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Handbook of

Treating Variants and Complications in Anxiety Disorders

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To my beautiful wife and children (Maya and Noah) for your endless love, faith, and support. You are my 'best'!

Eric A. Storch

To my graduate school professors, who taught me the importance of evaluating the full domain of functioning and to think beyond mere diagnoses

Dean McKay

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Part I

Overview of Complexities in Anxiety Disorders

Nature and Etiological Models of Anxiety Disorders

1

Marie S. Nebel-Schwalm and Thompson E. Davis III

The nature of anxiety is both familiar and complex. It is a common human experience that has served adaptive and protective purposes in our evolution as a species. Yet, the consequences, and even the presentation, of anxiety can differ drastically. On the one hand, anxiety can serve a useful and adaptive purpose by keeping one from harm. If one is looking over the Grand Canyon, it behooves the individual to be anxious enough to stay several feet back from the edge. In this way, anxious feelings prompt us to be cautious, and this can protect us. However, anxiety can also be problematic—usually when it is experienced in greater proportions than a situation typically calls for or experienced in situations in which there is no identifiable harm or danger. For example, experiencing intense sensations of trepidation, anxiety, and fear every time one has to leave the house may be cause to suspect the presence of a clinical disorder. It is these instances in which anxiety is maladaptive that are problematic and cause for concern—typically when there is anxiety of unusual intensity or anxiety that is particularly interfering in one's ability to live a productive life.

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Anxiety has been defined as "the tense anticipation of a threatening but vague event; a feeling of uneasy suspense" (Rachman, 1998, p. 2) and as the "future-oriented mood state" when one makes preparations to deal with potentially aversive situations (Barlow, 2002, p. 64). The closely related concept of fear is sometimes used synonymously with anxiety, but researchers have pointed to distinctions between them. Fear is defined as "an emotional reaction to a specific perceived danger" (Rachman, 1998, pp. 2–3) and as a "primitive alarm in response to present danger" (Barlow, 2002, p. 104). The key distinctions between anxiety and fear are the orientation with regard to a threat (i.e., in the future or in the present) and whether the trigger is ambiguous or specific. A third related concept is worry. Whereas anxiety and fear are considered emotional responses, worry can be understood as a primarily verbal thought process (rather than imagery-based) that is centered on potential negative outcomes (Borkovec, Ray, & Stober, 1998). Lang (1968) included worry as one of three core systems of the fear response: cognitive (e.g., thoughts and worry), behavioral (e.g., avoiding situations and seeking safety), and physiological (e.g., arousal and muscle tension). Because fear and anxiety refer to mood states (and worry is a negative cognitive rumination), theories typically address the etiology of fear and anxiety, and worry is treated as a potential symptom of these mood states.

When anxiety and fear are sufficiently intense and interfering to the point of becoming a clinical disorder, there is usually cause for concern.

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There are nine main anxiety disorders according to the *Diagnostic and Statistical Manual of Mental Disorders Fourth Edition Text Revision* (*DSM-IV-TR*; APA, 2000): separation anxiety disorder (SAD) (most commonly a childhood onset), panic disorder, agoraphobia, specific phobia, social phobia, obsessive–compulsive disorder (OCD), posttraumatic stress disorder (PTSD), acute stress disorder, and generalized anxiety disorder (GAD). Understanding the nature and causes of impairing levels of fear and anxiety can lead to better treatments and outcomes for those who are suffering. Toward this end, etiological theories for anxiety and fear will be discussed followed by a review of each of the main disorders individually (e.g., nosology, age of onset, prevalence rates, and course).

Etiology

Several theories have attempted to explain why people develop anxiety symptoms and disorders. Typically, these theories have variously emphasized the influence of four pathways: direct learning (i.e., classical conditioning), indirect learning (i.e., modeling and negative information transmission), a biological preparedness pathway, and a non-associative pathway that presumes no conditioning experiences (Coelho & Purkis, 2009). The latter model has received considerable debate (e.g., see Poulton & Menzies, 2002 and subsequent commentaries). Many theories feature one primary mechanism or factor; however, the current consensus favors integrating explanations to address the combined influence of multiple factors (e.g., Barlow's triple vulnerabilities theory, 2002, and Mineka and Zinbarg's contemporary learning theory, 2006). The following is a review of these components, including cognitive and biological theories, followed by a brief introduction to several key theories in the field of anxiety etiology.

Classical Conditioning

The role of learning is emphasized in the first three theories: classical conditioning, observational learning, and negative information transfer (Rachman, 1998). The acquisition of fear or anxiety by way of classical conditioning refers to anxiety or fear being associated with a stimulus or situation based on direct experience (e.g., someone who was bitten by a dog develops a phobia of dogs). This notion was famously documented by Watson and Rayner's work with an infant (1920/2000). After initially demonstrating that an 11-month-old boy, Little Albert, did not fear rats, Watson and Rayner classically conditioned a fear in him by pairing exposure to the rat with a loud noise. This resulted in a newly developed fear response by Albert when presented with a rat. Results of this experiment described Albert as crying and trying to leave when shown a rat, in direct contrast to his early reactions before going through the conditioning.

An important contribution to this understanding of anxiety and fear maintenance was Mowrer's two-factor theory of learning (1951). Although classical conditioning is involved with fear acquisition, operant conditioning (i.e., specifically negative reinforcement) is also thought to maintain the anxiety. In other words, while the initial fear response to a stimulus may have its origins in classical conditioning, it is the individual's desire to alleviate or avoid fearful sensations that perpetuates the fear (i.e., avoiding leads to a reduction in uncomfortable sensations which is thought to reinforce the avoidance response). Decades of research, however, have indicated several limitations with this theory. For example, a classical conditioning model has difficulty explaining situations where people are exposed to a distressing event (e.g., a snake bite) but do not develop a phobia. Conversely, there are instances of people who develop extreme anxiety and/or fear without experiencing an aversive event. Additionally, if over time one is negatively reinforced for avoiding the feared stimulus, why does extinction not occur and the fear dissipate? Additional components involved in the creation and maintenance of fear and anxiety were obviously missing.

The basis for these missing components would be found in additional theory and research on cognition and the nature of the cues being presented to the individual as well as additional variables affecting the situation. For example, increasingly many of the cues associated with anxiety disorders

(and especially panic disorder) were understood to incorporate interoceptive and exteroceptive cues (Bouton, Mineka, & Barlow, 2001). Moreover, theorists began to integrate cognitive science with classical conditioning leading to the hypothesis that thoughts themselves could even serve as conditioned stimuli and trigger panic (Bouton et al.). The deceptively straightforward notion of a "simple" classical conditioning explanation has become increasingly complex as there are problems with defining and determining the role of the actual conditioned stimulus (e.g., conditioned stimuli can trigger a response, modulate other responses, or even be impacted by other variables themselves; Bouton et al.). As a result, more integrated theories (discussed later) have come to accentuate, detail, and expand on the basic concept of classically conditioned anxiety and phobia.

Observational Learning

Fears can also be learned vicariously through observational learning—observing the actions and outcomes of others. Bandura demonstrated observational learning of aggressive behaviors by children who watched an adult act aggressively (Bandura, Ross, & Ross, 1963). More recently, the modeling of anxiety was examined in a study on parental modeling with 25 parent–child dyads (Burnstein & Ginsburg, 2010). Dyads were randomly assigned to either an anxious or non-anxious condition. Parents in the anxious condition were trained to make anxious statements about an upcoming spelling test for their child (e.g., "this test looks way too hard," "I don't think you can do this") and to pace around the room. Parents in the non-anxious condition were instructed to make statements such as, "I think you can do well on this test" and to look about the room. After seeing their parents behave in these manners, the children took a spelling test. The groups did not differ in spelling performance, but self-reported anxiety levels were significantly higher among children in the anxious group.

Animal research has also demonstrated this effect. Experimental research with rhesus monkeys has shown that laboratory-raised monkeys, although initially not fearful of a snake, can learn to behave fearfully after observing their wild-raised parents' reactions to a real or toy snake (Mineka, Davidson, Cook, & Keir, 1984). Interestingly, lab-raised monkeys can also learn not to fear, despite observing a fearful monkey (Mineka & Cook, 1986). In that study, three groups of monkeys were given the same amount of exposure across six sessions. Group 1 watched a non-fearful monkey behave calmly with snakes, group 2 interacted calmly with snakes, and group 3 watched a non-fearful monkey behave calmly with neutral stimuli. All three groups then watched six sessions of a monkey behaving fearfully with snakes. When later placed directly with snakes, monkeys from group 1 showed significantly fewer signs of fear acquisition as compared to the other groups. This phenomenon is referred to as latent inhibition and is a component of the integrated theory proposed by Mineka and Zinbarg (2006) that is reviewed later in this section.

Clearly, there are interesting implications for prevention from these results. Thus, providing a positive model (as shown in Mineka & Cook, 1986) can buffer the effects of later negative models. However, it is not clear why the group of monkeys who had calm exposure prior to watching the fearful interactions did not also benefit from this experience. Perhaps it is because group 1 shared the observation modality across both sessions (watching a non-fearful and then a fearful monkey) whereas group 2 personally experienced snakes and then observed a fearful monkey. The idea that previous exposure can create resilience is similarly reflected in the concept of stress inoculation. However stress inoculation presumes that the experience of mildly distressing events can buffer severe reactions to more aversive events that occur later (Lyons, Parker, Katz, & Schatzberg, 2009).

Negative Information Transfer

A third etiological influence on anxiety and fear has come to be called negative information transfer—the idea that one can learn to be anxious by hearing others talking negatively (or anxiously)

about a subject. Support for this theory has often used retrospective reporting (see King, Gullone, & Ollendick, 1998); however, other observational and experimental methodologies have also demonstrated this effect. When observing comments parents made to their children at a playground, parents of anxious children were more likely to say "be careful" and "don't climb too high" than parents of non-anxious children (Beidel & Turner, 1998). Further, the influence of family discussions was demonstrated in experiments that used ambiguous vignettes (Barrett, Rapee, Dadds, & Ryan, 1996). First, by themselves, children were asked to answer questions to situations such as what would you do if you found some kids playing a "great game?" Then, they were asked to answer the same question in a room with their parents. Finally, they were asked to talk it over with their parents and indicate their answer one last time. Even when anxious children initially endorsed wanting to join in the game, after the family discussion, they were more likely to endorse avoidance of the hypothetical situation.

Experimental tests carried out with research assistants, rather than parents, has also shown the effect of negative information. A study featuring a fictitious monster character (Field, Argyris, & Knowles, 2001) and one about unfamiliar animals (Field & Lawson, 2003) demonstrated that children hearing negative information about novel stimuli had more fearful beliefs. Further, children who heard positive information were less likely to be fearful of the novel stimuli than those who received no information.

Biological Preparedness

As classical conditioning would predict, having an aversive interaction with a stimulus can lead to the development of a phobic response. However, the types of stimuli that are feared do not appear to be randomly distributed. Seligman (1971) stated that this nonrandom distribution of easily conditioned stimuli is because people are biologically prepared to learn to fear stimuli that could threaten their survival. A review of studies found evidence that "fear-relevant" stimuli (such as snakes, spiders, and angry faces) were more easily conditioned and less likely to be extinguished than non-fear-relevant stimuli (e.g., flowers, triangles, and happy faces; McNally, 1987). More recent studies have also found evidence for biological preparedness. This was demonstrated in observational learning studies where lab-reared rhesus monkeys showed faster fear acquisition to snakes and crocodiles as compared to flowers and rabbits, even when controlling for the intensity of fear that was displayed by the observed monkey (Mineka & Ohman, 2002a). This theory presumes the individual has had a conditioning event (which distinguishes it from the non-associative account that is discussed later). It also proposes that fears and phobias of modern dangerous stimuli (e.g., guns and moving vehicles) are significantly less common than, for example, spiders, snakes, and heights because the modern stimuli have not had enough time (evolutionarily speaking) to be naturally selected as being fear inducing.

Non-associative Theory

Despite support for the previous associative or learning-based theories, in some cases people do not report any direct or vicarious events when discussing the onset of their anxiety and fear. For example, researchers studying water phobia found that most people did not know how the fear started (Menzies & Clark, 1993). This lack of clear etiology that is unrelated to a learning event (i.e., an associative event) is sometimes called the non-associative theory, and similar to biological preparedness, it is thought to have an evolutionary basis.

There is some debate about the state of this non-associative theory (see Mineka & Ohman, 2002b; Poulton & Menzies, 2002), and some have proposed a renaming of this theory to "nonspecific" rather than non-associative (Davey, 2002). While the biological and genetic contributions of fear and anxiety are not disputed, their role to the exclusion of associative accounts has been called into question. For example, a great deal of retrospective research examining the etiology of fear and anxiety has interpreted "I don't know how my fear/anxiety started"

responses as support for this account. Limitations to this type of interpretation, however, include problems with memory recall (and the use of retrospective studies), confusion about what is included in the definition of unconditioned stimuli (e.g., exteroceptive and/or interoceptive stimuli), and the lack of alternate explanations for current findings (e.g., that implicit memory may be involved; Mineka & Ohman, 2002b). The issue is in all likelihood less one of anxiety and fear being caused by associative *or* non-associative means, but rather an examination of how much associative experience is required for a given individual given his or her unique biological and genetic predisposition (Marks, 2002).

Cognitive Theories

Cognitive and cognitive–behavioral theorists generally accept the role associative and non-associative accounts play in anxiety acquisition, but their focus is more on how one interprets and processes events (Rachman, 1998). Clark's model of panic attacks (1986) places the appraisal of threat as the beginning feature of the development of panic, which is followed by physical sensations and catastrophic interpretations. Beck's (1996) theory of anxiety states that activated schema prime anxious beliefs and effect how information is organized in one's memory. Analysis of thought content can reveal information about a person's emotional functioning. Individuals with depression tend to think of loss whereas those with anxiety think of harm and danger (Clark, Beck, & Brown, 1989). Also, impaired cognitions have been found among those with anxiety, including biased information-processing that selectively (and hypervigilantly) perceives threat (Rapee, Schniering, & Hudson, 2009) and overpredicts the anticipation of fear (Rachman, 1994).

An interesting test of how perceptions affect anxious symptoms was done using CO_2-enriched air (Sanderson, Rapee, & Barlow, 1989). Half of the participants were told they could control the level of CO_2 when a light was lit, the other half were not given this option. In reality, participants had no control over the amount of CO_2 and all participants were exposed to the same level for 20 min. The frequency of catastrophic thoughts was higher among those in the "no-control" condition, and these participants were more likely to experience panic attacks (80%) compared to those in the "with-control" condition (20%).

Biological Theories

Several biological factors have been proposed to play a role in the etiology of anxiety. While there are numerous biological aspects to anxiety and phobia, two particular factors are important to discuss in the brief space available here. Current research into the genetic origins and heritability of anxiety (e.g., the results of twin studies) as well as the early temperamental foundations—especially behavioral inhibition—of anxiety and worry have been very important.

Genetic Heritability

Family concordance rates, particularly twin studies, provide evidence that having a parent with an anxiety disorder puts one at a higher risk of developing an anxiety disorder (Beidel & Turner, 2005). Although, rates of heritability are modest, ranging from 30 to 40% (Hettema, Prescott, & Kendler, 2001). Overall, a general tendency toward being anxious is implicated as opposed to a specific 1–1 risk of inheriting a particular disorder. For example, this general risk (vs. specific risk) is supported by twin studies. Namely, if one twin has GAD, the other is likely to have an anxiety disorder, but it may be social anxiety (Hettema et al.; Kendler, Neale, Kessler, Heath, & Eaves, 1992a). This general psychopathology risk was also found with twin studies that identified a genetic link between anxiety disorders and depression (Thapar & McGuffin, 1997). Eley and Stevenson (1999) speculated that environmental influences are responsible for the specific disorder that is expressed among those with a general genetic predisposition.

Temperament

One's predisposition to be inhibited behaviorally and shy is arguably an innate aspect that is observable

in young children. Of course, fearful behavior in children is adaptive (e.g., "stranger danger" reactions); however, the persistence of this behavior into older childhood appears to comprise an inhibited temperament. Commonly referred to as behavioral inhibition, this concept has also been associated with anxiety proneness and anxiety sensitivity (Beidel & Turner, 2005). Behavioral inhibition is characterized by the tendency to respond to novel situations with feelings of anxiety, avoidant behaviors, and increased distress. Some evidence suggests behavioral inhibition is a specific risk factor the later development of social anxiety disorder (Prior, Smart, Sanson, & Oberklaid, 2000). The stability of behavioral inhibition may go beyond biological vulnerability and involve parental influences. As discussed in the section on negative information transmission, parents play an important role in their children's anxiety and fear-based beliefs and behaviors. Parents who model encouragement, warmth, and encourage opportunities for positive novel interactions may help reduce feelings of anxiety in their children (Asendorpf, 1990).

Integrated Theories

While the different pathways and influential components said to cause and/or maintain anxiety and fear are extensive (only a brief overview was presented to this point), these individual variables acting in isolation are not generally considered to be the complete etiological picture. Increasingly, the field has attempted to integrate various variables of interest in the development of fear and anxiety to create a clearer picture of how associative and non-associative backgrounds, along with various other psychological variables, result in an anxiety disorder. Two such theories are briefly reviewed below, Barlow's triple vulnerability theory and Mineka and Zinbarg's update to traditional learning theory and associative accounts.

Triple Vulnerability Theory (Barlow, 2002)

Barlow (2002) includes three main vulnerabilities in his integrative theory of anxiety etiology. They are general genetic (or biological) vulnerability, general psychological vulnerability, and specific psychological vulnerability. The general biological vulnerability refers to ones temperament, such as behavioral inhibition, and is based in part on Gray's (1982) behavioral inhibition system (BIS), behavioral approach system (BAS), and the fight/flight system (FFS). Gray proposes that individuals with anxiety have an overactive BIS in response to novel stimuli. The FFS system roughly corresponds with escape or aggressive reactions and is thought to be correlated with fear and panic responses. The BAS is thought to be indicative of extraverted reactions and impulsivity. Whether a person will approach or withdraw from certain situations is thought to be related to one's temperament, and personality traits (e.g., introversion and extraversion) are genetically determined to some degree. Estimates of the genetic contributions of personality traits (e.g., the big five traits) range from 41 to 61% (Jang, Livesley, & Vemon, 1996).

The general psychological vulnerability includes feeling a lack of control; attributing negative events to internal, global, and stable factors; and parenting styles (i.e., whether parents foster autonomy in a warm, sensitive, consistent, and contingent manner; Suarez, Bennett, Goldstein, & Barlow, 2009). For some disorders (i.e., GAD and depression), the two general vulnerabilities may sufficiently explain their onset (Suarez et al.). However, other disorders require the third dimension of a specific psychological vulnerability. The specific vulnerability is referred to as "learning what is dangerous" (Barlow, 2002, p. 279), and its content is a function of the particular anxiety disorder. These vulnerabilities can develop many ways, including through direct exposure to a dangerous situation, having a false alarm in a specific situation (i.e., a physiological response that comes to be incorrectly associated with a stimulus or situation), and through vicarious conditioning (such as observing or being told something is dangerous; Suarez et al., 2009). Some examples include feeling that physical sensations are alarming or dangerous (panic disorder), having been in specific situations that are dangerous (specific phobia), feeling that social situations are to be feared and avoided (social anxiety disorder),

and irrationally believing thoughts have dangerous power (OCD).

The role of true and false alarms differs depending on the specific disorder. For example, panic disorder is said to be characterized by false alarms, whereas specific phobias are more likely to be related to true alarms. Specific vulnerabilities for social phobia can include a true alarm (being laughed at in a social situation) or a false alarm (feeling panic when interacting socially). Some do not experience any alarm but may think they lack social skills (regardless of how accurate their appraisal is). A key thought that is implicated in the etiology of social phobia is "social evaluation is dangerous" (Barlow, 2002, p. 462). Lastly, with regard to OCD, a specific psychological vulnerability includes the belief that some thoughts are "dangerous and unacceptable" (Barlow, p. 536).

Contemporary Learning Theory (Mineka & Zinbarg, 2006)

Mineka and Zinbarg (2006) proposed a revision to the older, problematic associative accounts and integrated many factors in one updated, comprehensive learning theory. They included two domains of vulnerabilities (genetic/temperament and previous learning experiences) with three contextual domains (perceptions of controllability and predictability, direct or vicarious conditioning, and properties of the conditioned stimulus). These pathways converge on the experience of an anxiety disorder, which is further affected by post-conditioning factors, including unconditioned stimulus inflation/reevaluation and the presence of inhibitory or excitatory conditioned stimuli.

With regard to the development of specific phobias, it is important to mention the phenomenon of latent inhibition (which was previously discussed in the "Observational Learning" section). For example, recall the previously described study where rhesus monkeys were exposed to a particular sequence of conditions (i.e., first they viewed a calm model interact with a snake, then a fearful one). The monkeys experiencing this condition were calmer when they were subsequently placed directly with snakes as compared to monkeys who did not initially observe the calm model (Mineka & Cook, 1986). Within the theory, this illustrates the impact of prior experiences on learning and has potential for treatment utility in the prevention of fears and/or anxiety. Previous experiences can also include one's history of feeling mastery and control over one's circumstances. Control can also play a role in the contextual domain-meaning (e.g., in the moment one is experiencing a traumatic event, is there the perception of control such as being able to escape?). Another contextual domain is the properties of the conditioned stimulus. This may include whether the stimulus was fear-relevant or fear-irrelevant, interoceptive or exteroceptive, and the temporal proximity to stressful events. Lastly, an example of a post-conditioning variable is the inflation effect. This occurs when a person experiences a minor trauma that does not lead to a phobia, but later, after a more intense trauma (even if it has nothing to do with the initial mild trauma), a phobia develops. For example, a person who experiences a minor trauma with a dog later develops a specific phobia of dogs following an intense trauma that was unrelated to dogs (e.g., a severe car accident; Mineka & Zinbarg, 2006).

The Nature and Description of Current Anxiety Disorders

While the theories surrounding anxiety and phobia etiology have grown and become increasingly complex, those theories are frequently considered and applied broadly to a circumscribed set of anxiety disorders. These disorders have largely grown to represent and capture certain aspects of fear or anxiety (e.g., matters of intensity or degree) or the target of the emotional response. For example, GAD encompasses problems with broad, pervasive worry, while social phobia is limited to specific instances in which one is anxious about social interactions and being evaluated by others. Given the unique characteristics and aspects of each anxiety disorder, the common *DSM-IV-TR* anxiety disorder diagnoses will briefly be described and reviewed below (though with the forthcoming *DSM-5*, some revision to these diagnoses and groups may occur).

Generalized Anxiety Disorder

GAD's hallmark symptom is excessive, uncontrollable worry over many domains (e.g., work, school, health, relationships, finances, and politics). The worry must be pervasive and long lasting (i.e., at least 6 months). To meet criteria, one must experience at least 3 of the 6 *DSM-IV-TR* symptoms (with the exception of children, who only need one of the following): restlessness, easily fatigued, difficulty concentrating, irritability, muscle tension, and sleep disturbance (APA, 2000, p. 476). In addition, there must be impairment and interference in daily routines, work, academics, or other areas of functioning.

A longitudinal study using a European sample recorded the onset of GAD symptoms (vs. a GAD diagnosis; Angst, Gamma, Baldwin, Ajdacic-Gross, & Rossler, 2009). They found the vast majority of individuals (75%) displayed their first "GAD symptoms" before the age of 20 years, and the average age of symptom onset was 15.6 years. Studies done in the United States reported on the age of onset for individuals meeting full *DSM-IV* criteria for GAD (Kessler, Berglund, et al., 2005). They found the median age of onset for GAD to be 31 years (which was the oldest among the anxiety disorders) and the lifetime prevalence to be 5.7% (Kessler, Berglund, et al.). The 12-month prevalence rate in the United States for GAD was 3.1% (Kessler, Chiu, Demler, Merikangas, & Walters, 2005).

When considering previous *DSM* versions, it is notable that the impairing nature of GAD has not always been appreciated (Brown, 1997; Persons, Mennin, & Tucker, 2001). Earlier versions of the DSM listed GAD as a condition that could be comorbid with other disorders, but not a primary diagnosis. Another change across revisions is the required duration of symptoms: previously one needed to show that the symptoms lasted 1 month or longer; currently the requirement is 6 months or longer. Preliminary recommendations for the upcoming *DSM-5* include renaming GAD as generalized worry disorder, pathological worry disorder, or major worry disorder to more accurately capture its distinguishing feature among the anxiety disorders and reinstating the shorter symptom duration requirement to 1 or 3 months (Andrews et al., 2010). Reasons for lowering the threshold include the noted difficulty of reliably reporting symptoms as long ago as 6 months and the benefit of identifying and treating individuals with impairing symptoms earlier.

There is evidence that GAD is a chronic and unremitting disorder (e.g., Weisberg, 2009; Wittchen & Hoyer, 2001; Woodman, Noyes, Black, Schlosser, & Yagla, 1999), although some studies have found evidence that most individuals had periods of remission and recurrence, as opposed to persistent chronicity (Angst et al., 2009). This may be due to differences between early and late onset of GAD. Early-onset GAD typically follows a more gradual increase in symptoms and with greater chronicity, whereas late-onset GAD is more likely to follow a stressful life event (Brown, 1997).

