlow, 2009) that include treatment components that address a wider spectrum of co-occurring problems. In addition, while it has been established that FCBT is efficacious in treating separation anxiety, generalized anxiety, and social anxiety disorder, further research should examine the inclusion of parents in treatments for other childhood anxiety disorders (such as panic disorder and OCD). Finally, we have very little information on the degree to which sociodemographic variables, including race/ethnicity and SES, impact perceptions of and response to CBT; it is imperative that further research determine the effectiveness of standard CBT for child anxiety in diverse populations and settings. With greater understanding of the factors underlying treatment complications that arise in CBT, we will be able to more effectively tailor CBT approaches for use with specific sub-groups of children with anxiety disorders.

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