Obsessive–Compulsive Disorder

OCD is characterized by distressing, intrusive, and uncontrollable thoughts (obsessions) that cause great anxiety and often compel the person to perform certain rigidly prescribed behaviors or mental acts (compulsions) that are marked by repetition and are not realistically related to the distressing obsession (APA, 2000). For example, "If I don't touch the light switch three times exactly, something bad will happen to my family." Individuals with OCD (as opposed to those with a psychotic disorder, for example) are aware that the thoughts and impulses are the result of their internal processes, rather than external influences. Compulsions are commonly reported to relieve anxiety, which further reinforces these behaviors. The obsessions and compulsions are time-consuming (at least 1 h a day but can be considerably more time-consuming) and/or cause significant distress or impairment. Usually the individual becomes aware that the obsessions and compulsions are excessive or unreasonable; however, it is possible not to have this awareness, which is indicated by the "with poor insight" specifier (APA). Though rare, an individual can

have only obsessions or compulsions, though the common presentation is the combination of both. The median age of onset for OCD is 19 years of age, its 12-month prevalence is 1.0% (Kessler, Chiu, et al., 2005), and its lifetime prevalence is among the lowest of anxiety disorders (1.6%; Kessler, Berglund, et al., 2005). The onset is not normally distributed, however. In one study, the vast majority (82%) of individuals reported the onset to be before 18 years, while the remaining 18% had adult onset (most of which were between 19 and 35 years; Pauls et al., 1995).

Unfortunately, OCD can cause considerable impairment, including the inability to carry out basic day-to-day activities and functions (Barlow, 2002), sleep disturbances, job loss, dropping out of school, and poor quality of life (Markarian et al., 2010). The experience of intrusive thoughts that are unwanted is often very distressing to the individual. Thoughts may include contamination, sexual, aggressive, or religious themes (such as associating words with the devil; Barlow, 2002).

The course of OCD is being increasingly recognized as heterogeneous. Men with OCD have an earlier age of onset, a higher comorbidity with tic disorders, and a slightly worse prognosis, whereas women's symptoms seem to fluctuate based on hormonal changes (e.g., menstruation and postpartum; Lochner et al., 2004). More so than the other anxiety disorders, support for OCD as a genetically based disorder has been strong (Nicolini, Arnold, Nestadt, Lanzagorta, & Kennedy, 2009), with reports as high as a 6.2-fold risk of OCD among first-degree relatives (Grabe et al., 2006). Research is underway to understand specifically what is transmitted (i.e., specific genetic markers and nongenetic attributes; Rector, Cassin, Richter, & Burroughs, 2009). Some findings have shown first-degree relatives of individuals with OCD to score higher on personality traits such as neuroticism (Samuels et al., 2000), maladaptive perfectionism (ruminating about mistakes vs. adaptively having high standards), and an inflated sense of responsibility (e.g., that one is responsible for the safety of others; Salkovskis, Shafran, Rachman, & Freeston, 1999). The most commonly reported obsessions among adolescents with OCD are contamination, fear of illness, need for exactness or symmetry, and religiosity (Swedo, Rapoport, Leonard, Lenane, & Cheslow, 1989; Toro, Cervera, Osejo, & Salamero, 1992) and the most common themes for adults were sexuality and aggression (Rasmussen & Tsuang, 1986).

Recent conceptualizations have discussed the merits of redefining OCD as a spectrum that features OCD, body dysmorphic disorder, trichotillomania, and possibly tic disorders, hypochondriasis, and obsessive–compulsive personality disorder (Phillips et al., 2010). Should such a change occur, this spectrum is recommended to be subsumed under an "anxiety and obsessive–compulsive spectrum disorder" category (Phillips et al., p. 528).

Another issue is whether it is necessary to distinguish between hoarding and OCD as separate entities (Pertusa, Frost, & Mataix-Cols, 2010; Rachman, Elliott, Shafran, & Radomsky, 2009). Confusion exists about the status of hoarding (which is not in the *DSM-IV-TR*, except under obsessive–compulsive personality disorder). When comparing OCD and hoarding, hoarding occurs more commonly and has a higher likelihood of poor insight (Rachman et al.). It has been proposed that, although comorbidity is possible between OCD and hoarding, most individuals with this behavior represent a distinct clinical syndrome that should receive its own diagnostic label of "hoarding disorder" (Pertusa et al., 2010, p. 1012).

Posttraumatic Stress Disorder

The current diagnostic criteria for PTSD include four main criterion groups and specify that symptoms must have persisted longer than 1 month (*DSM-IV-TR*). Criterion A is exposure to a trauma that involved threat of serious injury or death to self or others and the experience of intense fear, helplessness, or horror. Criterion B is reexperiencing the event in one or more ways (including dreams, physiological reactivity, and intense distress when exposed to cues of the event). Criterion C is persistently avoiding stimuli and having reduced responsiveness via feeling detached, inability to recall important aspects of the trauma,

avoiding thoughts or discussions about the trauma, avoiding people or places that remind one of the trauma, diminished interest in significant activities, restricted range of affect, and a sense of foreshortened future; and criterion D is two or more symptoms of arousal, including sleep disturbances, anger outbursts, difficulty concentrating, hypervigilance, and exaggerated startle response (APA, 2000). One can be diagnosed with acute PTSD if symptoms emerge 1 month after a trauma and last less than 3 months; chronic PTSD is when symptoms remain beyond 3 months post-trauma, and delayed onset is when symptoms did not appear until 6 months or longer after the trauma.

The 12-month prevalence of PTSD among a sample of over 5,000 adults was 3.5% (Kessler, Chiu, et al., 2005). Lifetime prevalence was 6.8% and median age of onset was 23 (Kessler, Berglund, et al., 2005). However the prevalence of a potentially traumatic event is estimated to be 25% by the age of 16 years (Costello, Erkanli, Fairbank, & Angold, 2002). PTSD can be extremely debilitating and is a robust predictor of suicide attempts among adolescents (Wilcox, Storr, & Breslau, 2009). When comparing physical anxiety symptoms among individuals with panic disorder or PTSD, individuals with PTSD had briefer symptom-free periods, experienced greater fluctuation, greater unpredictability, and greater uncontrollability (Pfaltz, Michael, Grossman, Margraf, & Wilhelm, 2010). Responses vary depending on the type of trauma. Early-onset, chronic, and interpersonal traumas (e.g., emotional, physical, or sexual child abuse) are associated with more impaired emotional regulation than single-event, non-interpersonal traumas (Ehring & Quack, 2010). Perhaps the most debilitating trauma for children and adolescents is childhood sexual abuse. Adolescents with a history of sexual abuse account for 20% of all adolescent suicide attempts (Fergusson, Horwood, & Lynskey, 1996). Because of the serious nature of chronic abuse, some have criticized the current description of PTSD as being too focused on single-trauma events rather than chronic trauma exposure (Briere & Spinazzola, 2005).

Although it is a prominent feature of PTSD, controversy exists regarding criterion A. Difficulties arise with how broadly or narrowly to define "trauma" (Weathers & Keane, 2007).

For *DSM-5*, some have proposed that criterion A be dropped entirely (Brewin, Lanius, Novac, Schnyder, & Galea, 2009). A related problem is the need for more developmentally appropriate standards for diagnosing PTSD in children and adolescents (Pynoos et al., 2009). If criterion A is retained, it is important to note that what may qualify as a trauma for children may not necessarily be the same as for an adult (e.g., witnessing domestic violence or experiencing a severe dog bite). Also, trauma-related sequelae in children and adolescents often differ from that of adults. Children may seek close proximity to their parent or caregiver, be preoccupied with being safe, regress with regard to developmental skills (e.g., toileting and speech), and develop new fears (Pynoos et al.; Scheeringa, Pebbles, Cook, & Zeanah, 2001). Adolescents may exhibit more reckless and risky behaviors (such as thrill seeking and substance use; Pynoos et al., 2009).

Acute Stress Disorder

Acute stress disorder was first introduced in the *DSM-IV*, in 1996. It shares criterion A (experiencing a trauma) with PTSD, but it differs regarding the timeline of symptoms. Posttraumatic stress disorder can be diagnosed 1 month following a trauma, whereas acute stress disorder can be diagnosed in the immediate aftermath, up until 1 month post-trauma. It also emphasizes the experience of dissociative symptoms. One must display at least three of the following: sense of numbing/detachment, reduced awareness of surroundings, derealization, depersonalization, and dissociative amnesia (APA, 2000).

Prevalence rates to date seem to be trauma-specific. Interestingly, the same rate (16%) was reported for survivors of injuries requiring hospitalization (Mellman, David, Bustamante, Fins, & Esposito, 2001), as was reported for moving-vehicle accident survivors (Harvey & Bryant, 1999). Because of its recency, information on

acute stress disorder is not as readily available, and this applies to information regarding children and adolescents as well (March, 2003). Another problem could be the practical difficulties of measuring reactions to trauma so soon after the event.

Criticisms of acute stress disorder include the ambiguity of criterion A, its reliance on dissociative symptoms, lack of data supporting its diagnostic validity, and the fact that a main purpose was to predict the onset of another disorder (namely, PTSD; O'Donnell, Creamer, Bryant, Schnyder, & Shalev, 2003). Studies have found various predictors of PTSD, but meeting a diagnosis of acute stress disorder is not consistently one of them (Bryant, Harvey, Guthrie, & Moulds, 2003; Elsesser, Sartory, & Tackenberg, 2005; Mellman et al., 2001). There is some evidence that dissociative symptoms may predict PTSD; however, these tend to be subclinical presentations of acute stress disorder. Other predictors of PTSD include prior traumas, prior psychopathology, heightened arousal immediately following the trauma, as well as avoidant coping and feeling overwhelmed (Mellman et al.). Certainly more research is needed to better understand the diagnostic validity of acute stress disorder, its predictive validity regarding PTSD, and developmental trajectories of trauma-related responses among children and adolescents (March, 2003).

Panic Disorder (PD)

Panic attacks are sudden and intense experiences of physical symptoms (e.g., chest pain, feelings of choking) that peak within 10 min and can lead the individual to believe they are experiencing a real medical emergency (e.g., commonly patients experiencing a panic attack believe they are having a heart attack). Panic attacks, by themselves, are not a diagnosable disorder; rather, they are a prominent feature in the diagnosis of PD (but can occur with any anxiety disorder). The main criteria for PD are the presence of recurrent and unexpected panic attacks with at least one of the following symptoms for 1 month or longer: persistent concern about subsequent attacks, worry about losing control or going crazy, and a significant change in behavior (APA, 2000). PD can be diagnosed with or without the presence of agoraphobia.

The 12-month prevalence of PD is 2.7% (Kessler, Chiu, et al., 2005). The lifetime prevalence is 4.7% and the median age of onset was 24 years of age, which is second only to GAD as the oldest median age of onset among anxiety disorders (Kessler, Berglund, et al., 2005). As many as 80% of individuals PD have a comorbid diagnosis (Olfson et al., 1997), such as major depressive disorder (Roy-Byrne et al., 2000), GAD, substance abuse (Otto, Pollack, Sachs, O'Neil, & Rosenbaum, 1992), and bipolar disorder (Goodwin & Hoven, 2002). Research on subthreshold experiences of panic disorder has found it to be common (when counted with full threshold panic disorder, the prevalence rate in a community sample is 40%; Bystritsky et al., 2010). Subthreshold panic disorder is also associated with greater rates of depression, dysthymia, psychosis, GAD, bipolar disorder, and substance use disorders (Bystritsky et al.).

Panic symptoms have been noted to occur in clusters (e.g., nocturnal panic, Craske & Tsao, 2005; respiratory symptoms, Abelson, Khan, Lyubkin, & Giardino, 2008; Onur, Alkin, & Tural, 2007; cognitive, cardiorespiratory, and mixed somatic, Meuret et al., 2006) leading some to propose that these clusters may represent meaningful subtypes of panic disorder. For example, Meuret et al. (2006) found three subtypes using factor analysis and determined whether these clusters significantly predicted various aspects of PD (such as intensity, frequency, interference, distress, and worry). Among the predictions, cardiorespiratory symptoms (i.e., heart palpitations, shortness of breath, choking, chest pain, and numbness) predicted the severity and frequency of panic and distress. Mixed somatic (i.e., sweating, trembling, nausea, chills, hot flashes, and dizziness) predicted severity, interference with life, distress, and worry. Lastly, the cognitive subtype (i.e., thoughts that one is going crazy, losing control, and a feeling of unreality) predicted worry, distress, interference, and severity (Meuret et al.). However, Kircanski, Craske, Epstein,

and Wittchen (2009) reviewed the literature and did not find adequate support for symptom subtypes. Although there is initial support for the idea of meaningful symptom clusters, they concluded that the research has not yet demonstrated sufficient external validation criteria with regard to functional differences between subtypes (Kircanski et al.). Lastly, the task force that reviewed the current DSM criteria of PD did not recommend including subtypes for *DSM-5* and recommended mostly minor changes (e.g., changing the wording of "hot flushes" to "heat sensations"; Craske et al., 2010).

Agoraphobia

The *DSM-IV-TR* does not allow one to be diagnosed with agoraphobia but rather agoraphobia without history of panic disorder or panic disorder with agoraphobia (APA, 2000). The *DSM-IV-TR* describes agoraphobia as anxiety in situations where escape is difficult or help is not easily available, and such situations are avoided or endured with significant distress. A diagnosis is made if one has these symptoms that relate to the fear of developing panic-like symptoms. However, if the avoidance of situations is limited in scope to either a specific stimulus or social interactions, one should consider the possibility of specific phobia or social phobia, respectively (APA).

The 12-month prevalence rate of agoraphobia without panic disorder was 0.8%, the lowest among several anxiety disorders included by Kessler, Chiu, et al. (2005). Lifetime prevalence was 1.4% and the median age of onset was 20 years of age (Kessler, Berglund, et al., 2005). The clinical presentation of agoraphobia without PD is low compared to population-based prevalence rates. This might be due to lower rates of treatment seeking among individuals with agoraphobia without PD (Mosing et al., 2009). Controversy also exists as to whether agoraphobia is part of the panic disorder spectrum (Andrews & Slade, 2002) or a distinct disorder (Nocon et al., 2008). Among the recent proposals by the *DSM-5* task force was the suggestion to eliminate the current hierarchy that exists between PD and agoraphobia by separating them as different disorders and allowing for comorbid diagnoses when appropriate (Wittchen, Gloster, Beesdo-Baum, Fava, & Craske, 2010). Because the task force was not unanimous in their proposed changes, they recommended a careful reanalysis of clinical data sets in order to facilitate a more consensual decision about this issue.

Specific Phobias

The key feature of a specific phobia is a marked and persistent fear of a certain circumscribed stimulus or situation (*DSM-IV-TR*). While potentially not as broad and debilitating as other more pervasive disorders, specific phobias are associated with significant long-term psychological and social effects from childhood even into adulthood (Davis, 2009; Davis, Ollendick, & Öst, 2009). Per the diagnostic criteria, exposure to the feared stimulus should evoke anxiety and avoidance (or distress if escape is not possible; *DSM-IV-TR*). In adults, one must also realize that the fear is excessive; though children are not required to have this degree of insight. Five separate types of specific phobia have been included in the *DSM-IV-TR*: animal type (e.g., dogs and snakes; includes insects), natural environment type (e.g., storms and dark), situational type (e.g., small spaces and airplanes), blood-injection-injury type (e.g., receiving injections or seeing blood), and other type (e.g., clowns, vomit, and other fears that do not fit within the other four categories).

Specific phobias have consistently been found to be one of the more common psychological disorders. Current research indicates specific phobias are the most common anxiety disorder with a lifetime prevalence rate of 12.5% and a 12-month prevalence rate of 8.7% (Kessler, Berglund, et al., 2005; Kessler, Chiu, et al., 2005). The average age of onset for a specific phobia has been suggested to be 9–10 years of age (Stinson et al., 2007); however, it is generally accepted that there is a great deal of variability depending on the type of fear. As a result, a range of onset is typically accepted spanning childhood to early adulthood (Öst, 1987) with onset typically

mirroring the emergence of certain developmental capacities in cognition (i.e., from concrete to increasingly abstract fears; Davis, 2009). In addition, specific phobias exact an unexpected toll on the health care system as well with those having specific phobias accessing medical care at rates higher than those with OCD and second only to those with panic disorder (Deacon, Lickel, & Abramowitz, 2008). Unfortunately, most individuals with a specific phobia have been found to have had it an average of 20 years and fewer than 10% have sought treatment (Stinson et al., 2007).

Social Phobia (Also Known as Social Anxiety Disorder)

Social phobia is marked by the fear of performing or interacting socially. It has a generalized subtype that indicates a person is anxious in most or all social situations. Without this subtype, one may be socially anxious only in certain situations, such as giving a speech. Its key criteria include a marked and persistent fear of social (or performance) situations, exposure to such situations evokes anxiety, the person recognizes the fear is excessive or unreasonable, and social (or performance) situations are avoided or endured with great distress (APA, 2000). The 12-month prevalence rate for social phobia among an adult sample was 6.8% (Kessler, Chiu, et al., 2005). Reported lifetime prevalence rates range from 12.1% (Kessler, Berglund, et al., 2005) to 13.3% (Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996), and the median reported age of onset was 13 years (Kessler, Berglund, et al., 2005). It is the second most common anxiety disorder (specific phobias are the most common).

The adolescent median age of onset coincides with a time when concerns about social evaluations increases (Bruch, Heimberg, Berger, & Collins, 1989); however, risk factors that occur prior to adolescence have been identified. One's temperament (e.g., being shy or behaviorally inhibited), even as a young child, is believed to play a role in the development of social phobia, along with environmental factors such as parenting styles (Kendler, Neale, Kessler, Heath, & Eaves, 1992b). Aspects of parenting that have been investigated include warmth, control, intrusiveness, and lack of encouragement (De Rosnay, Cooper, Tsigaras, & Murray, 2006). Parents have even been found to have an influence on how infants react. For example, studies with infants have noted the degree to which infants base their reactions on their caretaker's emotional state (known as social referencing; Murray et al., 2008). Two studies with infants illustrate this effect. The first was done with mothers who did not have social phobia. They were instructed to act either anxiously or non-anxiously when a stranger entered the room. Infants responded with more avoidance and fear when their mothers were anxious (De Rosnay et al., 2006). The second study compared mothers with and without social phobia. In this study, infants of mothers with social phobia, as compared to controls, were more fearful and avoidant when interacting with a stranger (Murray et al., 2008).

Research regarding the upcoming *DSM-5* has focused on clarifying some controversial aspects of social phobia. One criticism is that it is difficult to determine whether someone has generalized social phobia or avoidant personality disorder. Some have proposed that avoidant personality disorder is a severe form of social phobia rather than a distinct disorder. A recent review found mixed evidence on this issue and indicated the need for further research in order to obtain consensus (Bogels et al., 2010). Some have also questioned the utility of the "generalized" specifier. Bogels et al. (2010) found little evidence to support this and recommended a dimensional approach be adopted instead. However, with regard to specific situations, they suggested that a "predominantly performance" specifier would have clinical utility. Lastly, they noted that children can reliably be diagnosed with social phobia as young as 6 years of age, validity studies are still needed for children younger than 9 years, and that young children may manifest social phobia as selective mutism, indicating the need for further research to clarify the relationship between these two disorders.

Separation Anxiety Disorder

It is developmentally appropriate for a young child to be distressed when separating from his or her parent. This distress typically dissipates between the ages of 3–5 years (Masi, Mucci, & Millepiedi, 2001). For children older than this, intense distress when separating from a parent or caregiver can interfere with their social relationships and adjustment to day care or school, as well as disrupt the lives of parents and caregivers. Thus, SAD is characterized by excessive and developmentally inappropriate distress when anticipating (or experiencing) separation from a loved one or one's home (APA, 2000). A child must have three or more symptoms from the following list: recurrent distress when separated, persistent worry that something bad will happen (e.g., being kidnapped or getting lost), recurrent worry about harm coming to a loved one, reluctance or refusal to go to school or elsewhere, reluctance or refusal to be alone or to fall asleep alone, the experience of nightmares, and somatic complaints (e.g., stomachaches and headaches; APA). Among these symptoms, the most commonly endorsed ones are separation distress, avoidance of being alone/without an adult, and avoidance of sleeping away from home. The least commonly endorsed symptom is having nightmares (Allen, Lavalee, Herren, Ruhe, & Schneider, 2010). In addition to the impairing effects of these symptoms, children with SAD often display defiant and disruptive behaviors during times of separation (e.g., such as going to school or going to bed; Pincus, Santucci, Ehrenreich, & Eyberg, 2008). SAD has also been shown to be a risk factor for later depression (Keenan, Feng, Hipwell, & Klostermann, 2009; Lewinsohn, Holm-Denoma, Small, Seeley, & Joiner, 2008) and panic disorder (Lewinsohn et al.).

Depending on the type of prevalence being assessed (i.e., point, 6- or 12-month) and which respondents were queried (parents, adolescents, or both), prevalence rates for SAD among children and adolescents ranged from 0.2 to 1.5% (Canino et al., 2004; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). Point prevalence studies found 0.2% of 14–19-year-olds to have SAD (Lewinsohn et al.) and 1.2% of 5–15-year-olds (Ford, Goodman, & Meltzer, 2003). A 6-month prevalence study with 6–14-year-olds reported 0.9% (Breton et al., 1999), whereas a 12-month prevalence study with a slightly larger age range (children ages 4–17 years) found 1.5% with SAD (Canino et al., 2004). Lastly, lifetime prevalence is reported to be 5.2% (Kessler, Berglund, et al., 2005). As these figures reveal, the age of onset for SAD is primarily in childhood, and there is a decline in onset as the child matures (Keenan et al., 2009). The median age of onset is 7-years-old (which is similar to specific phobias) and the vast majority of cases occur between the ages of 5 and 17 years (Kessler, Berglund, et al., 2005).

The DSM stipulates that the disorder's onset must begin before 18 years of age (APA, 2000); however, there is growing interest into adult onset SAD (Silove, Marnane, Wagner, Manicavasagar, & Rees, 2010). An outpatient sample of 508 adults presenting with mood and anxiety disorders reported that 41% had adult SAD (20% without a childhood diagnosis and 21% with a childhood diagnosis; Pini et al., 2010). Thus some have argued that it is more prevalent than previously realized and that adults with this disorder experience more disabling effects as compared to children, who in one study were found to have low levels of impairment (Foley et al., 2008).

Conclusion

Overall, the etiology and nature of anxiety and fear are complex and not fully understood at this time. Their causes are usually multiply determined through various and repeated experiences over time. While single experiences may be sufficient to cause distress and disorder, the consensus at this time is that most anxiety disorders have varied roots extending back through an individual's unique learned and biological histories. As such, it is not a question of whether anxiety disorders (or simple anxiety and fear) are the result of nature or nurture but rather nature *and* nurture. The expression of anxiety and fear has

also been a topic of debate. Much of the work to classify and distinguish anxiety disorders has been along the lines of differentiating them based on degree (e.g., GAD), target of anxiety or fear (e.g., social phobia or specific phobia), or at times arbitrary duration (e.g., ASD vs. PTSD). In addition, *DSM-5* may see the criteria of various anxiety disorders change or entire diagnoses moved to different sections (e.g., OCD). Even so, the extensive research into anxiety and fear and disorders related to each has exploded over recent decades leading to more comprehensive understandings of the causes of anxiety and fear and the expression of and subsequent treatment for disorder in these emotions.

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Prognostic Indicators of Treatment Response for Adults with Anxiety

2

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As the field has moved beyond initial efficacy trials for the treatment of anxiety disorders, it becomes more crucial to consider the variants and complications that may arise in treatment. Although efficacious treatments for anxiety disorders have been developed, findings related to prognostic indicators of treatment response remain an important research priority. In particular, clinicians and researchers alike need information on translating nomothetic findings into idiographic treatment plans, addressing related conditions that frequently co-occur with anxiety disorders, and overcoming clinical impasses that may complicate treatment.

Nature of the Problem

This chapter will explore several factors that contribute to complexity in effectively conceptualizing and treating anxiety disorders in adults. Anxiety is frequently comorbid with other acute and enduring disorders, which has important implications for effective treatment (e.g., Brandes & Bienvenu, 2009; Huppert, 2009; Zahradnik & Stewart, 2009). For the purposes of this chapter, comorbidity is defined as having co-occurring clinically significant symptoms of both an anxiety disorder and another disorder at some time across the lifespan. Additionally, several therapeutic variables complicate the treatment of anxiety disorders. Transdiagnostic and integrative treatments are presented as promising means of addressing complications that arise in the treatment of comorbid and complex presentations of anxiety disorders.

Anxiety Disorders and Axis I Comorbidity

Anxiety disorders are frequently comorbid with other acute conditions. The following sections address Axis I disorders that frequently co-occur with anxiety, as well as etiological theories of their co-occurrence and considerations for treatment.

Multiple anxiety disorders. Comorbid acute disorders are complicating factors that may serve as important prognostic indicators for the effective treatment of anxiety disorders. Co-occurrence of two or more anxiety disorders in the same individual tends to be more the rule than the exception (e.g., Brown & Barlow, 1992; Kessler et al., 1996; Wittchen, Zhao, Kessler, & Eaton, 1994). Brown, Campbell, Lehman, Grisham, and Mancill (2001) examined comorbidity among anxiety disorders in a clinical sample. Among anxiety disorders, generalized anxiety disorder (GAD) and posttraumatic stress disorder (PTSD) were especially likely to co-occur with other disorders. Additionally, it is useful to examine comorbidity

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as a function of primary disorders. For example, while Specific Phobia has been highly comorbid with other anxiety disorders, it frequently presents as a less severe condition that co-occurs with other more debilitating anxiety disorders (Brown et al.). Thus, it may be that some forms of multiple anxiety disorder comorbidity, such as Panic Disorder and GAD, are more likely to complicate prognosis than others, such as any primary anxiety disorder and secondary Specific Phobia (Brown & Barlow, 1992). Although evidence suggests that comorbidity rates decrease after treatment for a primary anxiety disorder (e.g., Borkovec, Abel, & Newman, 1995; Brown, Antony, & Barlow, 1995), comorbidity rates tend to be substantially higher in those with more severe conditions (Kendall, Kortlander, Chansky, & Brady, 1992; Kessler et al., 1994) and severity serves as a negative prognostic indicator (Keller et al., 1992). As such, comorbidity of anxiety disorders may significantly complicate the clinical picture.

Anxiety disorders and comorbid depression. Unipolar depression and anxiety are frequently comorbid in adults, adolescents, and children, in both clinical and community samples (e.g., Angold, Costello, & Erklani, 1999; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Maser & Cloninger, 1990; Merikangas, Dierker, & Szamari, 1998; Mineka, Watson, & Clark, 1998). Although comorbidity exists across many disorders, the strength of association between anxiety and depression denotes a unique relationship (Axelson & Birmaher, 2001; Lewinsohn, Rohde, & Seeley, 1995). Also, rates of comorbidity between anxiety and depression remain elevated even after controlling for comorbidity with other disorders (Lewinsohn, Zinbarg, Seeley, Lewinsohn, & Sack, 1997).

The overlap between anxiety and depression is particularly important as those with the comorbid condition experience more impairment than those with pure presentations of either disorder. As compared to non-comorbid presentations of the disorders, comorbid anxiety-depression is associated with greater symptom severity (Bernstein, 1991; Coryell et al., 1988; Mitchell, McCauley, Burke, & Moss, 1988), higher rates of suicidal behavior (Lewinsohn et al., 1995; Reich et al., 1993; Rohde, Clarke, Lewinsohn, Seeley, & Kaufman, 2001), higher rates of mental health treatment utilization but poorer treatment response (Brent et al., 1998; Emslie, Weinberg, & Mayes, 1998; Lewinsohn et al., 1995), higher medical costs (Marciniak et al., 2005), and an increased risk of recurrence (Emslie et al., 1998). The presence of comorbid depression is also a negative prognostic indicator for anxiety disorder treatment outcome, as it has been shown to decrease the likelihood of remission of each of the disorders (Bruce et al., 2005).

Anxiety disorders and substance use. Substance use disorders also frequently co-occur with anxiety disorders, although there appear to be some differences among the anxiety disorders in their rates of comorbidity with substance use. First, although specific phobias are relatively prevalent in the population, they are less likely to be associated with substance use than GAD, Panic Disorder, Social Phobia, and PTSD (Zahradnik & Stewart, 2009). Additionally, drug use disorders are more frequently associated with anxiety disorders than alcohol use disorders (Zahradnik & Stewart). As it may be problematic to group drug use disorders into a homogenous category, further research is needed to explore drug use disorder by specific substances.

Several models have been proposed to explain the co-aggregation of anxiety disorders and substance use. Some models suggest that those with anxiety disorders may use substances to "self-medicate," or reduce emotional distress or affect-relevant withdrawal symptoms (Kushner, Sher, & Beitman, 1990). Alternatively, it is possible that anxiety symptoms result from chronic substance use (Kushner, Abrams, & Borchardt, 2000). For example, prolonged tobacco use may contribute to the development of panic disorder by producing chronic withdrawal symptoms, reduced health quality, or both (Breslau & Klein, 1999; McLeish, Zvolensky, Del Ben, & Burke, 2009). It may also be that a third factor related to individual differences underlies both anxiety and substance use. Anxiety sensitivity, or the tendency to fear bodily sensations most associated with anxiety, has been linked to both anxiety and substance use (Stewart & Kushner, 2001), suggesting its possible role as a third variable.

Generally, the literature supports the self-medication hypothesis for anxiety–substance use comorbidity (see Zahradnik & Stewart, 2009 for review). However, self-medication models may be limited in describing the complex relationship between anxiety disorders and substance use following the onset of both conditions (e.g., Stewart, 1996). Other models suggest that anxiety disorders and substance use reinforce one another over time through a mutual maintenance process, and their comorbidity is best explained by a complex transactional relationship. Thus, anxiety and substance use may co-occur through bidirectional negative effects (e.g., Zvolensky, Schmidt, & Stewart, 2003).

Anxiety disorder-substance use disorder comorbidity has important implications for effective treatment, as the comorbid conditions often result in less effective treatment for either condition, and higher rates of relapse to substance use (e.g., Bruce et al., 2005; Kushner et al., 2000). Treatment approaches designed to address anxiety-substance use comorbidity generally follow one of three treatment formats: sequential, parallel, or integrated (Zahradnik & Stewart, 2009). Sequential treatments first address one disorder then move on to the other in discrete stages. As clinicians commonly believe mental health issues cannot be effectively treated until substance use is controlled (Riggs & Foa, 2008), substance use disorders generally take first treatment priority over anxiety disorders. Parallel treatments ensure treatment for both disorders simultaneously; however, treatment is often conducted by different providers, so coordination of care represents a potential problem (Randall, Book, Carrigan, & Thomas, 2008). Lastly, integrated models of anxiety-substance use treatment attempt to create a hybrid treatment comprising intervention strategies that are effective in treating each disorder independently (Randall et al.). Ultimately, although integrated treatments may be the most promising, quality of care for anxiety–substance use is limited by systemic issues that tend to focus health care on discrete problems and not the full clinical picture (e.g., Weiss, Najavits, & Hennessy, 2004). Additionally, development and empirical evaluation of integrated treatments remains limited.

Anxiety Disorders and Comorbid Personality Disorders

Rates of comorbid anxiety and personality disorders range from 35 to 65% (Sanderson, Wetzler, Beck, & Betz, 1994; Skodol et al., 1995), and findings with regard to the impact on treatment outcome are mixed. Personality disorders interfere in instrumental and social relationships and are thought to impact the therapeutic process as well (Crits-Christoph & Barber, 2002). To date, none of the ten personality disorders have consistently been related to poor treatment prognosis, and no one anxiety disorder is sensitive to concomitant personality disorders on treatment outcome (Dreessen & Arntz, 1998). However, some patterns have emerged in the literature.

Models of comorbidity. An important first step is to examine the theoretical underpinnings of the relationship between anxiety and personality disorders. Researchers have proposed several models of the relationship between Axis I and Axis II disorders, including linear (i.e., causal), nonlinear (i.e., reciprocal), and common etiological models (Brandes & Bienvenu, 2009). Linear models suggest that either personality disorders are risk factors for anxiety disorders or personality disorders are consequences of anxiety disorders. Support for linear models has been partially established by several prospective and longitudinal studies. For example, controlling for the presence of Axis II disorders in adolescence, negative affectivity in adolescence predicted the onset of anxiety disorders in adulthood (Krueger, 1999). Similarly, higher negative affectivity predicted four-symptom panic attacks in adolescents (Hayward, Killen, Kraemer, & Taylor, 2000), and high neuroticism and low extraversion predicted the onset of PTSD in survivors of severe burns (Fauerbach, Lawrence, Schmidt, Munster, & Costa, 2000). Additionally, early experiences of anxiety disorders may influence developing personalities. Anxiety disorders in adolescence were shown to predict the development of personality disorders later in life, particularly Cluster C disorders (Goodwin, Brook, & Cohen, 2005;

Kasen et al., 2001). Findings that support a causal relationship in two directions (i.e., that personality disorders influence the development of anxiety disorders, and that early anxiety impacts the development of personality disorders) suggest that a bidirectional, reciprocal model may offer a better explanation than either linear model.

Beyond these, models that address common etiologies and overlap in Axis I and Axis II criterion are informative. Both anxiety disorders and Cluster C personality disorders are characterized by fear and avoidance; therefore, it is not surprising that these Axis I disorders would be particularly susceptible to comorbidity with Cluster C disorders. However, Saulsman and Page (2004) found that all personality disorders were associated with high neuroticism and disagreeableness, which lends an explanation for the comorbidity between anxiety and Cluster A and B disorders as well as Cluster C. Additionally, it has been shown that anxiety disorders are related to a personality style characterized by behavioral inhibition to the unfamiliar (Brandes & Bienvenu, 2009). This personality style may reflect a risk factor for the development of anxiety disorders, or it may be a marker of a range of inherited traits that includes anxiety disorders (Bienvenu & Stein, 2003).

Specific associations between personality disorder clusters and anxiety disorders. Due partly to similarities in diagnostic criteria (e.g., anxious, fearful traits), Cluster C personality disorders co-occur most often with anxiety disorders (Sanderson et al., 1994). Further, specific associations have been supported for particular Axis I and Axis II disorders that commonly co-occur, perhaps due to etiological relationships. Particularly high rates of PTSD were found in those with Borderline personality disorder while elevated rates of Social Phobia were found in patients with Avoidant personality disorder (McGlashan et al., 2000). Skodol et al. (1995) found Panic Disorder associated most highly with Borderline, Avoidant, and Dependent personality disorders; Social Phobia associated with Avoidant personality disorder; Obsessive-Compulsive Disorder (OCD) associated with Avoidant and Obsessive-Compulsive personality disorder; and Specific Phobia was not associated with any personality disorder.

Anxiety disorders and co-occurring personality disorders have also been identified empirically. In a longitudinal study of patients with anxiety disorders, the Harvard/Brown Anxiety Research Project (HARP) found 24% of patients to have at least one co-occurring personality disorder, with the most common diagnoses being Avoidant, Obsessive-Compulsive, Dependent, and Borderline personality disorders. Patients with Social Phobia and GAD were more likely to be diagnosed with a co-occurring personality disorder than those with other anxiety disorders (Sanderson et al., 1994). Taken together, findings suggest that anxiety disorders may co-occur with each of the three personality disorder clusters but also display some specific associations (e.g., Social Phobia and Avoidant personality disorder) at particularly high rates.

Many of the studies examining anxiety disorders and co-occurring personality disorders have focused the prevalence of the concomitant relationship and the relationship with symptom severity rather than the effects on treatment outcome. Additionally, a majority of the studies have been conducted on individuals with Panic Disorder (with or without Agoraphobia). In one study, co-occurring Panic Disorder and personality disorders were related to a more severe clinical picture (as indicated by more symptoms and suicidal behaviors; Ozkan & Altindag, 2005).

Effect of comorbidity on prognosis of anxiety disorders. In their review of the literature, Crits-Christoph and Barber (2002) suggest that personality disorders have shown a consistent adverse impact on the treatment outcome of a wide range of Axis I disorders. One way in which personality disorders may impede treatment of anxiety disorders is through difficulty establishing rapport and strong alliance, which is an indicator of treatment outcome (Ackerman et al., 2002). Individuals with personality disorders have difficulties developing relationships and trusting others, which may impair therapeutic rapport and lead to early termination and attrition (Ackerman et al.). Second, personality disorders may impede treatment simply by heightening the severity of the pathology in general. Third, personality disorders may impede treatment of Axis I disorders because individuals with Axis II

pathology frequently show impairments in self-insight, which may hinder treatment response. However, limited insight is a frequent complication to treatment and is not uniquely associated with personality disorders. Finally, unlike the ego-dystonic nature of Axis I disorders, personality disorders are frequently more ego-syntonic in nature. Because many of the underlying traits between Axis I and Axis II disorders are similar, it may be difficult to treat ego-dystonic symptoms that are related to an ego-syntonic trait. For example, the fear and avoidance based Cluster C Personality Disorders (i.e., Avoidant, Dependent, and Obsessive-Compulsive personality disorders) may particularly complicate anxiety disorders, which are also characterized by fear and avoidance. It may be that patients who identify with the fear as being consistent with their nature are less likely to seek treatment and have more difficulty engaging in treatments that seem to challenge their nature.

Despite these possible mechanisms of personality disorder interference in treatment, several factors hinder our ability to make broad conclusions about the impact of personality disorders on the outcome of treatment for anxiety disorders. First, the anxiety disorders have not been equally represented in the extant research, with a majority of the findings relating to Panic Disorder and OCD, and fewer data addressing GAD, Social Phobia, and Posttraumatic Stress Disorder. Second, very few studies have examined personality dimensionally. DSM-V field trials for Axis II disorders are testing dimensional approaches to personality disorders, reflecting a shift in the way personality characteristics are understood. Important information regarding the impact of Axis II disorders may be lost by using the dichotomous classification method. Lastly, most findings have not come from studies of comorbid personality disorders specifically but were secondary analyses of existing data. Thus, the mechanisms by which Axis II disorders impede treatment of Axis I have not been fully explored.

Considerations in interpreting findings. The findings related to co-occurring Axis I and Axis II disorders in treatment outcome studies are equivocal, and research methodologies may play an important role in the interpretability of the findings. A large body of research supports the assertion that personality disorders hinder treatment response (for a review, see Crits-Christoph & Barber, 2002). However, it may be that the reliability of findings is confounded by the study methods employed (Dreessen & Arntz, 1998). First, conclusions related to the deleterious impact of personality disorders on Axis I treatment outcome often were based on retrospective Axis II diagnoses made by raters who were not blind to the treatment condition (Kringlen, 1965; Lo, 1967; Mancuso, Townsend, & Mercante, 1993; Minichiello, Baer, & Jenike, 1987; Turner, 1987; Vaughan & Beech, 1985). Second, many studies which have concluded that personality disorders hinder treatment outcome are strictly comparing posttreatment symptomology (van den Hout, Brouwers, & Oomen, 2006), although the relative change from pre- to posttreatment severity scores indicates that individuals with and without personality disorders benefit equally from treatment (Dreessen & Arntz, 1998; van den Hout et al., 2006). Third, in both self-report questionnaires and clinical interviews, personality assessments are sensitive to mood states (Hirschfield et al., 1983; Pilkonis, Heape, Ruddy, & Serrao, 1991; Reich, Noyes, Coryell, & O'Gorman, 1986), so it may be that personality assessments are distorted by transiently high levels of anxiety (Stein, Hollander, & Skodol, 1993). Finally, the method of assessment used to make Axis II diagnoses contributes to the varied research findings. Van den Hout et al. (2006) concluded that the presence of personality disorders did not impact response to treatment when the Axis II diagnosis was made with the SCID-II but that it did attenuate treatment response when the diagnosis was determined using an unstructured interview. In light of these considerations, it is critical that the findings related to treatment outcomes of concomitant anxiety and Axis II disorders be interpreted in the context of the study methodologies.

In conclusion, the extant research on co-occurring anxiety and Axis II disorders is inconsistent. While the research findings are inconclusive, clinical experience suggests that the presence of an Axis II disorder interferes with treatment outcome.

Because personality disorders are most detrimental to relational functioning, it is intuitive that a disordered personality would impact the therapeutic bond, a most critical aspect of psychotherapy. However, this clinical intuition has yet to inform the theory related to the mechanism by which Axis II disorders may impact the treatment of anxiety disorders. Thus, in addition to the special considerations for research methods, it is important that future studies are theoretically driven.

Therapeutic Variables in Complications of Anxiety Disorders

In addition to factors of comorbidity discussed above, factors related to the therapist-client relationship may serve as complicating factors in the treatment of anxiety disorders. Factors such as client motivation and therapeutic alliance have been shown to impact treatment outcomes and serve as important prognostic indicators.

Therapeutic alliance. Therapeutic alliance is the bond between therapist and client that engages the client in the therapeutic process. Therapeutic alliance is believed to be an essential factor in the effective treatment of anxiety (Bordin, 1979; Hayes, Hope, VanDyke, & Heimberg, 2007). However, the research on therapeutic alliance and treatment outcomes is in its early stages and its findings remain inconclusive. Hayes et al. (2007) found a significant relationship between session helpfulness and client-rated working alliance, but not observer-rated working alliance. The researchers also found that alliance was associated with the level of engagement the client displayed during therapy. Similarly, Chiu, McLeod, Har, and Wood (2009) reported that poor early treatment therapeutic alliance predicted less improvement in parent-reported anxiety reduction at mid-treatment but not at posttreatment among children receiving cognitive-behavioral therapy (CBT) for anxiety disorders. They also reported that improvement in therapeutic alliance over the course of therapy predicted better posttreatment anxiety reduction. These findings indicate the importance of therapeutic alliance, especially early in treatment. However, findings on therapeutic alliance remain somewhat divided. Liber et al. (2010) reported that therapeutic alliance did not predict anxiety reduction in children who attended group or individual CBT. Numerous problems remain in studying therapeutic alliance, including measurement sensitivity and lack of variability in therapeutic alliance ratings (e.g., most ratings are high, likely due to demand characteristics). Furthermore, specific client characteristics and therapeutic techniques create difficulties in forming therapeutic alliance.

A client's difficulty forming social relationships may be a consequential variable that interferes with the forming of a therapeutic alliance. Moras and Strupp (1982) reported that clients who form successful personal relationships often form positive therapeutic alliance regardless of theoretical orientation. Similarly, Kokotovic and Tracey (1990) reported that building therapeutic alliance was more difficult when therapists viewed their clients as having poor social relationships. It is intuitive that therapeutic alliance is even more important in cases where clients struggle to build successful personal relationships, such as among those with social phobia. However, research involving social phobia and therapeutic alliance remains inconclusive. VanDyke (2002) reported that strong therapeutic alliance measured after the final session related to low posttreatment symptom severity after controlling for pretreatment severity. Conversely, Woody and Adessky (2002) reported that therapeutic alliance did not significantly relate to group CBT treatment outcomes in clients with social phobia. Neither study reported a relationship between early therapeutic alliance and treatment outcomes, which may suggest that therapeutic alliance is a result of treatment success. Further study is needed to elucidate the complex relationship between client variables, therapeutic alliance, and treatment outcome.

Client motivation. Problems with regard to motivation for treatment are common in individuals with anxiety disorders. Grant et al. (2005) reported that 80–95% of people with social phobia do not

seek treatment and that many people with social phobia only seek treatment after years of tribulations. Clients lacking motivation often display ambivalence in regards to anxiety treatment (Buckner, 2009). Clients are generally aware that excessive anxiety is distressing and interferes in their ability to partake in desirable activities. However, clients may also report hesitation to initiate services or prematurely terminate treatment for fear of others thinking negatively of them (Olfson et al., 2000). Given the importance of client participation in successfully reducing anxiety symptoms, it becomes imperative for clinicians to address client ambivalence during therapy.

Client motivation may be particularly salient when considering exposure therapy, as the treatment may be considered aversive to many clients, particularly when the approach is described before initiated. Indeed, Becker, Zayfert, and Anderson (2004) have reported that many CBT-oriented clinicians believe that the aversive nature of exposure therapy may increase client dropout, despite evidence that clients in exposure-based therapy for PTSD are not more likely to drop out compared to clients in other forms of CBT (Hembree et al., 2003). However, therapeutic alliance can be damaged as a result of conducting exposures at a rate in which clients are not ready for or capable of handling. Furthermore, damage to therapeutic alliance may occur if anxiety symptoms do not abate during an in-session exposure (Hayes et al., 2007). It may be that clients who partake in exposures and do not experience a decrease in anxiety during the exposures or between successive exposures doubt the benefit of continued treatment. A collaborative approach is especially valuable when structuring exposure sessions to help prevent nonadherence and ruptures to the therapeutic alliance.

Treatment Approaches to Address Anxiety Disorder Comorbidity and Complications

Given the range of client and therapeutic variables discussed above that may complicate the treatment of anxiety, it is important to develop flexible treatments to best address these problems. Although manualized cognitive-behavioral therapies have yielded very strong treatment effects (Norton & Price, 2007), the presence of complicating or negative prognostic variables may require adaptation of treatment models to address the specific case formulations. Transdiagnostic, unified, or integrated cognitive-behavioral treatments are presented as alternatives to disorder-specific protocols that may better address complicating factors of comorbidity, while treatments based on motivational interviewing models have begun to be used as stand-alone or adjunctive therapies for clients demonstrating motivational or relational complications.

Transdiagnostic and unified treatments. The efficacy of cognitive-behavioral therapy for the treatment of anxiety disorders has been well established. Evidence-based treatments have been developed for many specific anxiety disorders including panic disorder and agoraphobia (Craske & Barlow, 2001), GAD (Dugas et al., 2001), OCD (McLean et al., 2001), social phobia (Heimberg & Becker, 2002), and PTSD (Najavits, 2002). However, these studies' use of homogeneous samples (e.g., similar diagnoses, strict exclusion criteria) limits the generalization of these treatment packages for the application to complex factors such as comorbid anxiety, mood, personality, or substance use disorders. Recently, a number of researchers have suggested that a non-diagnosis-specific approach to treatment may allow for greater individualization and treatment flexibility by capitalizing on the common cognitive and behavioral processes that are shared across a range of anxiety, mood, and other emotional disorders (Barlow, Allen, & Choate, 2004; Mansell, Harvey, Watkins, & Shafran, 2009; McEvoy, Nathan, & Norton, 2009; Norton & Hope, 2005). Mansell et al. (2009) specifically suggest that unified or transdiagnostic approaches to treatment may be preferable for clients who do not fit a specific diagnostic category or for clients with complex or highly comorbid presentations. Unified treatment packages begin with an individualized case formulation that focuses on the functional links between the component pro-

cesses of most cognitive-behavioral models of anxiety: maladaptive cognitive appraisals, poor emotional regulation, emotional avoidance, and maladaptive behavior associated with disordered emotion (Barlow et al., 2004; McEvoy et al., 2009; Shafran, McManus, & Lee, 2008). The importance of an individualized and evidence-based case formulation is particularly significant as the complexity of a given presentation increases. Although the unified approach to the treatment of anxiety disorders is a relatively new idea, research has demonstrated empirical support for the treatment of anxiety disorders utilizing this framework (Erickson, 2003; Erickson, Janeck, & Tallman, 2007; Garcia, 2004; Norton & Hope, 2005).

While there has not been a specific anxiety disorder treatment package designed to accommodate the array of complicating factors that can arise in the treatment of anxiety, it is believed that a unified approach to treatment may represent the most desirable treatment strategy. With a unified approach, clinicians are able to treat the entire clinical picture rather than prioritizing diagnoses and sequentially treating each with diagnosis-specific treatment protocols (Blanchard et al., 2003; Brown et al., 1995; Norton, Hayes, & Hope, 2004).

Integrative treatment for anxiety–substance use comorbidity. The unified approach discussed above may represent an ideal framework for addressing the emotional dysregulation that underlies multiple comorbid anxiety disorder diagnoses as well as comorbid anxiety-depression. However, treatments designed to address both anxiety and substance use may be more difficult to develop and implement. Zahradnik and Stewart (2009) identify integrated and parallel interventions as more promising than sequential interventions in the treatment of anxiety–substance use. Theories that anxiety and substance use reinforce one another over time through mutual maintenance would suggest that integrated treatments may represent the best treatment option, but this has yet to be empirically established. One clear trend is that anxiety–substance use treatments that incorporate elements of cognitive-behavioral techniques (e.g., ERP for OCD, prolonged exposure for PTSD) perform better than those that do not. Despite difficulties in implementation, Zahradnik and Stewart (2009) identify preliminary results of integrated anxiety–substance use treatment as very encouraging.

Treatment addressing anxiety and comorbid Axis II. Overall, the extant research suggests that standard brief treatments for Axis I conditions often fail when Axis II pathology is also present (Crits-Christoph & Barber, 2002). However, this does not mean that the presence of personality disorders precludes effective treatment of anxiety. Instead, it may be that standard treatment for anxiety needs to be tailored to effectively address the presence of a personality disorder. An example of this is a case study by Walker, Freeman, and Christensen (1994) in which restricted environmental stimulation was used to enhance the exposure treatment of OCD in a patient with schizotypal personality disorder. Although treatment was focused on the OCD, restricted environmental stimulation was incorporated due to the attentional problems found in patients with schizotypal personality disorder. In sum, clinicians should be alert to the presence of a comorbid personality disorder as a potential prognostic indicator. Therapeutic progress should be carefully monitored and the treatment strategy may need to be reevaluated if progress is not made within the period of brief therapy. As effective treatment for Axis II conditions typically requires a longer period of time than treatment for Axis I conditions alone (Kopta, Howard, Lowry, & Beutler, 1994), it may be that a more intensive, longer course of treatment is required to address Axis II comorbidity.

Treatment addressing anxiety and therapeutic barriers. In order to decrease client ambivalence with regard to therapy, motivational interviewing (MI) represents one promising therapeutic technique. MI is a client-centered therapy that augments intrinsic motivation to change by openly discussing and resolving ambivalence to change (Miller & Rollnick, 2002). Comparison studies

conclude that clients receiving MI prior to CBT have superior homework compliance than clients receiving only CBT (Westra & Dozois, 2006). Kertes, Westra, Angus, and Marcus (2010) reported that clients receiving MI prior to CBT viewed their role in therapy as more active, while clients receiving only CBT who viewed their role in therapy as more passive. Simpson and colleagues (2008) noted some success in using MI as an adjunct to exposure and ritual prevention in the treatment of OCD, although the authors note that the intervention is likely to be successful only for patients whose ambivalence keeps them from participating fully in treatment, are able to access this ambivalence in session, and value something more than the status quo. Further study is needed to elucidate the full function of Motivational Interviewing in enhancing treatment for anxiety disorders.

It is also important for therapists to recognize the value of addressing any therapeutic alliance ruptures that may occur in treatment. In a CBT framework, therapeutic alliance is the foundation upon which the technical aspects of the intervention rest, so it is crucial for this alliance to be fostered. Safran and Muran (2000) suggest several direct and indirect ways of repairing a ruptured therapeutic alliance. Direct methods include providing rationale, examining core interpersonal themes with clients, and clarifying any misunderstandings. Indirect methods may involve changing the task or goal, reframing meaning of the task, displaying empathetic characterization, and helping to provide a corrective emotional experience. Overall, it is important for the therapist to recognize any problems in therapeutic alliance early and actively intervene to prevent interference in the therapeutic process.

Conclusions and Future Directions

In conclusion, as the anxiety literature moves beyond first generation efficacy studies, it becomes increasingly important to examine prognostic indicators in the effective treatment of anxiety. One promising area of the research literature is translational research that addresses narrowing the "scientist-practitioner gap" by implementing empirically supported treatments in the field. It is widely acknowledged that there is a need for more collaboration between clinician and researcher in the design and implementation of psychotherapy outcome research (Goldfried & Wolfe, 1998). This collaboration could help inform the study of integrated treatments that incorporate techniques from different therapeutic perspectives. The field is also in dire need of treatment manuals that provide flexibility and guide the clinician in handling problems that may arise during the course of treatment (e.g., ruptures in therapeutic alliance, homework noncompliance).

An additional direction for future anxiety research concerns the translation of nomothetic findings into idiographic treatment plans relevant to practitioners. Barlow and Nock (2009) discuss that, although the individual serves as the principle unit of analysis in the science of psychology, most psychological studies are conducted by comparing aggregated data from groups of individuals. Problems then result from generalizing a nomothetic result to an idiographic situation. The authors urge researchers to emphasize idiographic strategies that can be integrated into existing nomothetic research approaches in both clinical and basic science settings. While this is a practical and efficient approach, it can also directly address the causal relationships between key treatment-related variables.

Thus, to better understand the clinical picture, it may be necessary for researchers to focus more on individual cases than group averages in treatment response. In a recent poll of leaders of the field, one researcher noted, "it is the non-responders that should generate the research questions" (Donald F. Klein, as quoted in Norton, Asmundson, Cox, & Norton, 2000, p. 94). In this way, future research may best be guided by attending to those who do not respond to standard CBT protocols and attempt to better adapt and tailor treatment to these individuals. Idiographic approaches could then build on nomothetic findings to further advance our knowledge of effective interventions for the complications and variants of anxiety disorders.

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3 Prognostic Indicators of Treatment Response for Children with Anxiety Disorders

Lara J. Farrell, Allison M. Waters, Ella L. Milliner, and Thomas H. Ollendick

Anxiety disorders are the most common mental health problems in youth, affecting 8–27% of youth (Costello, Egger, & Angold, 2005). These disorders represent serious mental health problems for children and adolescents and lead to daily distress and impairment, peer and social relation problems (Chansky & Kendall, 1997; Langley, Bergman, McCracken, & Piacentini, 2004; Piacentini, Peris, Bergman, Chang, & Jaffer, 2007; Strauss, Forehand, Smith, & Frame, 1986), and significant difficulties in academic achievement (Kessler, Foster, Saunders, & Stand, 1995; King & Ollendick, 1989). Additionally, anxious youth often have poor self-esteem, more physical problems, and greater family conflict and distress than their peers (Ezpeleta, Keeler, Alaatin, Costello, & Angold, 2001; Harter, Conway, & Merikangas, 2003; Strauss, Frame, & Forehand, 1987). If untreated, childhood anxiety disorders tend to be chronic and unremitting in their course (Aschenbrand, Kendall, Webb, Safford, & Flannery-Schroeder, 2003; Keller, et al., 1992; Pine, Cohen, Gurley, Brooks, & Ma, 1998) and predict the development of other psychopathology later in life (Last, Perrin, Herson, & Kazdin, 1996; Woodward & Fergusson, 2001) including depression (Brady & Kendall, 1992; Cole et al., 1998; Pine et al., 1998; Seligman & Ollendick, 1998), externalizing disorders, and substance use disorders (Bittner et al., 2007, Costello et al., 2003, Last et al., 1996).

The seriousness of child internalizing problems such as anxiety disorders and the development of subsequent depression is highlighted by the World Health Organiation (WHO) prediction that by 2030, internalizing problems will be second only to HIV/AIDS in burden of disease (developed and developing countries combined; Mathers & Loncar, 2006). While treatment research for child anxiety has received a surge in interest over the past two decades, providing evidence for favorable treatment outcomes, there remains considerable room for improvement with less than 50% of children and youth evidencing full recovery following our best psychosocial treatments (e.g., Silverman, Pina, & Viswesvaran, 2008). Improving early identification, access to treatment, and understanding the predictors and moderators of treatment response is the current challenge for anxiety disorder researchers in order to improve the prognosis of children and youth most vulnerable to anxiety disorders. This chapter provides a review of the current state of treatment research for child anxiety disorders and will discuss what is currently known about predictors and moderators of treatment outcome.

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The review provides a comprehensive analysis across child anxiety disorder research by focusing on (1) child anxiety generally, including treatment outcome across generalized anxiety disorder, separation anxiety disorder (SAD), and social phobia (SoP); (2) latest research in child-specific phobia treatment; and (3) current evidence for pediatric obsessive–compulsive disorder (OCD). These three diagnostic categories require a separate focus of review given that each requires a specific and somewhat unique approach to treatment and given that each has a separate and independent treatment outcome literature.

Kraemer and colleagues (Kraemer, Wilson, Fairburn, & Agras, 2002), as well as March and Curry (1998), specify that predictors of treatment response are variables that exist prior to treatment and are related to treatment outcome. A predictor variable is said to have a main effect on outcome, meaning that its impact is not specific to a particular treatment condition (Garcia et al., 2010). Predictors of treatment outcome therefore inform us "for whom" and under "what conditions" treatments work. For example, a given treatment might work better for girls than boys or for younger children than older children. Of importance, predictor variables differ from moderator variables. A moderator variable, not unlike a predictor variable, is associated with treatment outcome; however, a moderator variable also predicts differential response to two or more treatments. As such, a moderator variable must *interact* with treatment assignment to specify for whom a specific treatment works. For example, a reinforcement-based program might work best for younger children whereas a cognitive-based procedure might work best for older children. This distinction is an important one because not all predictor variables are moderators of treatment outcome. The examination of predictors and moderators of treatment response presents a challenge in treatment research literature, due to the large sample sizes needed to conduct appropriate statistical analyses and, furthermore, the need for RCT designs using at least two active treatment conditions in order to establish moderator variables. As a result, the existing literature into predictors and moderators of treatment response is minimal, yet slowly growing with the publication of a number of large-scale multicenter RCT for various anxiety disorders in children and youth (see POTS, 2004; Walkup et al., 2008).

Childhood Anxiety Disorders

Current Status of Treatment Outcome

The vast majority of treatment research for childhood anxiety disorders has focused on cognitive–behavioral therapy (CBT) or variants of CBT (see Silverman et al., 2008 for review). Commonly used child CBT programs teach children to recognize emotional and physiological signs of anxiety and to employ somatic and cognitive strategies for managing these symptoms, in addition to encouraging children to gradually expose themselves to increasingly feared stimuli (Spence, 1994; Kendall et al., 2005). In the seminal work by Kendall (1994) and Kendall et al. (1997) on the efficacy of CBT for childhood anxiety disorders, 64% of anxious children receiving individual child-focused CBT no longer met criteria for an anxiety disorder by post-treatment compared to just 5% in the wait-list control, results that were maintained at 12-month follow-up. Using a group CBT format (GCBT), Silverman et al. (1999) found that 64% of children with anxiety disorders were diagnosis-free by post-treatment assessment compared with only 13% of wait-list children with effects that were maintained at 12-month follow-up. Since then, others have shown that anxious children improve significantly with CBT whether delivered in a group or individual format (e.g., Manassis et al., 2002; Flannery-Schroeder & Kendall, 2000). Consequently, individual or group CBT has been deemed to meet criteria as an efficacious treatment for childhood anxiety disorders (Silverman et al., 2008).

Based on extensive evidence regarding the role of parent factors in the etiology and maintenance of anxiety disorders (Craske & Waters, 2005), other work has examined the efficacy of CBT when parents of anxious children are involved in treatment. Multiple studies have reported superior

treatment outcomes from CBT interventions including parent anxiety management training compared with CBT alone (e.g., Barrett, Dadds, & Rapee, 1996; Cobham, Dadds, & Spence, 1998; Bogels & Siqueland, 2006; Rapee, 2000, 2003; Rapee, Abbott, & Lyneham, 2006; Spence, Holmes, March, & Lipp, 2006; Wood, Piacentini, Southham-Gerow, Chu, & Sigman, 2006). However, long-term superiority of CBT including parent training has been inconsistently observed (e.g., Barrett, Duffy, Dadds, & Rapee, 2001; Cobham, Dadds, Spence, & McDermott, 2010), and other studies have not demonstrated greater effects for a parental component (Nauta, Scholing, Emmelkamp, & Minderaa, 2001, 2003; Spence, Donovan, & Brechman-Toussaint, 2000). The traditional assumption that parenting casually influences child anxiety was challenged in a study by Silverman, William, Jaccard, and Pina (2009). In this study, Silverman and colleagues (2009) suggested that the association between negative parenting behavior and child anxiety may in fact reflect the influence of child anxiety on parenting variables, as opposed to vice versa. Hence they argue that as child anxiety improves, negative parenting similarly improves, providing one possible explanation why parental involvement versus noninvolvement is often deemed similarly efficacious (e.g., Silverman et al., 2008).

Interestingly, however, younger children (7–10 years) compared to older children (11–14 years) appear to respond better to CBT when supplemented with parent anxiety management (Barrett, 1998). Indeed, recent work has shown that CBT including parental involvement was effective in reducing clinical anxiety in 69% of anxious children as young as 4–7 years of age which resembles clinical outcomes with older children (Hirshfeld-Becker et al., 2010). Related studies have shown that between 59 and 80% of anxious children between 4 and 7 years of age were diagnosis-free following CBT interventions delivered solely with parents; rates that were comparable to those obtained from more traditional child–parent conditions (e.g., Mendlowitz et al., 1999; Cartwright-Hatton, McNally, & White, 2005; Thienemann, Moore, & Tompkins, 2006; Waters, Ford, Wharton, & Cobham, 2009). This work highlights that direct involvement of very young anxious children may not be necessary in treatment and could lead to significant time and resource savings.

Significant inroads have similarly been made in disseminating CBT to a greater number of children with anxiety disorders. This has included parent-implemented bibliotherapy supplemented with written materials (Rapee et al., 2006), and technological advancements including use of the Internet, DVD, and email/phone supplemented bibliotherapy implemented by parents of anxious children (Khanna & Kendall, 2010; Lyneham & Rapee, 2006; Spence et al., 2006). Bibliotherapy-based CBT relying on written materials was found not to be as effective as clinic-based CBT (Rapee et al., 2006); however, studies employing Internet delivery of CBT and email/phone supplemented bibliotherapy yielded treatment outcome rates between 56 and 81%, paralleling those of clinic-based CBT trials (Khanna & Kendall, 2010; Lyneham & Rapee, 2006; Spence et al., 2006). These innovations in CBT dissemination have high public health significance given the high prevalence of childhood anxiety disorders and their capacity to reach anxious children in rural and remote regions.

As can be seen, considerable advancement has been made over the past two decades establishing the efficacy and accessibility of CBT for childhood anxiety disorders. While approximately 60% of children are diagnosis-free following treatment (James, Soler, & Weatherall, 2006), not all children respond to CBT, with numerous factors contributing to relapse, dropout, or nonresponse to treatment. This has spurred new efforts to improve outcomes from CBT for childhood anxiety disorders, including the combination of CBT with pharmacotherapy agents, such as selective serotonin reuptake inhibitors (SSRIs; e.g., sertraline). In a recent large multisite US study of clinically anxious children, Walkup and colleagues (2008) were the first to evaluate a combined (CBT) and pharmacological treatment (SSRI) for childhood anxiety disorders including GAD, separation anxiety disorder, and social phobia. The authors concluded that combined CBT and SSRI (sertraline) produced superior

outcomes to CBT alone, SSRI alone, or placebo. These findings suggest that, for anxious children, adding SSRI medication to quality CBT offers the most favorable outcomes, relative to each treatment alone. Further research examining predictors and moderators of treatment response is needed in order to develop prescribed treatment options for individual children.

Predictors of Treatment Response

Silverman et al. (2008) provide the most recent and comprehensive systematic review of the treatment literature for child anxiety disorders, including 32 group design treatment studies spanning almost two decades since Kendall's (1994) seminal CBT treatment trial. The authors of this chapter dedicate a section to the cumulative evidence on predictors and moderators of treatment response arising from the published literature. Interestingly, there is no current study in the child anxiety treatment literature (excluding OCD-specific research) that systematically examines moderators of treatment response; however, there are several studies that explore predictors of treatment success and failure. Based on this literature, almost all the evidence for significant predictors of treatment response relate to familial factors, including maternal and paternal psychopathology, parenting approaches, and general family functioning.

A number of studies have found that parental psychopathology plays a significant role in response to child anxiety disorder treatment. Berman and colleagues (2000) examined predictors of response in 106 youth who participated in one of two treatment studies published by Silverman and colleagues (i.e., Silverman, Kurtines, Ginsburg, Weems, Lumpkin, et al., 1999; Silverman, Kurtines, Ginsburg, Weems, Rabian, et al., 1999). A number of significant familial predictor variables emerged, including parental symptoms of depression, fear, hostility, and paranoia. Interestingly, the importance of these parental predictors variables appeared to be related to child age, with variables being significant for child samples but not so for adolescents. Furthermore, the importance of parental predictor variables also diminished when comparing children who received group treatment, relative to those whom received individual CBT. Based on this chapter, the findings suggest that parental symptoms of psychopathology are more problematic in treating younger children and in individual delivery of CBT (Berman et al., 2000).

Crawford and Manassis (2001) specifically examined the impact of a wide range of familial variables on child outcomes in their treatment study of 61 children and youth aged 8–12 years. This study examined predictors of response across clinician-rated improvement, mother-rated improvement, and child self-reported improvement. Child ratings of family dysfunction and frustration were significant predictors of both clinician and child-rated treatment response. Further, mother and father reports of family dysfunction and maternal parenting stress predicted mother-rated treatment response, while father reported somatization also predicted child-rated treatment response. This study highlights that family dysfunction plays an important role in treatment response for children with anxiety disorders. Victor, Bernat, Bernstein, and Layne (2007) report similar findings in 61 treatment-seeking children (aged 7–11 years), with higher family cohesion associated with greater symptom reduction in child anxiety. This study, however, found no effect for parenting stress or parental psychopathology.

Since Silverman and colleagues' (2008) systematic review, Liber and colleagues from the Netherlands (2008) have published an examination of parenting variables and parental anxiety and depression as predictors of treatment outcome in child anxiety CBT treatment. This study included 124 outpatient treatment-seeking children, aged 8–12 years, as well as 123 mothers and 108 fathers. In this study, paternal anxiety and depression symptoms, paternal rejection, and maternal emotional warmth were significantly related to a less successful response to treatment. Interestingly, mother's and father's levels of anxiety, depression, and rejection were not significantly different at baseline; however, only father's experience of these variables impacted on child treatment outcome, suggesting a pivotal role of

paternal psychopathology on children's response to treatment. Maternal emotional warmth is a surprising predictor and largely inconsistent with other studies; however, the authors of this study suggest that children's ratings of maternal emotional warmth may actually reflect maternal overinvolvement—an interesting hypothesis which requires further exploration.

In regard to maternal anxiety as a predictor of child treatment response, Cooper, Gallop, Willetts, and Creswell (2008) found support for the significant role of maternal anxiety in predicting a less favorable treatment response in 55 children referred to the local community health service. This study also provided evidence for a specificity effect of maternal anxiety—with maternal social phobia related to a poorer response, whereas maternal GAD did not have a significant effect on child treatment response. Likewise, Gar and Hudson (2009) found a significant effect of maternal anxiety on child anxiety response to treatment in their study of 48 clinically anxious 6–14-year-old children. In this study, maternal anxiety was a significant predictor at posttreatment, with only 28% of children improved with anxious mothers, compared to 58% of children with non-anxious mothers. This difference in response rate was not, however, significant at 12-month follow-up suggesting that maternal anxiety might be related to a slower treatment response in children with anxiety.

Apart from familial and parenting variables, there is little support for other significant predictors of response to child anxiety treatment. Kendall and colleagues (Kendall, 1994; Kendall, Brady, & Verduin, 2001; Treadwell, Flannery-Schroeder, & Kendall, 1995) have examined child gender, ethnicity, comorbidity, perceptions of therapeutic relationship, and therapist perceptions of parental involvement as possible predictors of child treatment response and found none of these variables to be significant predictors (Silverman et al., 2008). In regard to the important issues of comorbidity, Ollendick and colleagues (2008) have published a review paper exploring specifically the role of comorbidity on child treatment outcomes across child anxiety disorders, affective disorders, and disruptive behavioral disorders (including attentional disorders and oppositional disorders). This study reviewed 43 child anxiety randomized controlled trials and found that only 14 of these trials systematically examined the predictive or moderating role of comorbidity on treatment outcome. Ollendick and colleagues (2008) found that there were only two trials in the published general anxiety literature (excluding studies from the OCD treatment literature, which are reviewed later in this chapter) that reported significant, albeit small, differences on treatment response depending on comorbidity. Berman et al. (2000) found that children with comorbid depression in a sample of anxious children from two Silverman et al. trials (Silverman, Kurtines, Ginsburg, Weems, Lumpkin, et al., 1999; Silverman, Kurtines, Ginsburg, Weems, Rabian et al., 1999) were more likely to be in the treatment failure group. Rapee (2003) examined the influence of comorbidity across three groups of anxious youth—no comorbidity, comorbid anxiety diagnosis, and comorbid non-anxiety diagnosis—and found that children with comorbidity had higher parent-reported externalizing symptoms from posttreatment to follow-up and attended fewer therapy sessions relative to the no comorbidity group. The prevailing evidence therefore, based on Ollendick et al.'s (2008) comprehensive review of this issue, suggests that comorbidity has little impact on child anxiety treatment response (excluding OCD), although large studies that systematically examine comorbidity as a moderator of treatment response are needed.

And finally, Liber and colleagues (2010) investigated the associations between treatment adherence, child–therapist alliance, and child clinical outcomes across individual and group treatment of 52 anxious youth (aged 8–12 years). This study found that neither treatment adherence nor therapeutic alliance predicted child outcomes; however, results did provide support that a strong alliance in individual therapy was associated with better diagnostic outcomes relative to group CBT. Contrary to initial results, using a more stringent measurement of outcome, child alliance was associated with greater reliable change—a finding that is also inconsistent with findings of Kendall

and colleagues (see Kendall, 1994; Kendall et al., 1997). Further research is clearly necessary to examine the role of therapeutic process variables in predicting child outcomes. Research specifically exploring the role of *different* therapeutic processes variables across individual and group therapy (e.g., child alliance versus group cohesion; Liber et al., 2010) would also progress our understanding about the role of technical and process variables in improving treatment outcome across different modalities of treatment.

The cumulative research to date in the general child anxiety treatment literature suggests that family functioning, parental rearing approaches (i.e., rejection, warmth, hostility), and parental psychopathology (i.e., depression and anxiety) are consistently important predictors of treatment response for children with anxiety disorders. Interestingly, research is suggestive of a stronger influence of father's psychopathology on child outcomes (e.g., Liber et al., 2008), highlighting the need for more focused research on the role of fathers in both the etiology and treatment of child anxiety and most certainly greater efforts to involve fathers in child anxiety treatments. Gender, ethnicity, and comorbidity (apart from depression, see Berman et al., 2000) appear to not be important in relation to a child's responsiveness to treatment, and further research exploring the impact of therapeutic alliance and treatment adherence on child treatment response is necessary. In regard to moderating variables, to date, there are no studies that systematically explore this in the child anxiety treatment literature; however, child age and parental psychopathology are hypothesized to moderate response with regard to parent involvement in CBT. Evidence for this comes from two studies, including one RCT of individual CBT versus CBT plus family involvement (Barrett et al., 1996), which demonstrated that younger children responded significantly better to CBT involving a family component relative to CBT alone, and that for older children there were no differential effects. Secondly, Cobham et al. (1998) found that when anxious children had at least one parent with clinical anxiety, children responded significantly less well at posttreatment to CBT alone relative to CBT including a parental anxiety management module. Further research is clearly needed, involving multiple group design RCTs to elucidate moderators of treatment response for child anxiety disorders.

Specific Phobias in Children and Adolescents

Current Status of Treatment Outcome

Behavioral and cognitive–behavioral procedures have also received strong empirical support in the treatment of childhood phobias (King, Muris, & Ollendick, 2005; Ollendick, Davis, & Sirbu, 2009). Techniques such as in vivo exposure, participant modeling, and reinforced practice or contingency management have been shown to be particularly effective with these youth. For those youth with a specific phobia diagnosis, three large randomized controlled trials (Öst, Svensson, Hellström, & Lindwall, 2001; Ollendick et al., 2009; Silverman et al., 1999) and two smaller clinical trials have been conducted (Flatt & King, 2010; Muris, Merckelbach, Holdrinet, & Sijsenaar, 1998).

In the first RCT, Silverman and colleagues (1999) compared the effectiveness of exposure-based cognitive self-control (SC) and exposure-based contingency management (CM) treatments to an education support (ES) control condition. Eighty-one children and adolescents (aged 6–16 years) from the United States (US) participated. Youth presented with a diverse range of phobias. Treatments were manualized and involved ten sessions (80 min each) during which children and their parents were first seen separately by the same therapist and then seen conjointly at the end of the sessions. Findings were mixed. Although the three treatment conditions showed comparable improvements on child self-report and parent report measures at posttreatment, significant differences were observed between the conditions on two major clinically significant treatment outcome measures (e.g., diagnostic outcomes and fear thermometer ratings). Eighty-eight percent of participants in the SC condition were recovered (no longer met diagnostic criteria for a pho-

bia) at posttreatment, compared to 55% in the CM condition and 56% in the ES condition. Additionally, 80% of participants in the SC and CM conditions reported either no or little fear on their fear thermometer ratings (a measure of subjective distress toward their feared object or event) at posttreatment compared to 25% in ES condition. Hence, Silverman et al. found considerable support for exposure-based therapies particularly SC in the treatment of youth phobias.

More recently, cognitive–behavioral procedures have been incorporated into an intensive one-session treatment (OST) package in the treatment of phobias in children and adults (Öst, 1989). OST involves a single, 3-hour session of massed exposure which includes aspects of psychoeducation and skills training, cognitive restructuring, graduated in vivo exposure, participant modeling, and reinforced practice (Cowart & Ollendick, 2013). In a small randomized clinical trial, Muris et al. (1998) compared OST to EMDR and a computerized exposure control group in 26 spider phobic children and adolescents from the Netherlands. OST was found to be superior to the two other interventions on measures of subjective distress and ratings of anxiety during the behavioral avoidance test. Unfortunately Muris and colleagues did not report diagnostic recovery rates.

In a subsequent trial conducted in Sweden, Öst et al. (2001) evaluated the relative efficacy of OST alone and OST with parent present to a wait-list control condition. In the parent present condition, the parent sat in on the session with the child and was enlisted primarily as a support figure to the child during the in vivo exposures. Sixty youth (7–17 years) with a diverse range of phobias participated in the study. Both OST conditions were found to be superior to the wait-list control condition on the primary outcome measures of subjective distress, behavioral avoidance, and independent assessor ratings of the severity of phobias at posttreatment. However, as with Silverman et al. (1999), the three groups did not differ significantly on child self-report and parent report measures following treatment. Overall, there was a trend for youth in the alone OST condition to fare better than youth in the parent present condition; however, no significant differences were observed between the two conditions. This outcome was not expected as it was hypothesized that the presence of the parents during treatment would facilitate change. Although speculative, this unexpected outcome may have been due to the fact that most of the parents were not actively involved in the treatment process; rather, for the most part, they were passive observers. Nonetheless, treatment gains in both groups were maintained at one-year follow-up.

In the largest randomized trial, Ollendick et al. (2009) assigned 196 children and adolescents (7–16 years) with various specific phobias to OST (alone, without parent present), education support treatment, or a wait-list control condition. Participants were recruited from Sweden and the USA. OST and education support treatment were superior to the wait-list control condition. Furthermore, OST was found to be superior to education support treatment on clinician ratings of phobic severity, percentage of participants diagnosis-free (55% OST vs. 23% EST) at posttreatment, child ratings of anxiety during the behavioral avoidance test, and treatment satisfaction as reported by youth and their parents. Treatment effects were maintained at six-month follow-up. However, similar to Öst et al. (2001) and Silverman et al. (1999), no differences were observed on child self-report measures.

Finally, Flatt and King (2010) replicated the Ollendick et al. (2009) study with a smaller sample of 43 Australian phobic youth aged 7–17 years. Children and adolescents were randomized to OST (alone, without parent present or involvement in the treatment), a psychoeducation package or a wait-list control. Both active treatments were superior to the wait-list control on the behavioral avoidance test and percentage of participants who were diagnosis-free at posttreatment. Unexpectedly, however, differences in treatment effectiveness were not found between OST and psychoeducation treatments at posttreatment and one-year follow-up. This unexpected finding might be explained by the psychoeducation program used by Flatt and King (2010). In addition to education and support, this condition also taught participants about gradual exposure and actively

encouraged them to practice exposure in real-life situations. Hence, exposure may have been the active ingredient in both interventions.

Overall, the aforementioned studies provide strong empirical support for cognitive and behavioral treatments for phobic youth. In particular, OST is an effective and rapid treatment for phobic youth, with four randomized trials in four different countries supporting its use (Flatt & King, 2010; Muris et al., 1998; Ollendick et al., 2009; Öst et al., 2001). Interestingly, to date, no large-scale randomized trials have been conducted with less intensive exposure programs delivered in a more standard weekly format over a period of time as is typical in most outpatient settings. Nor, for that matter, have the more intensive programs been compared to the less intensive ones. Moreover, the potential role of parents in the treatment of phobic youth has not yet been systematically explored—however, such a trial is presently under way by Ollendick and colleagues in the USA.

Predictors of Treatment Response

Unfortunately, there is limited information about predictors of treatment response in youth with specific phobias, and worse still, there is presently little to no evidence for moderators of treatment outcome with specific phobias (Seligman & Ollendick, 2011). We examine the findings for the major randomized control trials and smaller clinical trials reviewed earlier in this chapter, which showed the efficacy of cognitive–behavioral procedures in the treatment of well-characterized youth with specific phobias. In the first major randomized controlled trial, Silverman and colleagues (1999) examined the utility of contingency management, self-control, and education support in the treatment of childhood anxiety, including phobias. As will be recalled, this study showed that exposure-based contingency management and exposure-based self-control treatments proved more efficacious than an education support condition on major treatment outcome variables (e.g., fear ratings, diagnostic outcomes). As previously reviewed, Berman, et al. (2000) explored the predictors of treatment success for the two active exposure-based interventions and found that children in the treatment failure group had significantly higher rates of comorbid depressive diagnoses and higher levels of self-report trait anxiety and depression than those in the treatment success group. Similarly, parents in the treatment failure group also reported higher levels of depression, fear, and hostility for themselves than did parents in the treatment success group.

In contrast to Silverman et al. (1999), the other randomized control trials and smaller clinical trials for youth with specific phobias implemented exposure-based treatments in one session of approximately 3 h duration (Muris et al., 1998; Ollendick et al., 2009; Öst et al., 2001). Muris and colleagues (1998) evaluated the relative efficacy of OST, EMDR, and a computerized exposure control group in a small clinical trial with spider phobic youth. OST was found to be superior to the other two interventions on measures of subjective distress and ratings anxiety during the behavioral avoidance test. Unfortunately, however, Muris and colleagues did not report on diagnostic recovery rates or investigate predictors of treatment outcome in their study.

It will be recalled that Öst and colleagues (2001) compared two variants of the intensive OST to a wait-list control condition. As expected, the OST groups responded better than the simple passage of time in the wait-list group. Clinical improvement was defined in various ways, but for our purposes here, we will use the criterion that was similar to that used by Silverman and colleagues (1999): significant reductions in clinical severity on the structured diagnostic interview. Predictor variables in this study included age, gender, comorbidity, and type of phobia (e.g., animal, situational, environmental, and blood-injection-injury type). Measures of psychopathology in the parents were not obtained. Findings revealed that none of the predictor variables was related to treatment outcome as defined by significant reductions in clinical severity on the diagnostic interview. It should be noted that on a secondary measure of treatment outcome, improvements on a behavioral approach test, girls responded better than

boys as did youth with animal phobias compared to those with other types of phobias.

Ollendick et al. (2009) compared this intensive intervention to not only a wait-list control condition but also to an education support condition. As expected, youth in the OST responded better than those in either the education support or the wait-list control condition: These salutatory effects were found both in terms of those who evinced reduced clinical severity ratings and those who were diagnosis-free. Predictor variables included age, gender, phobia type, and comorbidity of diagnosis (see also Ollendick et al., 2010). As with Öst et al. (2001) these variables were not related differentially to treatment success or treatment failure. Additional analyses are currently under way to explore parental psychopathology and family functioning variables and their relations to treatment success.

Finally, Flatt and King (2010) replicated Ollendick et al. (2009) and randomized youth to OST, a psychoeducation package or a wait-list control. Both active treatments were superior to the wait-list control on the behavioral avoidance test and percentage of participants who were diagnosis-free at posttreatment. Predictor variables examined in this study included age, gender, and phobia type. Consistent with Öst et al. (2001) and Ollendick et al. (2009), these variables did not predict treatment outcome.

Collectively, although the findings with diagnosed children and adolescents are limited, these studies show that sociodemographics of the child (e.g., age, gender, socioeconomic status, and ethnicity) and severity of the diagnosis as well as type of phobia are not related to treatment success or failure. In the two one-session treatments that explored comorbidity (Ollendick et al., 2009; Öst et al., 2001), the presence of comorbidity was not related to treatment outcome; however, in the 10-week exposure-based program of Silverman and colleagues (1999), comorbidity with depression was related to outcome. Still, even in this study, only a few of the youth were comorbid with depression and the findings have not yet been replicated. Further, in this latter study, parental psychopathology characterized by anxiety, depression and hostility, and heightened anxiety and depression in the children themselves were associated with treatment failure. These results also remain to be replicated at this time. As is evident, the study of predictors of treatment response for youth with specific phobias is extremely sparse at this time and much awaits to be done before we will have a clear picture of the youth for whom and under what conditions our treatments will be shown to be effective.

Obsessive–Compulsive Disorder in Children and Youth

Current Status of Treatment Outcome

The OCD Expert Consensus Guidelines (King, Leonard, & March, 1998) for treatment of childhood OCD recommend CBT alone as the first-line treatment for children and adolescents with mild to moderate OCD and the combination of an SSRI medication in addition to CBT for severe OCD. The current status of treatment research, including long-term outcome studies and meta-analytic reviews, provide support for these current guidelines, with evidence to support the efficacy for CBT, based on exposure plus response prevention (ERP), either alone or in combination with a serotonin reuptake inhibiting (SRI) medication (see Abramowitz, Whiteside, & Deacon, 2005; Barrett, Farrell, Pina, Peris, & Piacentini, 2008; Barrett, Healy-Farrell, Piacentini, & March, 2004).

CBT for children and adolescents with OCD is typically based on March and colleagues' individual CBT protocol ("How I ran OCD off My Land"; see March et al., 1994; March and Mulle, 1998) and involves three treatments components including (1) psychoeducation, "externalizing OCD," anxiety management, and cognitive therapy; (2) intensive therapist assisted ERP and associated homework; and (3) maintenance of gains, including problem-solving and relapse prevention. ERP is the active ingredient in CBT for OCD and involves exposing patients to stimuli that triggers fear while simultaneously encouraging them to resist engaging in compulsive behaviors. Most approaches to child treatments for OCD also

involve a parent or family component; however, the nature and intensity of this aspect of treatment varies greatly. Parental or family adjuncts to CBT often includes psychoeducation, problem-solving skills, strategies to reduce parental involvement and accommodation to the child's OCD symptoms, along with encouraging family support of home-based ERP, and developing contingency management schedules to support ERP gains.

Barrett, Farrell, and colleagues (2008) published a systematic review of the current status of evidence-based for psychosocial treatments of pediatric OCD. Studies were evaluated for methodological rigor according to the classification system of Nathan and Gorman (2002) and were then assessed relative to the criteria for evidence-based treatments specified by Chambless et al. (1998), Chambless et al. (1996), and Chambless and Hollon (1996). Findings indicate that individual CBT with ERP for children and adolescents with OCD meets criteria for designation as *probably efficacious* based on the treatment literature to date. To meet criteria for designation as a *well-established* treatment (e.g., Chambless et al., 1998; Chambless et al., 1996; and Chambless & Hollon, 1996), a treatment requires at least two "good" RCTs from different investigative teams showing the treatment to be superior to pill placebo or alternate treatment or equivalent to an already established treatment in studies with adequate statistical power. Furthermore, outcomes from the Pediatric OCD Treatment Study (POTS, 2004) provide evidence to support combination treatment of individual CBT plus sertraline (SRI medication) as also meeting criteria for *probably efficacious*. Family-based CBT, delivered as individual or group therapy, was deemed as *possibly efficacious* based on the review by Barrett et al. (2008); however, with the publication of a more recent RCT examining family-based CBT for younger children with OCD (aged 5–8 years), this treatment would now meet criteria as *probably efficacious* also, given the favorable effects of CBT relative to relaxation training on remission rates (see Freeman et al., 2008).

The comparative effectiveness of CBT versus SRI medication alone and combination treatment (CBT + SRI) has been examined in a systematic review by O'Kearney, von Sanden, and Hunt (2010). O'Kearney and colleagues (2010) identified three studies (Asbahr et al., 2005; de Hann et al., 1998; POTS, 2004) to date, which have examined the relative efficacy of CBT versus medication alone and found no evidence to suggest that either CBT or medication alone were superior over the other on symptom severity or remission rates. Two studies were identified by O'Kearney et al. (2010) to have examined the relative efficacy of combined CBT and medication treatment over medication alone (Neziroglu et al., 2000; POTS, 2004), and one study has examined the relative efficacy of combined treatment versus CBT alone and medication alone in a placebo-controlled design (POTS, 2004). These studies were consistent in demonstrating that combined treatment (CBT + SRI) was superior to medication alone in reduction of OCD severity as well as remission rates; however, combined treatment did not differ significantly from CBT alone (O'Kearney et al., 2010). In the POTS trial (2004), there was an interesting and significant site X treatment condition interaction, indicating that while combined treatment was favorable over CBT at one site, it was not superior to0 CBT alone at another, with both condition providing very large effect sizes. Results from two further meta-analyses (Abramowitz, Whiteside, & Deacon, 2005; Watson & Rees, 2008) also provide consistent results, that is, that both treatments alone (CBT, SRI) and in combination are significantly superior to placebo, with large CBT treatment effect sizes relative to medium treatment effect sizes for pharmacotherapy. Taken together, the results of these systematic reviews provide evidence for the efficacy of CBT, either alone or in combination with an SRI medication, and support the recommendations outlined in the expert consensus guidelines.

In regard to the magnitude of change associated with CBT on children's OCD symptoms and disorder, examination of effect sizes provides a favorable picture. Barrett and colleagues (2008) in their systematic review provide estimates of between-group effect sizes (CY-BOCS ratings) ranging from 0.99 to 2.84 for type 1

studies (i.e., based on Nathan and Gorman's classification system, 2002) and within-group effect sizes on the CY-BOCS from 1.57 to 4.32 (individual CBT) and 0.82 to 1.15 (group CBT) for type 2 and type 3 studies. While effect sizes for CBT are impressive, examination of the actual percentage of children experiencing remission from disorder following treatment is less favorable with rates ranging from 40 to 85% across studies (Barrett, Healy-Farrell, & March, 2004; POTS, 2004; Storch et al., 2007; Waters, Barrett, & March, 2001). In fact, results from the largest multisite RCT (POTS, 2004) indicates that as many as 60% of children receiving CBT alone, 50% receiving combined CBT and serotonergic medication, and almost 80% receiving serotonergic medication alone fail to fully remit following treatments. Understanding predictors and moderators of treatment response for childhood OCD represents an important focus for psychological treatment research and will assist in (1) the refinement of our current best treatment approaches, (2) the development and evaluation of innovative interventions, and (3) the advancement of clinical guidelines for prescribing the most appropriate treatment for a given individual.

Predictors of Treatment Response

Although the majority of children and adolescents with OCD do experience clinically significant reduction in OCD symptoms following our best treatments, the outcomes in terms of remission rates provide less than optimal results, with at least 50% of children deemed as non-remitters (e.g., POTS, 2004). These findings suggest that one in two treatment-seeking children and youth will continue to suffer clinically significant OCD even after combined CBT and SRI treatment. There is therefore a pressing need to understand the predictors and moderators of treatment response in pediatric OCD, in order to determine appropriate ways to augment or refine our current best treatments and provide more effective management for those with difficult-to-treat OCD. In the pediatric OCD treatment literature there are few studies examining predictors of treatment outcome, due to the large sample sizes needed for these analyses. Furthermore, there are only two studies to date which examine moderators of treatment response, given that these studies require designs with more than one treatment condition. From the limited research conducted to date, however, there is some consistency with regard to variables that might define subgroups of children and adolescents with more difficult-to-treat OCD.

A recent review of the treatment literature published between 1985 and 2007 (Ginsburg, Newman Kingery, Drake, & Grados, 2008) identified 21 studies which examined predictor variables in pediatric samples of primary OCD. Of these studies, six evaluated CBT only, 13 evaluated medication treatment only, and two studies reported on combined CBT and medication (Ginsburg et al., 2008). In this chapter, Ginsburg and colleagues (2008) identified nine predictor variables that were examined in more than one study, including child gender, age, duration of illness/age at onset, baseline severity of OCD, type of OCD symptoms, comorbid disorders/symptoms, psychophysiological factors, neuropsychological factors, and family factors. The authors of this chapter concluded that gender, age, or duration of illness (age of onset) were not associated with treatment response. Baseline severity of OCD symptoms and family dysfunction, however, were associated with poorer response to CBT, and comorbid tics and externalizing disorders were associated with poorer outcome in medication only studies. Garcia and colleagues (2010) have recently published an examination of predictors and moderators, based on the multisite POTS (2004) study, and examined 15 variables of interest across four categories, including demographic variables, severity of illness markers, comorbid disorders/symptoms, and family factors. This study found that higher baseline OCD symptom severity, OCD-related functional impairment (as rated by parents), higher externalizing symptoms, and higher family accommodation were all significantly associated with a poorer treatment response across treatment conditions in the POTS trial—which included

CBT alone, sertraline alone, combined treatment, and placebo control.

In addition to these findings, Storch and colleagues (2008) have added to the predictor of outcome literature, examining the impact of comorbidity on response to CBT treatment in a treatment-seeking sample of 96 youth with a primary diagnosis of OCD. In their study, Storch and colleagues (2008) found that having one or more comorbid conditions was associated with a poorer response to CBT outcome and that the number of comorbid condition was negatively related to outcome. Furthermore, Storch and colleagues (2008) found that the presence of comorbid externalizing disorders (i.e., attention deficit/hyperactivity disorder, oppositional defiant disorder, and conduct disorder) was associated with a poorer treatment response and that both externalizing disorders and depressive disorders were associated with lower treatment remission rates. The authors of this study did not find evidence to suggest that comorbid anxiety disorders or comorbid tic disorders were associated with a poorer response to CBT. The collective findings by Ginsburg and colleagues (2008), Garcia et al. (2010), and Storch et al. (2008) on the impact of comorbidity are also consistent with a recent study by Farrell and colleagues (2012), whom also found that higher frequency of comorbid conditions was associated with poorer response to CBT in children and youth with OCD, and that specifically, comorbid child disruptive behavioral disorders was associated with a poorer response to treatment.

Two studies have recently examined the important issue of moderators of treatment response in pediatric OCD; offering valuable information about which of the current best treatments available (e.g., CBT alone or combined CBT + SRI) is best for specific subgroups of clients with OCD. March and colleagues (2007) reported on the impact of comorbid tic disorder on outcomes in the POTS trial (2004), examining treatment response for the 15% of the POTS sample ($n = 17$ of 112) whom had a comorbid tic disorder. In patients *without* tic disorders, outcomes were consistent with the entire intent-to-treat sample (POTS, 2004) with combined treatment (CBT + sertraline) being superior to CBT alone, which was superior to sertraline alone, which was superior to the placebo condition (POTS, 2004). However, for the sample *with* comorbid tic disorders, sertraline alone did not differ significantly from the placebo condition, while combined treatment (CBT + sertraline) remained superior to CBT, and CBT remained superior to PBO. This finding, consistent with Ginsburg et al. (2008), provides strong evidence that children with comorbid OCD and tic disorders respond differentially to medication alone versus cognitive–behavioral treatments, which they appear to respond to equally as well as children without comorbid tic disorders. March and colleagues (2007) recommend that children with OCD and comorbid tic disorder should begin treatment with CBT alone or a combined treatment of CBT and SRI, given that medication alone does not provide any benefit over a placebo pill for these children.

Garcia and colleagues (2010) identified another moderator variable—family history of OCD. For those without a family history, combined treatment (CBT + sertraline) was superior to placebo and sertraline alone, and CBT alone was superior to placebo. However, for those with a family history of OCD, there were no significant differences in outcome across the treatment conditions. Inspection of the effect size, however, demonstrated smaller effects for those with a family history across all conditions. Further, for CBT monotherapy, this reduction in effect size was marked and in fact was 6.5 times smaller than those without a family history of OCD. Garcia and colleagues (2010) examined whether differences in degree of family accommodation was associated with this reduction in effect size and found that patients with and without family history of OCD did not differ in the amount of family accommodation. The authors conclude that a family history of OCD may attenuate CBT because this treatment generally requires more family support and engagement (e.g., with assisting the child in ERP homework), perhaps more so than medication compliance. For parents and family members with OCD, this degree of family involvement in therapy may actually serve to interfere with a child's treatment progress. The recommendation by Garcia and colleagues was that combined

treatment should be offered as first-line treatment in the instance of a family history of OCD, as combined CBT and sertraline was found to be more robust than CBT alone (2.5-fold decrease in effect size) in the POTS trial.

Given that family accommodation (Garcia et al., 2010; Merlo, Lehmkuhl, Geffken, & Storch, 2009), family dysfunction (Ginsburg et al., 2008), and family history of OCD (Garcia et al., 2010) have all been identified as attenuating treatment response, treatments specifically addressing family interactions and functioning are likely to improve outcomes. Furthermore, the issue of comorbidity of pediatric OCD warrants further consideration in terms of approaches to treatment. For children with tics disorders, CBT is an important first-line approach given that SRI medication appears to be less effective for these children (e.g., POTS, 2004). For children with comorbid OCD and externalizing disorders, it may be that we can improve outcomes for these children by developing and evaluating multicomponent treatments that first address externalizing symptoms and the impact of behavioral problems on the family, prior to addressing OCD symptoms.

Concluding Remarks and Future Directions

This chapter highlights three important issues based on the cumulative child anxiety disorder treatment research of the past two decades—(1) there is currently considerable evidence to demonstrate that cognitive–behavioral treatments are efficacious for the treatment of child anxiety, phobic, and obsessive–compulsive disorders, producing large effects sizes, good long-term maintenance of gains, and delivering clinically significant improvements in functioning; (2) while outcomes are broadly favorable and suggest overall improvement following treatment, there is considerable room for improvement, with approximately 1 in 2 children continuing to suffer symptoms of clinical significance following treatment, indicating more is needed in terms of understanding "who" are the nonresponders and "how" can we improve outcomes for them; and (3) our current understanding about specific predictors and moderators of treatment response across the child anxiety disorders is fairly limited, therefore hindering the development of innovative and idiographic treatment approaches targeting the more difficult-to-treat anxiety and phobic presentations. This chapter has, however, highlighted what is currently known about predicting treatment response for children and adolescents with an anxiety disorder, and we emphasize here what appears to be the important prognostic indicator's that may be the focus of future research enquiry and novel treatment developments.

For child anxiety disorders including social phobia, generalized anxiety disorder and separation anxiety disorder, familial factors such as parental psychopathology, family functioning, and parental rearing approaches appear to be important variables that influence a child's response to CBT. More specifically, maternal and paternal anxiety, depression (particularly in fathers), parental rejection and hostility, and family functioning and cohesion are all important aspects of the family environment that appear to play a role in a child prognosis. There is some evidence to suggest that familial variables may play a more pivotal role in the treatment of younger versus older youth and that parental involvement in treatment might be more important for younger versus older children; however, the research is not yet clear on these issues due to the absence of any moderation analyses. There is little evidence to suggest that specific comorbidities play an influencing role in child anxiety outcomes; however, child depression appears to be the one exception that should be considered more carefully. There is currently very limited research by comparison into prognostic indicators for child-specific phobias; however, research across the treatment trials conducted to date indicate that neither age, gender, nor type or severity of phobia effect response to treatment. Large-scale RCTs, however, are under way into OST of specific phobias by Ollendick and colleagues, which will provide data for analyses of both predictors and moderators of response in the near future which will inform this limited field of research.

The status of research into predictors and moderators of treatment response for pediatric OCD is the most progressed of the literatures reviewed in this chapter. The research to date, albeit not large, has involved sophisticated analyses of predictors and moderators of treatment response, based on the large multisite POTS (2004) trial. These analyses, combined with outcomes from meta-analysis reviews and individual treatment studies, have provided some emerging clarity and consistency about prognostic factors for children and youth with OCD. Obsessive–compulsive symptom severity, functional impairment, family dysfunction, and high family accommodation are all consistently related to poorer treatment response. Furthermore, unlike with other child anxiety disorders, comorbidity does seem to be important in predicting and moderating treatment response. Evidence suggests that externalizing disorders appear to *generally* reduce treatment success (Garcia et al., 2010; Storch et al., 2008) and comorbid tic disorders moderate outcome to medication-alone treatment, with children responding poorer to SRI treatment with a comorbid tic disorder (Ginsburg et al., 2008; March et al., 2007). Family history of OCD also appears to moderate response to CBT alone, with a considerable reduction in treatment effect size for CBT alone when there is a family history of OCD. This cumulative research into predictors and moderators of treatment response for pediatric OCD now provides evidence for some specific treatment recommendations, including (a) when OCD is comorbid with tics, children should be prescribed CBT alone or in combined with medication, over medication alone; (b) children with a family history of OCD may respond better to CBT when combined with an SRI; (c) family accommodation and dysfunction should be routinely assessed in the case of pediatric OCD and addressed in treatment; and (d) targeting comorbid externalizing symptoms and disorders in treatment for OCD might improve treatment success for those children with comorbid disorders.

The treatment literature for child anxiety disorders over the past two decades has most certainly come of age, providing evidence for efficacy, durability and, more recently, preliminary evidence on predictors and moderators of treatment response. The implications of this research suggest a number of important implications for clinical practice, including (1) when treating children with anxiety disorders, clinicians should also routinely assess for comorbid psychopathology, parental psychopathology, the quality of the parent and child interaction, and the quality of the family environment and general functioning of the family, to inform a family-based idiographic functional analysis and problem formulation of the child's anxiety; (2) clinicians should routinely involve parents in therapy for a child anxiety disorder and should carefully consider the type of involvement and intensity of parental involvement, offering a flexible idiographic formulation-informed approach (e.g.,, where there is parental anxiety present, the clinician might provide a parental anxiety management module, in addition to parental education and support for child anxiety, or when family functioning is poor and there is high parental rejection and criticism, the clinician may opt for a family therapy approach to delivering child CBT, involving parents in the therapy process thereby providing observational learning opportunities for parents in providing support and positive problem-solving approaches to assisting their child). Finally, this chapter of the treatment research and predictors of treatment response highlights the need for (3) routine ongoing assessment of a child's *progress and response* to CBT throughout treatment, so that clinician's can augment CBT when there appears to be poor responsiveness, in order to optimize treatment success. Examples of augmenting CBT include the addition of more intensive parental involvement in CBT or parental anxiety management; alternatively augmenting CBT with an SRI medication might be indicated when there is only a partial response to CBT, or when there is increased severity and impairment of anxiety/phobia/OCD, or when anxiety is combined with complex comorbidity; and/or increasing the intensity of CBT by offering sessions at home, twice weekly, or with combined telephone support.

Ongoing research into prognostic indicators of treatment response across child anxiety disorders, specific phobias, and OCD is necessary to inform clinical innovations and to ultimately improve outcomes for all children and youth. As our knowledge expands to this extent, so will our treatments improve, policies and practices evolve, and our clients and families benefit.

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4 Continuing to Advance Empirically Supported Treatments: Factors in Empirically Supported Practice for Anxiety Disorders

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Across the lifespan, anxiety disorders are highly prevalent. For adults, anxiety disorders are among the most common mental disorders, with 18.1 % meeting criteria for any anxiety disorder. The 12-month prevalence rates of different anxiety disorders range from 0.8 % (agoraphobia without panic disorder) to 8.7% (specific phobia) in adulthood (Kessler, Chiu, Demler, & Walters, 2005). Adults with anxiety disorders are often at risk for relationship impairment (Senaratne, Van Ameringen, Mancini, & Patterson, 2010), physical health concerns (Sareen et al., 2006), and occupational disability (Mancebo et al., 2008), as well as substance abuse (Kushner, Abrams, & Borchardt, 2000) and suicidality (Sareen et al., 2005). Prevalence rates in youth range from 10 to 20% (Chavira, Stein, Bailey, & Stein, 2004; Costello, Mustillo, Keeler, & Angold, 2004) and are associated with multiple impairments, including difficulties in peer relationships (Greco & Morris, 2005; Verduin & Kendall, 2008), comorbidity with other mental health disorders (Kendall et al., 2010; Masi, Mucci, Favilla, Romano, & Poli, 1999), and poor academic achievement (Van Amerigen, Manicini, & Farvolden, 2003). Given the prevalence and interference caused by anxiety disorders, the development, implementation, and evaluation of evidence-based therapies is warranted.

Treatments labeled variously as "behavioral," "cognitive," and "cognitive-behavioral" are the most widely studied psychological treatments for anxiety disorders (e.g., Deacon & Abramowitz, 2004). Evidence supports cognitive-behavioral therapy (CBT) as an efficacious treatment for both adults (for a review, see Deacon & Abramowitz, 2004) and children (see Ollendick & King, 2011; Silverman, Pina, & Viswesvara, 2008) with anxiety disorders. This chapter will first review the status of research surrounding empirically supported treatments (ESTs) for anxiety disorders in children and adults. Next, factors that potentially impact the delivery and/or outcomes of ESTs for anxiety disorders will be discussed, including comorbidity, familial and cultural components, and therapeutic process variables. Finally, future directions for research and practice will be offered.

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Empirically Supported Treatments for Anxiety Disorders: Social Phobia/ Separation Anxiety Disorder/ Generalized Anxiety Disorder/Specific Phobia

Because treatment of social phobia (SoP), separation anxiety disorder (SAD), generalized anxiety disorder (GAD), and specific phobia (SP) is very similar, the ESTs for these disorders will be reviewed together. Treatment for these disorders follows a cognitive-behavioral perspective of anxiety including physiological, cognitive, and behavioral components. Therapy addresses each of these components with various strategies, including somatic management techniques, cognitive restructuring, and behavioral exposure. With adults, CBT treatments for SoP incorporate cognitive restructuring, applied relaxation, exposure to feared stimuli, and social skills training (Jorstad-Stein & Heimberg, 2009; Rodebaugh, Holaway, & Heimberg, 2004). Similarly, for SP, CBT typically focuses on exposure to the relevant stimulus (e.g., an individual with a phobia of elevators rides elevators until the fear decreases), with consideration also given to the person's cognitive processing of the event. Exposure tasks can be in vivo, imaginal, and virtual reality (Rothbaum, Hodges, Smith, Lee, & Price, 2000), and some specific phobias have been treated in as little as one extended session (e.g., Ost, Alm, Brandberg, & Breitholtz, 2001). The one-session treatment (OST) has been defined as a "probably efficacious" treatment for adults with spider phobias, small animal phobias, and flying phobia. Research is needed on the mediators and moderators of OST (see review by Zlomke & Davis, 2008), and individuals with more complex symptoms may require additional sessions. Treatment of generalized anxiety disorder (GAD) may be complicated by the less clear-cut role of exposure tasks (Borkovec & Whisman, 1996), but CBT typically includes self-monitoring, relaxation training, cognitive restructuring, and worry exposures (Olatunji, Cisler, & Deacon, 2010). By also targeting interpersonal difficulties and emotional avoidance (Borkovec, Newman, Pincus, & Lytle, 2002), CBT outcomes have been improved (Newman, Castonguay, Borkovec, Fisher, & Nordberg, 2008). The findings from meta-analytic studies of psychological treatments for adults with SoP, GAD, and SP provide consistent positive support for CBT interventions (see reviews by Beidel, Turner, & Alfano, 2003; Butler, Chapman, Forman, & Beck, 2006; Deacon & Abramowitz, 2004; Olatunji, Cisler, & Deacon, 2010).

When working with youth, Albano and Kendall (2002) describe components of CBT for anxiety disorders as including (1) psychoeducation about anxiety disorders, (2) somatic management for physical symptoms, (3) cognitive restructuring, (4) exposure tasks, and (5) relapse prevention. One "empirically supported" CBT treatment for youth is the *Coping Cat program* (Kendall & Hedtke 2006a, 2006b). The *Coping Cat* consists of 16 sessions, separated into two segments: skills training and skills practice (exposure tasks), and has been adapted for adolescents (i.e., The *C.A.T. Project*; Kendall, Choudhury, Hudson, & Webb, 2002). Several randomized clinical trials (RCTs) have examined the efficacy of the *Coping Cat program*, with sample sizes ranging from 47 (Kendall, 1994) to 488 youth (Walkup et al., 2008). Overall, study findings indicate significant reductions in anxiety among children who participated in individual child CBT (ICBT; *Coping Cat program*) and family CBT (FCBT; Howard, Chu, Krain, Marrs-Garcia, & Kendall, 2000) compared to waitlist participants and to a family-based education/support/attention control (Kendall, 1994; Kendall et al., 1997; Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008). Additionally, gains were maintained at 1-year to 7.4-year follow-ups, and a meaningful percentage of successfully treated participants had reduced problems associated with substance use (Kendall, Safford, Flannery-Schroeder, & Webb, 2004; Kendall & Southam-Gerow, 1996).

The largest RCT, the Child Anxiety Multimodal Study (CAMS), evaluated the efficacy of CBT (the *Coping Cat program* for

7–13-year-olds; *C.A.T. Project* for 13–17-year-olds), sertraline (Zoloft), a combination of the two treatments (CBT + MED), and a pill placebo among 488 youth (ages 7–17). This trial was conducted at six different clinics (medical schools, hospitals, university clinics) across the United States. Response rates indicated very favorable outcomes, with 80.7% of CBT + MED participants, 59.7% of CBT participants, 54.9% of sertraline participants, and 23.7% of placebo participates found to be treatment responders (rated as "very much" or "much improved") at week 12 (Walkup et al., 2008). Additionally, various adaptations of the *Coping Cat program* have demonstrated efficacy, such as the Australian *Coping Koala* (Barrett, Dadds, & Rapee, 1996; Heard, Dadds, & Rapee, 1991), the Canadian *Coping Bear* (Manassis et al., 2002; Mendlowitz & Scapillato, 1994), and the Dutch *Coping Cat* translation (Nauta, Scholing, Emmelkamp, & Minderaa, 2003).

Group CBT (GCBT) has been implemented and compared to ICBT. Barrett (1998) examined the efficacy of a group CBT (GCBT) program for youth with SAD, overanxious disorder, and SoP. Three conditions were compared: GCBT, GCBT plus family management, and a waitlist control. At posttreatment, 64.8% of treated children no longer met criteria for an anxiety disorder, compared with 25.2% of waitlist children, and improvements were maintained to 12-month follow-up. Differences between the two treatment groups were nonsignificant at posttreatment and 12-month follow-up (see also Flannery-Schroeder, Choudhury, & Kendall, 2005). Similarly, Silverman et al. (1999) found 64% of participants in GCBT no longer met criteria for their primary anxiety diagnosis at posttreatment compared to only 13% of the waitlist control, and these gains were maintained to 3-month, 6-month, and 12-month follow-up. Several research groups have conducted comparisons of GCBT to ICBT, often demonstrating equivalent efficacy (Flannery-Schroeder & Kendall, 2000; Manassis et al., 2002), with maintenance of gains to 1-year follow-up (Flannery-Schroeder et al., 2005); see Table 4.1.

Panic Disorder

Cognitive-behavioral therapy has shown efficacy in the treatment of adults with panic disorder (PD), both with and without agoraphobia (Gould, Otto, & Pollack, 1995). Deacon and Abramowitz (2004) determined that these treatments typically consist of (1) psychoeducation regarding the nature of anxiety and panic, (2) cognitive strategies to combat tendencies to misinterpret bodily sensations as catastrophic, (3) exposure to feared bodily sensations, and (4) coping skills to manage physical symptoms. Cognitive-behavioral therapy for PD has been shown to be efficacious in reducing adults' panic symptoms acutely (e.g., Barlow, Gorman, Shear, & Woods, 2000; van Balkon et al., 1997) and at long-term follow-up (Bakker, van Balkon, Spinhoven, Blaauw, & van Dyck, 1998; Craske, Brown, & Barlow, 1991).

One form of CBT for PD is *Panic Control Treatment* (PCT; Barlow, 1988; Barlow et al., 2000). Panic Control Treatment is an 11-session treatment that includes correcting misinformation about panic, breathing retraining, cognitive restructuring, and interoceptive and in vivo exposure. Panic Control Treatment has shown efficacy as an individual (Aaronson et al., 2008) and group CBT (Heldt et al., 2006; Penava, Otto, Maki, & Pollack, 1998). Further, Ollendick (1995) and Hoffman and Mattis (2000) have adapted PCT for adolescents. Hoffman and Mattis (2000) piloted PCT-A with two adolescents, both of whom showed significant improvements after treatment. Pincus, May, Whitton, Mattis, and Barlow (2010) reported a randomized trial of PCT-A: in comparison to a self-monitoring control group, the PCT-A group experienced significant reductions in severity of panic disorder, self-reported anxiety, anxiety sensitivity, and depression ratings. Gains were maintained to 6-month follow-up. See Table 4.2.

Obsessive-Compulsive Disorder

For obsessive-compulsive disorder (OCD) in adults and youth, CBT with exposure and response prevention (ERP) has been established

Table 4.1 Sample Studies of Cognitive Behavior Therapy for Specific Phobia, Social Phobia, Separation Anxiety Disorder, and Generalized Anxiety Disorder

Authors	Sample Characteristics	Findings
Rodebaugh, Holaway, Heimberg (2004)	Review of the available treatments for social phobia	Treatment typically incorporates cognitive restructuring, applied relaxation, exposure to feared stimuli, and social skills training
Rothbaum, Hodges, Smith, Lee & Price (2000)	Adults ($N = 49$; $M_{age} = 40.5$) with SP (flying) were assigned to virtual reality exposure, standard exposure, and WLC	Virtual reality exposure and standard exposure therapy both showed positive treatment gains and were superior to WLC
Ost, Alm, Brandberg, & Breitholtz (2002)	Adults ($N = 46$; $M_{age} = 41.3$) with SP (claustrophobia) were randomly assigned to 1-session exposure, 5-sessions of exposure, 5-sessions of cognitive therapy, or WLC	Treatment was superior to WLC, with no significant differences between the 3 treatment groups
Borkovec, Newman, Pincus, & Lytle (2002)	Adults ($N = 69$; $M_{age} = 37.1$) with GAD were assigned to either applied relaxation and self-control desensitization, cognitive therapy, or a combination of the 2	The majority of participants had significant improvements in anxiety and depression, with no differences between the groups. Remaining interpersonal difficulties at posttreatment were negatively associated with treatment improvement
Newman, Castonguay, Borkovec, Fisher, & Nordberg (2008)	Adults ($N = 15$; $M_{age} = 37.9$) with GAD were assigned to CBT + interpersonal processing therapy and 3 were assigned to CBT + supportive listening	CBT + interpersonal processing therapy led to decreased GAD symptoms maintained to one year follow-up. Effect sizes were higher than previous studies of CBT for GAD
Kendall (1994) Kendall & Southam-Gerow (1996)	Children ($N = 47$; age 9–13) with OAD, SAD, or AVD were assigned to ICBT or WLC	Children in ICBT experienced significant improvements in anxiety symptoms, and gains were maintained at 1-year and 2 to 5-year follow-ups
Kendall et al. (1996) Kendall, Safford, Flannery-Schroeder, & Webb (2004)	Children ($N = 94$; age 9–13) with OAD, SAD, or AVD were assigned to ICBT or WLC	The majority of children in ICBT showed clinically significant gains compared to WLC. Gains were maintained to 7.4-year follow-up
Kendall, Hudson et al., (2008)	Children ($N = 161$; $M_{age} = 10.27$) with GAD, SAD, or SoP were assigned to either ICBT, FCBT, or a family-based education/support/attention control	Treatment gains were evident in all conditions, with ICBT and FCBT superior to the control condition. Gains were maintained to 1-year follow-up
Walkup et al. (2008)	Children ($N = 488$; $M_{age} = 10.7$) with GAD, SAD, or SoP were assigned to either CBT, sertraline, CBT + sertraline, or placebo drug	CBT + sertraline was superior to both CBT and sertraline. CBT and sertraline were equivalent and all therapies were superior to placebo
Barrett (1998)	Children ($N = 60$; age 7–14) with SAD, OAD, or SoP were assigned to GCBT, GCBT + family management, and WLC	More children in treatment conditions were diagnosis-free at post-treatment and 1-year follow-up than WLC, with marginal benefits of GCBT + family management above GCBT
Flannery-Schroeder & Kendall (2000) Flannery-Schroeder, Choudhury, & Kendall (2005)	Children ($N = 24$; age 8–14) with GAD, SAD, or SoP were randomly assigned to ICBT, GCBT, or WLC	Children in the ICBT and GCBT conditions experienced significant reductions in anxiety while the WLC did not. Gains were maintained at 1-year follow-up
Manassis et al. (2002)	Children ($N = 78$; age 8–12) with primary diagnoses of SAD, GAD, SoP, SP, and panic disorder were randomly assigned to either GCBT or ICBT	Anxiety significantly decreased regardless of treatment group. Children with SoP had higher gains in ICBT than GCBT

Note: CBT = cognitive-behavior therapy; *SP* = specific phobia; *SoP* = social phobia; *GAD* = generalized anxiety disorder; M_{age} = mean age; *WLC* = waitlist control; *ICBT* = individual cognitive-behavior therapy; *GCBT* = group cognitive-behavior therapy; *FCBT* = family cognitive-behavior therapy; *OAD* = overanxious disorder; *SAD* = separation anxiety disorder; *AVD* = avoidance disorder

Table 4.2 Sample Studies of Cognitive Behavior Therapy for Panic Disorder

Authors	Sample Characteristics	Findings
Van Balkon et al. (1997)	Meta-analysis of adult studies comparing the impact of pharmacotherapies, CBT, and combination treatments for PD	Pharmacotherapy, exposure in vivo, pharmacotherapy + exposure, and psychological pain management combined with exposure were all effective treatments
Bakker, van Balkon, Spinhoven, Blaauw, & van Dyck (1998)	Meta-analysis of adult studies comparing long-term efficacy of different treatments for PD	All treatments (psychopharmacological treatments, psychological panic management, exposure in vivo, antidepressants combined with exposure, and psychological pain management with exposure) showed gains maintained to follow-up
Barlow, Gorman, Shear, & Woods (2000)	Adults ($N = 314$; $M_{age} = 36.1$) with PD with or without mild agoraphobia were randomly assigned to either CBT, imipramine + medication management, pill placebo + medication management, and CBT + placebo	After 6 months of maintenance, imipramine and CBT were superior to placebo, with imipramine showing a better quality of response and CBT showing more durability. Combined treatment had limited benefit over monotherapy
Aaronson et al. (2008)	Adults ($N = 381$; $M_{age} = 38.8$) with PD who participated in CBT	CBT was effective for both severe PD and less severe PD
Heldt et al. (2006)	Adults ($N = 36$; $M_{age} = 34$) with PD refractory to pharmacological treatment who participated in GCBT	Significant improvement in all areas of quality of life was observed. Reductions in general and anticipatory anxiety and agoraphobia avoidance were associated with quality of life improvements
Penava, Otto, Maki, & Pollack (1998)	Adults ($N = 37$; $M_{age} = 35.8$) with PD participating in CBT	Subjects achieved treatment gains on all PD dimensions, with the largest symptom reduction occurring in the first third of the program
Ollendick (1995)	Adolescents ($N = 4$; age 13–17) with PD treated with CBT	Panic attacks and agoraphobia avoidance were reduced. Self-efficacy for coping with future attacks was increased
Hoffman & Mattis (2000)	Adolescents ($N = 2$; age 13) were treated with Panic Control Treatment adapted for adolescents in a case study format	Each adolescent experienced reductions in panic attack frequency, fear and avoidance, and self-reported anxiety
Pincus, May, Whitton, Mattis, & Barlow (2010)	Adolescents ($N = 26$; age 14–17) were randomized to Panic Control Treatment for adolescents and or self-monitoring control group	Participants showed improvement in PD, self-reported anxiety, and depression in comparison to a control group. Gains were maintained to 6-month follow-up

Note: CBT = cognitive-behavior therapy; *PD* = panic disorder; *GCBT* = group cognitive-behavior therapy

as an efficacious treatment (e.g., Franklin, Abramowitz, Kozak, Levitt, & Foa, 2000; see review by Abramowitz, Taylor, & McKay, 2005). It is important to note that response rates may vary depending on symptom profile and some subtypes, such as cleaning and checking compulsions, have received more research attention than others, such as multiple-ritual, exactness, and hoarding presentations (Ball, Baer, & Otto, 1996; Sookman, Abramowitz, Calamari, Wilhelm, & McKay, 2005). A goal of exposure-based CBT is to teach the individual that, with repeated exposure to the feared object or behavior, the obsession-triggered anxiety will dissipate. As the individual reaches habituation, she/he learns that the feared consequences of not ritualizing will not materialize (Barrett, Farrell, Pina, Peris, & Piacentini, 2008).

Exposure and response prevention is an empirically supported treatment for OCD in youth (Barrett et al., 2008; Storch et al., 2007). The Pediatric OCD Treatment Study (POTS) was a multisite trial for children aged 7–17. The 112 participants were randomly assigned to receive CBT, sertraline (SER), combined CBT and sertraline (COMB), or pill placebo (PBO) for 12 weeks. All three active treatments significantly outperformed PBO, and COMB was superior to CBT and SER. Further, a significantly greater number of CBT patients entered remission than SER patients.

Combined treatment showed a 53.6% remission rate, compared to 39.3% for CBT, 21.4% for SER, and 3.6% for PBO. However, the investigators noted site differences for CBT and SER (POTS Team, 2004). Predictors of attenuated response included higher OCD severity, higher levels of OCD-related functional impairment, higher levels of comorbid externalizing symptoms, and higher levels of family accommodation. Family history of OCD moderated the effect of treatment condition: for participants with a positive family history of OCD, there were no significant differences in outcomes across the treatment groups. Additionally, treatment effect sizes were smaller for those with a family history of OCD, and this reduction in effect sizes was particularly high for the CBT group (Garcia et al., 2010). The presence of a comorbid tic disorder moderated outcomes: among patients with a tic disorder, SER did not differ from PBO, whereas COMB remained superior to CBT, and CBT remained superior to PBO (March et al., 2007) see Table 4.3.

Post-traumatic Stress Disorder

Cognitive-behavioral programs for post-traumatic stress disorder (PTSD) typically involve exposures, cognitive restructuring, and anxiety-management skills. Exposures consist of confrontation with fearful memories of the trauma and can be imaginal or in vivo (Cahill, Foa, Hembree, Marshall, & Nacash, 2006; Foa et al., 1999). Exposures are theorized to be effective by (1) reducing conditioned fear responses associated with trauma cues and (2) challenging cognitive distortions surrounding perceived danger and threat (Foa, Steketee, & Rothbaum, 1989). In their review, Ponniah and Hollon (2009) describe trauma-focused CBT (TF-CBT) as efficacious for PTSD.

For youth with PTSD, exposure-based CBT has established support (Ford & Cloitre, 2009; La Greca, 2008). TF-CBT components are summarized by the acronym "PRACTICE," including psychoeducation/parenting component, relaxation, affect modulation, cognitive processing, trauma narrative, in vivo exposure and mastery of trauma reminders, conjoint child-parent session, and enhancing safety and future development (Cohen, Mannarino, Perel, & Staron, 2007; see also TF-CBT Web, 2005). Trauma-focused CBT has demonstrated efficacy in RCTs (e.g., Cohen, Deblinger, Mannarino, & Steer, 2004; Cohen, Mannarino, & Knudsen, 2005). One study compared TF-CBT to child-centered therapy among 229 children (aged 8–14) who had been sexually abused. Trauma-focused CBT was superior on almost all measures, although 21% of children treated with TF-CBT still met diagnostic criteria for PTSD (Cohen et al., 2004). A pilot study randomly assigned 24 female youth (aged 10–17) with PTSD symptoms to either TF-CBT + placebo or TF-CBT + sertraline. Both groups showed significant improvements on PTSD symptoms, depression, anxiety, and behavior problems. There were no significant differences between the groups. The authors noted significant limitations including inadequate statistical power due to a small sample size. Further, the investigators noted difficulty recruiting a representative sample willing to take sertraline (Cohen et al., 2007); see Table 4.4.

Implementation in Clinical Practice

Several treatments for anxiety disorders across the lifespan have been established as efficacious. At present, the focus is on transporting efficacious treatments into use in everyday practice (Kendall & Beidas, 2007; Weersing & Weisz, 2002). Despite this recognition and related efforts, CBT remains underutilized in the community (Becker, Zayfert, & Anderson, 2004; Gunter & Whittal, 2010; Shafran et al., 2009). Several potential complications in implementing CBT for anxiety will be discussed, including comorbidities, cultural/family factors, and therapeutic process variables.

Comorbidity

High rates of comorbidity have been well documented in children (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Hammerness et al., 2008; Kendall et al., 2010) and adults (van Balkon

Table 4.3 Sample Studies Cognitive Behavior Therapy (CBT) for Obsessive Compulsive Disorder (OCD)

Authors	Sample Characteristics	Findings
Franklin, Abramowitz, Kozak, Levitt, & Foa (2000)	Examined adult treatment outcome data from comparing patients receiving ERP on an outpatient basis to those receiving ERP during clinical trials	Patients receiving ERP on an outpatient basis achieved similar reductions in OCD symptoms to patients participating in randomized clinical trials
Ball, Baer, & Otto (1996)	Meta-analysis of the prevalence of various OCD subtypes in adult samples	Patients with cleaning and checking compulsions made up 75% of the samples, while patients with multiple compulsions, or compulsions not within those categories only made up 12% of the treatment literature reviewed
Barrett et al., (2008)	Meta-analysis of child treatment studies for OCD	Exposure-based CBT for child and adolescent OCD is a probably efficacious treatment. CBT (family-focused and group formats) is a possibly efficacious treatment
Storch et al. (2007)	Children ($N = 40$; age 7–17) with OCD randomized to either 14-weekly or intensive (daily) CBT sessions	Both intensive and weekly CBT were efficacious treatments, with intensive treatment showing some immediate advantages
Storch et al. (2008)	Children ($N = 92$; age 7–19) with OCD who received either weekly or intensive family-based CBT	Overall, treatment response did not appear to differ across OCD subtypes. Some differences were observed (e.g. hoarding symptoms and sexual/religious symptoms) showed less favorable response to treatment. However, lower power limited these analyses
POTS Team (2004)	Children ($N = 112$; age 7–17) with OCD were randomly assigned to either CBT, sertraline, and combined treatment	Combined treatment was superior to CBT alone and sertraline alone. All 3 were superior to placebo. Site differences emerged which limit findings
Garcia et al. (2010) March et al. (2007)	Examined moderators and predictors among participants in the POTS trial	Lower OCD severity, less functional impairment, greater insight, fewer comorbid externalizing symptoms, and lower levels of family accommodation predicted improved outcome. Family history of OCD and comorbid tic disorders moderated treatment outcome

Note: CBT = cognitive-behavior therapy; *OCD* = obsessive compulsive disorder; *ERP* = exposure and response prevention; *POTS* = pediatric obsessive compulsive treatment study

Table 4.4 Sample Studies Cognitive Behavior Therapy (CBT) for Post-Traumatic Stress Disorder (PTSD)

Authors	Sample Characteristics	Findings
Ponniah & Hollon (2009)	Reviews randomized controlled trials in the PTSD and ASD literature	
Cohen, Deblinger, Mannarino, & Steer (2004) Deblinger, Mannarino, Cohen, & Steer (2006)	Children ($N = 229$; age 8–14) with PTSD symptoms (89% met diagnostic criteria) were randomly assigned to either TF-CBT or child-centered therapy	Participants assigned to TF-CBT demonstrated significant greater improvements than child-centered therapy on a variety of measures. Gains were maintained to 6 and 12-month follow-ups
Cohen, Mannarino, & Knudsen (2005)	Children ($N = 82$; age 8–12) who had been sexually abused were randomly assigned to TF-CBT or non-directive supportive therapy	TF-CBT showed significant greater improvements at 6-month and 12-month follow-ups
Cohen, Mannarino, Perel, & Staron (2007)	Children ($N = 24$; age 8–15) with PTSD were randomly assigned to either TF-CBT + sertraline or TF-CBT + placebo	Both groups showed improvements, with minimal evidence indicating sertraline had added benefits. The investigators noted some difficulty recruiting a sample willing to take sertraline

Note: CBT = cognitive-behavior therapy; *PTSD* = post-traumatic stress disorder; *ASD* = acute stress disorder; *TF-CBT* = trauma-focused cognitive-behavior therapy

et al., 2008) with anxiety disorders. Despite its frequency, several questions regarding the impact of comorbidity remain. Is the presence of comorbidity a factor that influences treatment effects? Is comorbidity a moderator that impacts the strength and/or direction of treatment effects? Might comorbidity inform "for whom" and "under what conditions" treatments work (Kendall & Comer, 2011; Kraemer, Wilson, Fairburn, & Agras, 2002)? Comorbidity may operate as a patient characteristic that exists prior to intervention and that may help inform optimal treatment (Kraemer et al., 2002). It has been suggested that treatment outcome might be less successful when treating individuals with comorbid disorders. The following will describe current research regarding comorbidity among individuals with anxiety disorders, focusing on the co-occurring disorders that have been studied: depression and externalizing disorders. Intellectual disabilities will also be briefly discussed.

Comorbidity in Adults

Personality psychopathology has been shown to negatively impact CBT treatment for adults with anxiety disorders (Mennin & Heimberg, 2000), but the impact of other comorbidities is less clear. Studies comparing patients with a range of comorbidities have shown that axis I comorbidity is associated with greater symptom severity, but did not result in differential rates of treatment improvement for the anxiety disorders (Turner, Beidel, Wolff, Spaulding, & Jacob, 1996; van Velzen, Emmelkamp, & Scholing, 1997). A meta-analysis on the topic concluded that comorbidity was generally unrelated to effect size at posttreatment and at follow-up (Olatunji, Cisler, & Tolin, 2010). Similarly, Storch et al. (2010) found that although OCD severity at baseline was higher among OCD patients with comorbidities (specifically, GAD, major depressive disorder, SoP, and PD), pretreatment comorbidities had no impact on posttreatment symptom severity, treatment response, or treatment remission. In a naturalistic sample of adult anxiety patients (principal diagnoses consisted of mostly anxiety disorders but also some mood disorders), comorbidity did not predict therapy dropout or poor treatment response (Davis, Barlow, & Smith, 2010).

Comorbid Depression. The co-occurrence of anxiety and depression has been frequently reported (Costello et al., 2003). Both disorders have affective, cognitive, behavioral, and physiological components. Although anxiety and depression share overlapping emotional features, they differ in identified key components (i.e., fear as a key component for anxiety and hopelessness for depression). Nevertheless, both depressed and anxious individuals exhibit negative affectivity as a broad category of self-reported emotional distress (Watson & Tellegen, 1985). Some differentiation between anxiety and depression is linked to the consideration of positive affectivity (positive emotional states such as joy, enthusiasm, and energy). High negative affectivity and low (not moderate or high) positive affectivity is more linked to depression than anxiety (Watson, Clark & Carey, 1988), while physiological hyperarousal (PH) is common to anxiety (Clark & Watson, 1991; see also Brown, Chorpita, & Barlow, 1998).

In terms of treatment response, comorbid mood disorders have been associated with greater pre- and posttreatment symptom severity among adults with anxiety, compared to comorbid additional anxiety disorders (Erwin, Heimberg, Juster, & Mindlin, 2002). It is important to note that Erwin et al. (2002) did not find differential treatment rates for patients with comorbid depression compared to those with comorbid anxiety in the CBT treatment of socially phobic adults. Additionally, patients with comorbid anxiety and depressive disorders may be more likely to exhibit higher severity of their principal anxiety disorder than patients without comorbidity (Davis et al., 2010), but it is not conclusive that comorbidity has a detrimental impact on outcome (see also Chap. 15 this volume).

Comorbidity in Youth

Some research suggests that comorbid diagnoses can complicate treatment for anxious youth (Berman, Weems, Silverman, & Kurtines, 2000;Storch, Larson et al., 2008; Storch, Merlo

et al., 2008) and are associated with more severe symptomatology (Kovacs & Devlin, 1998). However, other studies have found that comorbidity is not associated with differential treatment outcome (Barrett, Duffy, Dadds, & Rapee, 2001; Kendall, Brady, & Verduin, 2001). As noted by Ollendick, Jarrett, Grills-Taquechel, Hovey, and Wolff (2008), most RCTs for anxiety disorders in youth have not found significant differences on posttreatment outcomes to be linked to comorbidity. Some replicated findings also demonstrated that the number and type of comorbid diagnoses do not significantly predict anxiety treatment outcomes (e.g., Beidel, Turner, & Morris, 2000; Manassis et al., 2002; Ost, Svensson, Hellstrom, & Lindwall, 2001; Smith et al., 2007). A noteworthy complication for comorbidity and treatment outcome research is that anxious youth with comorbid diagnoses may be more likely to attend fewer therapy sessions (Rapee, 2003).

Comorbid Depression. As with adults, the frequent comorbidity of depression among youth with anxiety disorders is well documented (see Seligman & Ollendick, 1998). Some research with youth suggests that the magnitude of this comorbidity may vary depending if anxiety or depression is the principal disorder (e.g., Brady & Kendall, 1992). In one report, exposure-based treatment had poorer outcomes in anxiety-disordered youth with comorbid depression, compared to anxious youth without comorbid depression (Berman et al., 2000). Despite such findings, this study found no group differences between responders and nonresponders in terms of total number of diagnoses, comorbidity with externalizing disorders, and comorbidity with other anxiety disorders (Berman et al., 2000). O'Neil and Kendall (2012) reported that although a comorbid depressive diagnosis did not predict poorer outcomes, self-reported co-occurring depressive symptoms were associated with poorer outcome for youth receiving anxiety treatment. Comorbid depressive symptoms seem to play a role and may warrant special consideration in the treatment of anxiety-disordered youth.

Comorbid Externalizing Disorders. Externalizing disorders (e.g., ADHD, oppositional defiant disorder, conduct disorder) may require adjustments when implementing an empirically supported treatment for anxiety, but the data can inform us of the accuracy of this concern. Although ADHD is more often comorbid with externalizing rather than internalizing disorders, there are also sizeable comorbid rates of ADHD with anxiety and depression (~25%) in epidemiological studies (Angold, Costello, & Erkanli, 1999). To date comorbid externalizing disorders, secondary to the principal anxiety disorder, do not moderate treatment outcomes, as evidenced by limited effect on outcome of CBT treatment in youth (Flannery-Schroeder, Suveg, Safford, Kendall, & Webb, 2004). It seems that anxious youth with comorbid externalizing problems sometimes respond better to treatment than their non-comorbid peers (Costin & Chambers, 2007; Kazdin & Whitley, 2006). Regardless, savvy implementation of treatments may be necessary, even during manual-based treatments, to address problematic features of comorbidity that impede progress (see Hudson, Krain, & Kendall, 2001).

Treatment That Targets Multiple Disorders

Should treatment be focused entirely on the primary anxiety disorder or should treatments also be tailored to the comorbid issues? This topic has been raised and debated, leading some to develop treatments that cut across diagnostic categories (Wilamowska et al., 2010). Cognitive-behavioral therapy focused on the primary disorder, as compared to CBT focused on the primary disorder and the most severe comorbid condition, has been shown to be more beneficial for both principal and comorbid disorders in PD patients (Craske et al., 2007). Such findings suggest that for anxiety, like panic, the greatest benefit to clients is to pour therapeutic energy into the most debilitating domain of psychopathology (i.e., the principal diagnosed disorder). Successful treatment of PD has also been associated with reductions of both comorbid anxiety and depressive symptoms (Allen et al., 2010). The development of integrated or "transdiagnostic" treatments is ongoing (for adults, e.g., Barlow, Allen, & Choate, 2004;

for youth, e.g., Chu, Colognori, Weissman, & Bannon, 2009). Despite preliminary findings for these interventions, additional research is needed to examine the efficacy of these treatments in controlled and randomized trials.

Intellectual Functioning and Implementation of Treatment

Appropriately, RCTs for the treatment of anxiety-related disorders exclude individuals with psychotic disorders, intellectual deficits, or pervasive developmental disorders. Nevertheless, such exclusions do prevent conclusions about treating such individuals, particularly given that intellectual functioning can be a factor when implementing CBT. In older adults this may be important, especially if there are age-related deficits. Doubleday, King, and Papageorgiou (2002) found no significant association between level of fluid intelligence and benefit from CBT in the treatment of anxiety, though higher fluid intelligence was associated with positive impact for patients receiving supportive counseling. Conversely, poor performance on the Mini-Mental State Exam orientation domain in older adults with GAD has been associated with poorer outcome 6 months after CBT (Caudle et al., 2007). Low intellectual functioning (and problems with receptive and expressive communication) may be associated with patients that have trouble establishing a collaborative interaction with their therapist (Jahoda et al., 2009). Although factors related to intelligence are important to consider in the context of treatment, mild or age-related intellectual decline may not have substantial negative impact on therapeutic alliance and outcome in CBT for anxiety.

Intellectual Functioning in Youth. When working with children with limited cognitive functioning, therapists may need to modify manual-based CBT protocols to ensure that treatment strategies are compatible with the child's developmental capacities. Individualized treatment may employ an increased focus on physical involvement (e.g., active games that illustrate session content) and a de-emphasis on meta-cognitive content that is generally appropriate for youth of average intelligence (Suveg, Comer, Furr, & Kendall, 2006). Learning difficulties may need to be taken into account, as it is not uncommon for youth with learning problems to experience elevated anxiety and self-consciousness in the classroom (Dekker, Koot, van der Ende, & Verhulst, 2002). For a child with limited intellectual functioning, treatment can be more parent oriented. When such factors are taken into consideration, CBT can result in positive outcomes among children with intellectual deficits (Suveg et al., 2006).

Family and Cultural Factors in CBT for Anxiety Disorders

Family and cultural factors can play a role in the implementation of ESTs for anxiety disorders. The following section considers how the family context and racial/ethnic or cultural background may influence treatment for both adults and children with anxiety disorders.

Family Factors

The family context, potentially important in CBT for anxiety disorders, is not limited to youth. For adult clients, the family context may include spouses or romantic partners, as well as children, and their own parents. With regard to spousal relationships, there is mixed evidence as to whether the quality of the spousal relationship predicts treatment outcome for adults with anxiety disorders (e.g., Durham, Allan, & Hackett, 1997; Marcaurelle, Belanger, & Marchand, 2003). Nevertheless, some data indicate that spouse involvement in treatment can be beneficial (e.g., Barlow, O'Brien, & Last, 1984; Billette, Guay, & Marchand, 2008).

For youth clients, the family context typically includes parent(s) and may include siblings. Bottom-up research, studying the parents of children with anxiety disorders, indicates that such parents have elevated psychopathology (e.g., Hughes, Furr, Sood, Barmish, & Kendall, 2009)

and less favorable parenting style (McLeod, Wood, & Weisz, 2007). In particular, parental anxiety predicts poorer acute treatment outcome for youth who receive child-focused CBT (Bodden et al., 2008; Cobham, Dadds, & Spence, 1998), although it is not clear if parental anxiety predicts long-term child outcomes (Cobham, Dadds, Spence, & McDermott, 2010). Some studies also suggest that a less warm, more rejecting, and over-involved parenting style may negatively impact treatment outcome for anxious children (Creswell, Willetts, Murray, Singhal, & Cooper, 2008; Liber et al., 2008). It has also been shown that families of anxious youth are characterized by poorer family functioning (e.g., Hughes, Hedtke, & Kendall, 2008) when compared to families of non-disordered youth and that family dysfunction is associated with poorer treatment outcome for anxiety-disordered youth (Crawford & Manassis, 2001).

Given the role that parental psychopathology, parenting style, and family dysfunction may play in treatment outcome, there is ongoing debate as to how parents should be involved in CBT for childhood anxiety disorders. In individual child-focused CBT for anxious youth, parents are typically involved as consultants (e.g., provide information about symptoms and impairment) and collaborators (e.g., bring youth to treatment, assist with exposures; Kendall, 2010). Parents may also be involved as co-clients, to the extent that their own anxiety or behavior may be maintaining the child's anxiety or interfering with treatment (see also Barmish & Kendall, 2005; Kendall, 2010). In some work, parents may serve as co-therapists (see Renshaw, Steketee, & Chambless, 2005).

Research examining the benefit for child outcomes of including parents in treatment has resulted in mixed findings. There is support for family-based CBT for childhood OCD, and some studies suggest better child outcomes for SAD, SoP, and GAD with increased parental involvement in CBT (Barrett et al., 1996; Cobham et al., 2010; Wood, Piacentini, Southam-Gerow, Chu, & Sigman, 2006). Other research findings indicated no added benefit of parental involvement (Bodden et al., 2008; Kendall et al., 2008; Nauta et al., 2003). These mixed findings suggest that there may be client or family characteristics that predict for whom parental involvement is beneficial. Cognitive-behavioral therapy with increased parental involvement may be more beneficial for younger children and females compared to older children and males (Barrett et al., 1996), although in another study the benefit of family CBT was greater for early adolescents than for younger children (Wood, McLeod, Piacentini, & Sigman, 2009). Furthermore, Cobham et al. (1998) reported that an additional parent component (e.g., parental anxiety management) resulted in better outcomes than child CBT only for youth with anxious parents. Similarly, Kendall et al. (2008) found that FCBT outperformed ICBT when both parents had anxiety disorders. Taken together, these findings suggest that increased parental involvement in CBT for child anxiety may be beneficial for youth with anxious parents.

Cultural Factors

The prevalence, symptom expression, treatment-seeking behavior, and treatment outcome of anxiety disorders in adults and youth can be influenced by cultural factors. Racial/ethnic minority adults in the United States (Latinos, African Americans, Caribbean Blacks, Asian Americans) have lower reported rates of PD, SoP, and GAD than non-Latino Whites, whereas racial/ethnic differences in prevalence rates for agoraphobia, SP, and OCD are less clear (Lewis-Fernandez et al., 2010). Racial/ethnic minorities tend to seek mental health care services at lower rates than Caucasians (e.g., Snowden, 1999; Zhang, Snowden, & Sue, 1998) and are more likely to seek help from a primary care physician than a mental health care provider (Snowden & Pingitore, 2002). Factors that may contribute to lower treatment-seeking and higher attrition among minorities may include the presence of stressors (e.g., SES), lack of trust in mental health professionals, lack of familiarity with treatment, and reliance on family, friends, or faith-based sources for mental health needs.

Hunter and Schmidt (2010), for example, described a sociocultural model of anxiety in African American adults in which an awareness of racism, stigma of mental illness, and salience of physical illness influence rates of anxiety disorders. Empirical investigations of the factors that contribute to lower treatment-seeking and higher treatment attrition among racial/ethnic minority groups are needed.

For anxious youth, the available literature suggests some cultural differences in symptom expression. There is evidence that Latino youth tend to report higher rates of somatic symptoms compared to Caucasian youth (Canino, 2004; Pina & Silverman, 2004). Asian American youth tend to exhibit somatic symptoms as early signs of anxiety (Gee, 2004). African American youth tend to score higher than Caucasian youth on measures of anxiety sensitivity (Lambert, Cooley, Campbell, Benoit, & Stansbury, 2004), although African American youth are less likely to be diagnosed with GAD (Kendall et al., 2010). Additionally, similar to adult patterns, race and ethnicity predict lower rates of treatment-seeking behavior and higher attrition rates among youth (Bui & Takeuchi, 1992; Gonzalez, Weersing, Warnick, Scahill, & Woolston, 2011; Kendall & Sugarman, 1997; Sood & Kendall, 2006), although the variation in treatment-seeking patterns for different racial/ethnic groups indicates that generalization of one pattern for all minority groups would be inaccurate.

A majority of the participants in RCTs examining the efficacy of CBT for anxious youth have been Caucasian, limiting the examination of race and ethnicity as potential predictors of treatment outcome. However, available research suggests that CBT is an appropriate treatment option for youth from various racial/ethnic groups. Treadwell, Flannery-Schroeder, and Kendall (1995) reported comparable outcomes for Caucasian and African American youth who received the *Coping Cat* program for their anxiety. Pina, Silverman, Weems, Kurtines, and Goldman (2003) found comparable outcomes for Caucasian and Latino youth who received exposure-based CBT for anxiety. Several researchers have called for greater consideration of culture in research regarding ESTs for childhood psychological disorders (e.g., Jackson, 2002).

Therapeutic Process Variables

Appropriately, the bulk of treatment research for anxiety has focused on evaluating the efficacy and effectiveness of specific therapies and specific strategies, both in children and adults. Indeed, the published reports have contributed greatly to our knowledge of what works for anxiety disorders. Exactly *how* these treatments bring about change has become an area of recent, yet still understudied, focus (Chu & Kendall, 2004; Fjermestad, Haugland, Heiervang, & Ost, 2009). Theory and discussion suggest that process factors, common across many of the effective therapies, contribute to outcomes for both children and adults. Although these processes differ somewhat for children and adults, three process-relevant variables (alliance, client involvement, collaboration) have been viewed as important to treatment outcome across many disorders (Chu & Kendall, 2004; Creed & Kendall, 2005; Fjermestad et al., 2009).

Alliance

Many psychological therapies assign importance to a variously labeled and described "therapeutic alliance." This alliance has become a pantheoretical variable considered to be important for change (Horvath, 2000; Martin, Garske, & Davis, 2000). Definitions of alliance converge around three themes: (1) the "work-together" nature of the relationship, (2) the affective bond between client and therapist, and (3) the patient and therapist's ability to agree on treatment goals and tasks (Karver et al., 2008; Martin et al., 2000).

According to a meta-analysis of the adult literature, therapeutic alliance as a stable and unmediated variable has a moderate relationship ($r = 0.22$) with therapeutic outcome (Martin et al., 2000). Similarly, a study examining CBT outcome for socially anxious adults indicated that greater alliance measured at the final session

was related to lower posttreatment symptomology. Here, alliance is indicated as a secondary but important influence on treatment outcome (van Dyke, 2002). Additionally, socially anxious adult clients, with high self-reported levels of alliance, reported higher levels of therapeutic helpfulness (Hayes, Hope, VanDyke, & Heimberg, 2007). This group evidenced greater changes in self-reported anxiety during exposures when observers rated their alliance at a moderate level.

The relationship between outcome and therapeutic alliance in children is, like that in adults, only moderate (Karver, Handelsman, Fields, & Bickman, 2006; Shirk & Karver, 2003). Alliance has been associated with effective CBT for anxious children (e.g., Kendall, 2001; Southam-Gerow & Kendall, 1996). However, the number of studies to assess and evaluate the therapeutic alliance in children has been few. It is possible that therapeutic alliance among children is more difficult to assess as they do not come to therapy/treatment of their own volition (and might even be resistant to change; DiGiuseppe, Linscott, & Jilton, 1996). Youth, in general, may be particularly resistant or have limited insight, making the forming of an alliance more difficult (Diamond, Liddle, Hogue, & Dakof, 1999). An additional complication to considerations of the therapeutic alliance in child therapy is the presence of another relationship: with the child's parent. This suggests that clinicians must also engage in and develop a positive relationship with the child's primary caregiver (McLeod & Weisz, 2005). Parental beliefs about therapy can influence child attitudes toward treatment (Chu & Kendall, 2004).

Despite the potential complications, a therapeutic alliance with youth can facilitate their engagement in therapeutic activities, and a strong relationship with their clinician can prompt involvement (Chu & Kendall, 2004; Kendall & Ollendick, 2004). In a recent evaluation of alliance in manual-based treatment for anxiety disorders, Liber et al. (2010) found that a stronger observer-rated alliance was associated with greater reliable change in child-reported anxiety symptoms. A stronger observer-rated alliance was also related to better treatment adherence and associated symptom reduction at early and midpoints in treatment (Chiu, McLeod, Har, & Wood, 2009; Liber et al., 2010). This change in alliance preceded change in symptomatology. A recent study by Marker, Comer, Abramova, and Kendall (2013) examining multiple reports of therapeutic alliance on treatment outcome not only found that greater therapist- and mother-rated alliance prospectively predicted improved treatment outcomes, but they also identified a reciprocal relationship between therapist- and father-reported alliance and symptom reduction whereby alliance increases as anxiety decreases among children receiving CBT for anxiety.

Involvement

Client involvement in therapy has been found to significantly contribute to therapeutic outcomes for adults (e.g., Gomes-Swartz, 1978; O'Malley, Suh, & Strupp, 1983; Tryon & Kane, 1995). The relationship between involvement and outcome holds for client-rated involvement (e.g., Holtzworth-Munroe, Jacobson, DeKlyen, & Whisman, 1989; O'Malley et al., 1983), therapist-rated involvement (e.g., Gomes-Swartz, 1978; O'Malley et al., 1983), and involvement assessed via an independent evaluator (e.g., Gomes-Swartz, 1978; Soldz, Budman, & Demby, 1992). The relationship holds within individual therapies, for group therapy (Soldz et al., 1992), in general clinical practice (versus a research trial; Eugster & Wampold, 1996; Goren, 1991), and with couples in marital therapy (Holtzworth-Munroe et al., 1989). Not surprisingly, the degree to which someone is involved in therapy has a favorable association with the magnitude of the therapeutic outcome. Additionally, anxious patients have identified a preference for participating in making treatment decisions, although this effect was moderated by ethnicity, with some minorities showing more passive preferences for involvement in decision-making (Patel & Bakken, 2010). In other research, involvement has been related to other variables, such as therapeutic alliance and therapy completion (e.g., Reandeau & Wampold, 1991; Tryon & Kane, 1995). Overall,

research supports adult client involvement as a direct predictor of treatment outcome across a variety of therapeutic settings.

For children experiencing distressing anxiety, involvement in therapy may be particularly important given the previously mentioned complications this population brings to therapy and their characteristic avoidance and withdrawal when feeling threatened. These behaviors are likely to impede the progress of therapy, and exploring methods of fostering improvement is an important research question. However, involvement in child psychotherapy has infrequently been studied in rigorous scientific trials or within evaluations of the efficacy of specific treatments. Furthermore, where involvement is examined, the results are often inconsistent (e.g., Chu & Kendall, 2004; Karver et al., 2008).

Despite complications, studies have examined child involvement as a factor in treatment outcome for various problems (e.g., disruptive classroom behavior; Braswell, Kendall, Braith, Carey, & Vye, 1985). In one project, independent observers' ratings of child involvement during the psychoeducation phase of CBT (prior to exposure tasks) were associated with both improved diagnostic status and impairment outcomes (Chu & Kendall, 2004). A recent meta-analysis indicated that child involvement in treatment has a strong association with outcome (mean $r=0.7$; Karver et al., 2006; twice that of alliance), although this association varies across the studies included in the meta-analysis. Particular therapist behaviors have been shown to foster youth involvement, including exploring the child's motivation for therapy or change, attending to the youth's experience, and providing less structure in the initial session (Jungbluth & Shirk, 2009). The association between child involvement in therapy and the quality of the therapeutic alliance is not specific to CBT (see Karver et al., 2008).

Collaboration

Often conceptualized as a necessary component of the therapeutic alliance, collaboration between therapist and client has been identified as an important factor in determining the quality of therapeutic alliance and in facilitating change (Creed & Kendall, 2005; Tee & Kazantzis, 2011). Therapist-client collaboration consists of a sense of teamwork, where the therapist encourages feedback, and specific contributions to therapeutic goals. In collaboration, therapist and client share the therapeutic work, which progressively allows the client more leading control in the inception and testing of ideas and goals, boosting their self-efficacy and motivation for change (Tee & Kazantzis, 2011). Krupnik et al. (1996) found that not only therapeutic alliance but also patient contribution to alliance, which may be considered a piece of this collaborative effort, was significantly predictive of treatment outcome among individuals with depression. Collaboration has often been emphasized as an important piece of the therapeutic alliance (e.g., Bordin, 1979; Horvath, 2000). Additional research is warranted to explore its particular contribution to treatment outcome in the adult anxiety literature.

A collaborative process with children allows the therapist to personalize the specifics of treatment (i.e., exposure tasks) and the case conceptualization (Tee & Kazantzis, 2011). Findings from studies of child treatment outcome indicate that collaboration predicts a stronger child rating of the therapeutic alliance (Creed & Kendall, 2005), higher levels of treatment satisfaction in adolescents (Church, 1994), and more successful treatment outcomes with anxious youth (Chu & Kendall, 2004).

Future Directions

Our review discussed ESTs for anxiety disorders across the lifespan. In an effort to continue to advance these ESTs, we reviewed comorbidity, familial and cultural components, and process variables as factors that may influence the implementation of these ESTs for anxiety disorders. Based on this review, we offer several suggestions for future work.

The convention in anxiety treatment research is to examine improvement based on the diagnosed principal disorder. The presence of high

rates of comorbidity, and related concerns about the current diagnostic categories, suggests overlapping features among anxiety disorders and even some mood disorders (Bahadurian, 2008; Brown & Barlow, 2009; Brown, Campbell, Lehman, Grisham, & Mancill, 2001). Such overlap has implications for classification and treatment of anxiety disorders. For example, several investigators are developing and evaluating transdiagnostic emotion-focused CBT for the treatment of shared emotional disorders. Preliminary evidence is encouraging (e.g., Wilamowska et al., 2010). The future of psychological intervention research may lie within an empirically driven consolidation of therapy strategies for treatment of comorbid psychopathology.

Family and cultural influences suggest directions for future research. Given inconsistent findings regarding the benefits of inclusion of family members in treatment, research needs to examine under what circumstances and for what conditions, there is a favorable impact associated with including family members in anxiety treatment. Investigations of potential moderators of family involvement in CBT are warranted. Regarding cultural factors, the current data suggest that future work addresses the factors that contribute to lower treatment-seeking and higher treatment attrition rates for racial/ethnic minorities.

It is a decided advance that process is now studied within treatments of known outcome (i.e., ESTs). With regard to the contribution of specific process variables, there is merit in studying the interactions and causal patterns among particular components (e.g., alliance, involvement, collaboration). For example, does collaboration influence the child's view of alliance or does the child's perception of a strong alliance promote collaboration? Knowing the direction by which collaboration and alliance interact and influence treatment outcome would inform practice. Researching client characteristics that moderate process variables would help to personalize intervention. Furthermore, evaluating processes at multiple time points and identifying trajectories of change may provide ways to early identify clients at risk for less favorable outcomes.

In addition to the areas discussed, there is concern for translating empirically efficacious treatments into effective therapies for use in everyday practice. Dissemination of manual-based cognitive-behavioral therapy for anxiety disorders remains a critical next step. And, despite substantial evidence documenting the efficacy of cognitive-behavioral therapy for anxiety disorders, reviews of the literature identify that a portion of children maintain their anxiety symptoms after treatment (Cartwright-Hatton, Roberts, Chitsabesan, Fothergill, & Harrington, 2004; Silverman et al., 2008). We need to improve our ability to "personalize" CBT, tailoring the approach to meet each child's specific needs. In this way, we will continue to advance empirically supported treatments.

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Part II

Complexities in Childhood and Adolescent Anxiety Disorders

Treatment of Childhood Anxiety in Autism Spectrum Disorders

5

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Nature of Problem

Prevalence

Considerable research indicates that youth diagnosed with autism spectrum disorders (ASD) experience psychiatric symptoms meeting clinical diagnostic criteria for a range of disorders, including anxiety disorders (Gadow, Devincent, Pomeroy, & Azizian, 2005; Kim, Szatmari, Bryson, Streiner, & Wilson, 2000; Sukhodolsky et al., 2008; White, Oswald, Ollendick, & Scahill, 2009). The presence of anxiety disorders in ASD has been widely documented, but the prevalence rate varies across the literature (e.g., de Bruin, Ferdinand, Meester, de Nijs, & Verheij, 2007; Leyfer et al., 2006). For example, White et al. (2009) demonstrated in a comprehensive review that significantly impairing anxiety *symptoms* were present in 11–85% of youth diagnosed with ASD. More specifically, studies using robust diagnostic criteria indicate that anxiety *disorders* occur in at least 45% of youngsters with ASD (Leyfer et al., 2006; Simonoff et al., 2008; Sukhodolsky et al., 2008). Simonoff et al. (2008) found that up to 71% of the youth met criteria for at least one psychiatric disorder and, of these, 42% met criteria for an anxiety disorder according to the Diagnostic and Statistical Manual of Mental Disorders—4th Edition criteria (DSM-IV-TR; American Psychiatric Association, 2000) based on a population-derived sample of children and adolescents diagnosed with an ASD.

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Symptomology and Diagnostic Issues

Youth with ASD have been reported to present frequently with simple phobias, generalized anxiety disorder, separation anxiety disorder, obsessive-compulsive disorder, and social anxiety. There does not seem to be one anxiety disorder that is specifically associated with ASD. Rather, heterogeneity exists in the rates in which the different types of anxiety disorders have been reported. In one clinical sample of school-aged children diagnosed with ASD, of those who met diagnostic criteria for anxiety disorders, simple phobia was the most widely endorsed (31%), then social phobia (20%), separation anxiety disorder (11%), and generalized anxiety disorder (10%; Sukhodolsky et al., 2008). In another study, social phobia was the most commonly diagnosed (30%) followed by generalized anxiety (13%; Simonoff et al., 2008).

Among youth with ASD, anxiety disorders occur at commensurate or higher frequency and severity levels than that observed in the general community (e.g., Kim et al., 2000; Lecavalier,

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2006; Russell & Sofronoff, 2005; White et al., 2009). Gadow et al. (2005) found that 25% of youth with ASD in their sample met diagnostic criteria for generalized anxiety disorder relative to 20% of a typically developing outpatient comparison group. Social anxiety appears to occur at higher rates in youth with ASD than in the typically developing population, with results from a number of studies indicating 20–57% of children and adolescents with high-functioning ASD exhibit clinically relevant symptoms of social anxiety, as compared to 1–5% in typically developing youth (Kuusikko et al., 2008; Muris, Steerneman, Merckelbach, Holdrinet, & Meesters, 1998). Also, findings are emerging that youth with ASD who demonstrate higher cognitive and functioning levels may be more susceptible to developing anxiety disorders and experience greater symptom severity (Sukhodolsky et al., 2008; Weisbrot, Gadow, DeVincent, & Pomeroy, 2005); however, others have not observed such a trend (Kim et al., 2000; Pearson et al., 2006).

The high occurrence of comorbid anxiety disorders in ASD has raised some concerns about the validity of diagnosis and assessment methods with this population. Wood and Gadow (2010) underscore that diagnostic methods need to be refined to tease apart anxiety and core ASD symptoms in order to improve differential diagnosis and obtain more accurate prevalence rates. For example, they suggest taking into account the emotional valence of symptoms when differentiating between obsessive-compulsive symptoms and ASD-related restricted interests and ritualistic behaviors. Positive affect is more likely to be associated with restricted interests than with disturbing OCD-related obsessions. They also point out that anxiety in ASD can be phenotypically identical to the anxiety disorders in non-ASD groups, but it can also uniquely manifest stemming from the interplay between ASD core symptoms and anxiety (e.g., anxiety stemming from a child prevented from engaging in autistic routines). Accurate diagnosis will inform treatment development and ensure appropriate access to mental health services for children on the autism spectrum.

The extant research presents strong evidence that comorbid anxiety disorders have direct implications for the overall functioning and quality of life of youth with ASD and their families (Matson & Nebel-Schwalm, 2007). Youth with comorbid mood or anxiety were found to engage in increased aggression and oppositional behaviors and experience poorer social relationships compared to youngsters with ASD who did not have such comorbidities (Kim et al., 2000). Other functional impairments such as poor social responsiveness and social skill deficits have been associated (Bellini, 2004; Sukhodolsky et al., 2008). The relationship between the presence of anxiety and overall impairment in affected youth underscores the importance of treatments to relieve such symptoms.

Potential Contributing Factors

The most common hypotheses to explain the high occurrence of psychiatric comorbidity in ASD have been: (a) that there may be a common genetic linkage between ASD and other psychiatric disorders, increasing the risk of each (e.g., Gadow, Roohi, DeVincent, Kirsch, & Hatchwell, 2009), (b) that the stresses caused by having ASD (e.g., social rejection, sensory over-responsiveness, confusion in light of communication challenges) overwhelm coping skills and induce emotional and behavioral disorders (e.g., Meyer, Mundy, Van Hecke, & Durocher, 2006), or (c) that core autism symptoms are sometimes "counted as" aspects of a comorbid disorder that has phenotypically similar features (Gillott, Furniss, & Walter, 2001; Wood & Gadow, 2010).

A cognitive-behavioral model of psychopathology in high-functioning youth with ASD provides a framework for understanding the development and treatment of co-occurring anxiety. The interaction between behavioral, environmental, and cognitive factors may explain the clinical presentation of youth in this population. The combination of their increased intellectual capabilities (capacity for insight) and ASD features likely contribute to the development of maladaptive schemas (e.g., low self-efficacy;

Bandura, Adams, & Beyer, 1977), limited coping strategies, and shape anxiety symptomology.

Similar to typically developing children, categorical psychosocial stressors such as parental discord or peer victimization (Shytayermman, 2007) have been associated with increased emotional stress in youth with ASD. Higher anxiety levels have been linked to the presence of such stressors, impaired behavioral flexibility to minor life changes, and a decreased ability to manage resulting emotions (Evans, Canavera, Kleinpeter, Maccubbin, & Taga, 2005; Green et al., 2006; Tantam, 2000). Biological factors such as an inhibited temperament style in ASD (Bellini, 2006) and a familial predisposition for psychiatric conditions (Ghaziuddin, Ghaziuddin, & Greden, 2002) also seem to contribute to these risk factors. Impairments associated with core ASD features likely limit the repertoire of coping skills to effectively manage emotional disturbance associated with adverse life experiences.

High-functioning youth with ASD have a capacity for awareness of their social-communicative limitations (Meyer et al., 2006). Children with Asperger Syndrome endorsed greater social worries relative to typically developing peers (Russell & Sofronoff, 2005). Severity of generalized anxiety symptoms is more pronounced in Asperger disorder relative to high-functioning autism (Thede & Coolidge, 2007). Contrary to the belief that youth with ASD are satisfied being alone, affected children endorse greater social difficulty, social distress, dissatisfactory interpersonal relationships, and decreased social competency relative to a matched control group of children diagnosed with learning disabilities (Burnette et al., 2005) and typically developing peers (Bauminger & Kasari, 2000). Youth with ASD likely experience great distress and concern with interpersonal relationships.

Adaptive functioning impairments can also contribute to poor social-emotional functioning. Youth with high-functioning ASD demonstrated adaptive functioning levels markedly below their cognitive potential (Klin, Saulnier, et al., 2007) and only 50% of a clinical sample of individuals with ASD independently completed basic self-care needs such as grooming (Green, Gilchrist, Burton, & Cox, 2000). Anxiety symptoms likely compound these deficits given that even among typically developing children diagnosed with separation anxiety disorder, decreased adaptive living skills was positively correlated with anxiety severity (Wood, 2006). Youngsters with ASD seem to lack the social self-sufficiency or "real-life skills" to lead independent lives, highlighting the importance of incorporating these skills into intervention.

The meditational role of cognition may influence the development of maladaptive beliefs in ASD. Difficulties with perspective taking, drawing inferences from contextual information, and executive functioning can contribute to social-emotional issues in ASD; however, further research exploring this area is necessary (Meyer et al., 2006; Thede & Coolidge, 2007). Similar to typically developing children with social-emotional disturbances (Dodge, 1993), youth with ASD displaying greater atypical social attribution processes and a hostile attribution bias (tendency to attribute hostile intent in others) were more likely to endorse anxiety and depression symptoms (Meyer et al., 2006).

Treatment Approaches

Cognitive-behavioral therapy (CBT) is an effective form of treatment for typically developing youth with childhood anxiety disorders (Barrett, Duffy, Dadds, & Rapee, 2001; Gosch, Flannery-Schroeder, Mauro, & Compton, 2006; Kendall, 1994; Walkup et al., 2008; Wood, 2006), particularly those incorporating family-based approaches (e.g., Wood, Piacentini, Southam-Gerow, Chu, & Sigman, 2006). According to this treatment approach, the theory of change primarily focuses on cognitive and behavioral mechanisms for symptom improvement. The main components of CBT for children include psychoeducation; developing coping skills (e.g., awareness of anxiety feelings, cognitive restructuring); and applying skills in graduated in vivo exposures (e.g., Kendall, 1994). An integral aspect to CBT is that children collaborate with clinical guidance to actively engage in empirical and logical question-asking

and evaluation of anxiety-related situations through sequential and graduated experiences. Exposures are a core element as it provides mechanisms for hierarchical counterconditioning, extinction and habituation, thereby altering children's expectations of themselves and others (Gosch et al., 2006). Kendall et al. (1997) showed that in typically developing youth *cognitive* intervention aspects of the treatment (e.g., challenging irrational beliefs) alone—when not paired with in vivo exposure elements—was not effective in reducing children's anxiety levels.

Contemporary CBT methods promote the development of schemas that guide adaptive responses while suppressing maladaptive ones. Generally, schemas are underlying mental frameworks or memory representations that broadly encompass an individual's network of attitudes, emotional associations, and episodic memories linked with a concept or situation. This notion of a schema differs from those that may be more specific to the patterns of thinking styles and cognitive models associated with particular psychological disorders such as depression (e.g., Beck, 1987). One model of memory retrieval competition in CBT (Brewin, 2006) suggests that adaptive schemas may need to be encoded with positive information and rehearsed in relevant situations in order to successfully be retrieved over coexisting maladaptive ones. The development of salient adaptive schema can be enhanced by elaborated rehearsal of such adaptive responses through deep semantic processing using active discussion, practice of skills within settings to encode schema relevant to actual situations, and incorporating emotionally positive elements such as humor.

Research on the efficacy of CBT in reducing comorbid anxiety in youth with high-functioning ASD and Asperger's disorder has been promising and spans from case studies (Lehmkuhl, Storch, Bodfish, & Geffken, 2008; Reaven & Hepburn, 2003; Sze & Wood, 2008) to group-design clinical studies (e.g., Reaven et al., 2009; Sofronoff, Attwood, & Hinton, 2005; Wood et al., 2009). The established efficacy of CBT for the treatment of childhood anxiety disorders in typically developing youth has served as a foundation for the development of this intervention for children and adolescents with ASD. Due to the complex clinical presentation of ASD and unique cognitive and emotional profile of this group of youth, modifications to the implementation of treatment have been evident across studies. The general consensus in modifying traditional CBT highlights the importance of tailoring CBT to meet the clinical needs of children with ASD to maximize the uptake and active use of coping skills. Some modifications to standard CBT methods for comorbid anxiety include the incorporation of visual aids (e.g., cartoons and thought bubbles) to supplement discussion of clinical material with the child, increased instruction on emotion recognition (self-awareness of anxiety symptoms), and clear and concrete presentation of ideas and materials (breaking down abstract ideas, direct and explicit directions). Reaven et al. (2009) modified CBT to treat anxiety in children with ASD in an unrandomized, open enrollment of a 12-week group intervention that included individual child, individual parent, and conjoint parent–child components. The authors drew upon several existing CBT manuals to develop an original program to accommodate ASD. They incorporated visual and concrete approaches to teach coping skills, emphasized drawing, photography, and video modeling to enhance generalization of skills and concepts. The results from their study demonstrated an improvement in anxiety symptoms in the active treatment group in comparison to the waitlist group. Trials of CBT conducted with typically developing children and youth with anxiety disorders (e.g., Barrett, Dadds, & Rapee, 1996) indicate that including parent training in the intervention can lead to superior intervention effects as compared to exclusively child-focused treatments. Sofronoff et al. (2005) found evidence that a CBT program for children with ASD and anxiety that included a combined child and parent treatment was more effective than working with children alone. Also, CBT programs for individuals with ASD and high anxiety in the current literature vary widely with regard to the emphasis placed on in vivo exposure relative to less active treatment elements (e.g., role-playing). Only a few intervention studies

included in vivo exposures on a daily basis (e.g., Wood et al., 2009).

Some researchers have remarked that modifications to CBT, alone, may not fully address the expression of anxiety in ASD (Reaven et al., 2009; White et al., 2009; Wood et al., 2009). CBT interventions for anxiety in ASD are based on treatment that was initially developed for typically developing children, potentially limiting the efficacy of treatment. Some have questioned whether CBT for anxiety in children with ASD should be tailored specifically for this group (e.g., White et al., 2009). Current evidence suggests that despite this concern, CBT interventions have produced positive treatment gains in remediating anxiety in ASD. The effectiveness of CBT for children with ASD seems comparable to that observed in typically developing children with anxiety (e.g., Chalfant, Rapee, & Carroll, 2007) suggesting that some manifestations of anxiety in ASD may be similar to that in typically developing youth given the positive response to treatment. For example, up to 84% of children with ASD and co-occurring anxiety who received CBT with adjunctive family intervention (Chalfant et al., 2007; Wood et al., 2009) no longer met criteria for a primary anxiety disorder which was consistent with that observed in randomized clinical trials (RCT) of CBT for typically developing children with anxiety (e.g., Silverman et al., 1999; Storch et al., 2010). The basic elements of CBT are likely foundational components in treating anxiety disorders, applicable across populations.

The traditional model of CBT has been widened to develop interventions that are uniquely tailored to youth with ASD. *Enhancement* of CBT by expanding both treatment conceptualization and methods to go beyond the immediate implementational concerns (i.e., making the treatment materials and skills accessible) has given way to develop CBT that specifically targets ASD characteristics that could contribute to the manifestation of anxiety in order to enhance efficacy of treatment. The degree to which CBT has been expanded to meet the clinical needs of youth with ASD ranges on a continuum. Three RCTs for the treatment of anxiety in ASD currently exist in the literature (Chalfant, Rapee, & Carroll, 2007; Sofronoff et al., 2005; Wood et al., 2009). Additional RCTs are currently underway (e.g., White et al., 2010), including a multi-site investigation by the authors of this chapter in early adolescents with ASD and comorbid anxiety. All three of these studies had some methodological limitations, but overall, the results demonstrate a reduction in anxiety symptoms (with two of the more scientifically methodologically sound studies demonstrating that up to 71% of the children in treatment no longer met diagnostic criteria at the completion of CBT treatment) (Chalfant et al., 2007; Wood et al., 2009). More importantly, these studies employed methodological components (e.g., random assignment to conditions) that were consistent with the criteria necessary to establish empirically supported interventions (Chambless & Hollon, 1998). The RCTs will be described in more detail below in order of the increasing degree to which CBT was expanded. These exemplars will be used to demonstrate efficacy for the respective interventions and the specific adaptations that were made to the traditional model of CBT.

In the first example, Chalfant et al. (2007) developed an adapted CBT model that was tailored to accommodate the visual and concrete learning style of ASD. Forty-seven children aged 8–13 years-old diagnosed with high-functioning ASD were provided with group CBT and were randomly assigned to either immediate or wait-list conditions. The CBT was adapted from a program intended to treat core anxiety symptoms in typically developing children. The sessions were 2 h in duration and intervention was extended to 6 months (12 weekly sessions and three monthly booster sessions) to accommodate additional skill-building opportunities. Treatment effects were assessed with a structured diagnostic measure; child self-report measures; parent-report measures; and a teacher-report. Results at posttreatment revealed that about 71% of the children in the immediate treatment group no longer met criteria for a primary anxiety disorder in comparison to 0% of the youth in the wait-list group. Children in the CBT group demonstrated a greater reduction in the number of anxiety

diagnoses from pre- to post-treatment and the self-, parent-, and teacher-reports generally showed that the CBT group reported significantly less internalizing thoughts about anxiety and self-esteem, reduced anxiety symptoms, and less emotional difficulties relative to the waitlist group. Some methodological concerns for this study were that independent evaluators blind to treatment assignment was not employed to administer the post-treatment diagnostic interviews and treatment fidelity was not examined.

Modifications to the CBT program were primarily in the presentation of materials and enhanced skill-building support through concurrent parent training. Specifically, Chalfant et al. (2007) sought to accommodate the visual and concrete learning style of youth with ASD. Visual aids and structured worksheets were used extensively for psychoeducation, anxiety symptom recognition, and skill-building of coping skills. For example, the youngsters were provided with worksheets to encircle their bodily feelings associated with anxiety from a list in order to alleviate demands on verbal skills. Cognitive restructuring activities (e.g., developing coping thoughts) were also simplified to accommodate language impairments. Concrete and behaviorally based activities were of focus through relaxation and exposure activities. However, the exposure activities were completed at home as sessions focused on planning exposure activities with the child and their family. No live coaching was provided to parents through in vivo exposures. Although parents completed a daily diary entry to record the outcomes of the home-based exposures, no checks were in place to validate the completion or fidelity of home-based exposures.

Parents were provided with a training program in parallel to their children's group therapy. To supplement the development and practice of the children's coping skills (e.g., parents providing exposure activities) the parent component was comprised of anxiety education and teaching relaxation strategies, cognitive restructuring exercises, graded exposure, parent management training for behavioral problems associated with anxiety, and relapse prevention. Conjoint parent–child sessions were not a part of this CBT model, providing little opportunity for direct practice or in vivo feedback from trained clinicians.

In the second example, Sofronoff et al. (2005) conducted an RCT with 71 children, ages 10–12 years, diagnosed with Asperger's Disorder who were randomly assigned to: (a) child-based intervention, (b) combined child and parent intervention, or (c) waitlist condition. CBT was provided in 2-h sessions for 6 weeks in group therapy format. The child condition consisted of therapy provided in group format to children, with no parent training (parents were only informed of weekly assigned home-based exposures). In the child–parent condition, children received therapy in group format while parents also were trained to be "co-therapists" in parallel to the child sessions. These separate, concurrent parent sessions involved teaching parents intervention strategies and distal coaching on implementing exposures. Treatment effects were examined using an exploratory measure to assess for children's self-generation of coping strategies and traditional parent-report measures.

Across measures, a significant improvement was observed in the CBT groups in comparison to the waitlist condition, with greater improvement observed in the parent–child intervention condition. Children in the CBT groups demonstrated an increased ability to generate coping strategies to a hypothetical scenario, and a significant reduction in the total number of anxiety symptoms and social worries relative to the waitlist group youth. However, this study lacked more rigorous diagnostic assessment, psychometrically sound measures, and methodology (e.g., did not employ independent evaluators). Interestingly, most of the significant changes in the measures were observed in follow-up (6 weeks after treatment was completed), rather than posttreatment. The authors cited that the children may have needed additional time to benefit from the coping strategies.

This treatment program went beyond modifying materials to make them more understandable to the youth (simplifying materials or using visual aids). Sofronoff et al. attempted to make the concepts more *relatable* and targeted a few core ASD areas implicated in compounding anxiety

symptoms. First, the presentation of coping concepts and skills incorporated children's special interests. For example, capitalizing on a common special interest in science among children with ASD, the youth in this intervention were given the role of a "scientist" or "astronaut" to practice and learn coping skills. Also, the metaphor of a tool box (tools to fix feelings, social tools, and thinking tools) was used in presenting coping strategies and emotion awareness. Second, social awareness about the behaviors of the children and other people around them within anxiety provoking situations was targeted. Last, the authors used cartoons and thought bubbles in relation to children's anxiety-related scenarios that were borrowed from an intervention strategy used by youth with ASD to promote awareness and development of core social skills.

In the third example, Wood et al. (2009) significantly expanded upon traditional CBT by developing a comprehensive CBT model that emphasized treatment elements to target both core ASD features associated with anxiety symptomology. In this RCT, 40 children aged 7–11 years were randomized to either 16 weeks of 90 min sessions of a family-based CBT program plus 2 school consultation sessions or a waitlist. Treatment effects were assessed with a structured diagnostic interview; independent rating of improvement in anxiety (Clinical Global Improvement Scale (CGI-I)); and parent and child report of anxiety symptoms. The results showed large effect sizes for most outcome measures; remission of all anxiety disorders for more than 50% of the children in the immediate treatment group by posttreatment or follow-up; and a high rate of positive treatment response on the CGI-I (78.5% from intent-to-treat analyses). The children in the study had an average of 4.18 psychiatric disorders at intake, yet despite a high level of comorbidity, they demonstrated primary outcomes comparable to those of other studies treating childhood anxiety in typically developing patients (e.g., Barrett et al., 1996; Wood et al., 2006). For treatment completers, 64% of the children in the treatment group did not meet criteria for *any* anxiety disorder at posttreatment. Parent-reported anxiety symptoms also decreased significantly in the CBT group as compared to the waitlist group. However, child-reported anxiety did not differ significantly from pretreatment to follow-up. The authors described that a floor effect was expected, as baseline levels were low and decreased with treatment. This study had several methodologically rigorous elements including randomization and use of independent evaluators.

Wood et al. (2009) significantly enhanced the traditional CBT model by specifically targeting core ASD areas associated with the expression of anxiety in addition to making the CBT relatable to this group of children. In conjunction with the traditional coping skills training (developing coping thoughts) and in vivo exposure elements concerted efforts were made to treat both anxiety and associated ASD features. The core deficits of social-communication, perspective-taking skills, and the presence of idiosyncratic restricted interests and repetitive behaviors were actively targeted concurrently with anxiety symptoms. Social skills closely tied to anxiety and likely to interfere with the practice of more adaptive coping skills were addressed. For example, social skills deficits have been associated social anxiety in ASD (Bellini, 2006); therefore, Wood et al. used social coaching techniques to teach functional social skills to children and their parents. Specific strategies included the identification and practice of age-appropriate social overtures (e.g., joining in games with peers), friendship (e.g., listening to friends) and playdate hosting skills (e.g., playing flexibly, giving complements), reciprocal conversational skills, and perspective-taking skills (e.g., understanding the thoughts of peers). Peer intervention techniques (training peers to promote increased interactions with the target child) to develop positive peer relationships within naturalistic such as park and school were also implemented.

Wood et al. integrated the use of special interests into treatment. They specifically used special interests as motivators for treatment and as a medium for learning and practicing adaptive coping skills. For example, a child's interest in cartoon characters was used as a reward for practicing coping skills and to develop thought bubbles

about anxious and coping thoughts through related cartoons, which was drawn from the perspective that idiosyncratic interests and repetitive behaviors can be used to motivate children with ASD (Baker, Koegel, & Koegel, 1998). Engagement in these interests was gradually suppressed for increasing lengths of time through a contingency management plan to increase the likelihood that children would benefit from using functional and coping skills given that these behaviors can detrimentally interfere with functioning over time (Klin, Danovitch, Merz, & Volkmar, 2007).

Self-help skills necessary for daily, adaptive functioning was also an intervention enhancement. Poor adaptive skills associated with ASD (Howlin, Goode, Hutton, & Rutter, 2004) and impaired self-help skills in typically developing children with anxiety disorders (Wood, 2006) served as guiding posts to promote youth with ASD and their parents to practice age-appropriate self-help skills (e.g., showering independently). Wood et al. targeted comorbid externalizing symptoms associated with anxiety in children with ASD (Kim et al., 2000) by promoting children's perspective of these behaviors (through role-play and Socratic Questioning) and developing a contingent reward plan for the gradual increased display of appropriate behaviors and use of emotional regulation strategies.

Child motivation and active treatment participation was also an element of the expanded CBT. The authors concentrated their efforts in parent participation, practice within natural settings to enhance generalization of skills, and continuously used rewards (e.g., access to playing videogames) and other positive experiences (humor, restricted interests) for the youth to actively participate in treatment. Parent- and teacher-training components were included to ensure that coping skills were employed in daily settings. The program incorporated these elements to maintain engagement and to promote the recall of adaptive responses that were informed through the long-established efficacy demonstrated by the literature on treatments targeting core ASD skill development (Hwang & Hughes, 2000; Koegel & Egel, 1979; Koegel, Koegel, & Brookman, 2003).

Integrating the treatment and efficacy considerations from the available research highlights a number of important aspects in treating anxiety in youth with ASD using CBT. First, at the most basic level, CBT must be presented in a way that is understandable to youth with ASD to ensure the uptake of concepts and skills. Presenting materials, concepts, and opportunities to practice both more adaptive coping thoughts and behaviors need to be modified to accommodate the learning profiles of children and adolescents with ASD. This largely has been accomplished through the use of visual aids (cartoons, lists, diagrams), increased structure in the sessions (developing predictable routines in the layout of the sessions), presenting concepts using clear, explicit, and simple language, and increased practice identifying and recognizing emotional and body feelings related to anxiety. Second, CBT concepts and skills should be made relatable to children with ASD to increase the likelihood of their active participation in treatment and generalization of skills in real world settings. This requires going beyond the simple modification of materials by individualizing treatment and incorporating elements of these children's interests to serve as a medium for developing skills and capitalize on the motivational and reinforcing properties of special interests in ASD. Additionally, it requires skill building through in vivo exposures in naturalistic settings rather than through more distal role-play or limited to a clinic setting. Practice and mastery in real world settings will make the skills relatable to children with ASD and develop adaptive schemas that are relevant to their lives and likely to be employed by the children in actual situations. Parent training components also ensure that the skills will be practiced by the children and make the concepts and skills relevant to both the family and the child. Third, enhancement of CBT for youth with ASD also requires going a step further to address the complex integration between anxiety expression and core ASD features. It is clear from the research that anxiety can be manifested in a unique way in ASD. Although anxiety can be expressed in prototypical form similar to non-ASD cases of anxiety, anxiety seems to have a reciprocal, dynamic relationship with core ASD

features. Anxiety can exacerbate core ASD features and ASD characteristics can contribute to anxiety expression. Therefore, in the case of CBT for youth with ASD, additional components of treating this unique intersection of anxiety and ASD characteristics seems to be an integral aspect of treatment for this group. In tandem with core CBT skills of cognitive restructuring and mastering coping skills, social-communicative skill enhancement (e.g., conversation skills or playdate skills), mastery of age-appropriate adaptive skills, development of flexibility in interests and the ability to suppress restricted interests when necessary are some behaviors that can be promoted and acquired alongside traditional CBT skills. Increased research in both the theoretical understanding of ASD and anxiety and components associated with efficacious treatment will further guide the field in ensuring that children with ASD receive effective interventions.

Case Study

Case description. Oliver was an 8-year-old boy who attended the second grade at a local public elementary school. He was fully included in the general education classroom with support from a one-to-one aide. Oliver was diagnosed with high-functioning autism at age 3. He was referred for psychosocial treatment by his psychiatrist due to impairing symptoms of anxiety. A modified and enhanced family-based CBT program (Wood & McLeod, 2008) was provided consisting of 16, 90-min sessions, one follow-up booster session, and two school visits. Each session consisted of individual child, individual parent, and conjoint child–parent portions.

Clinical profile. Oliver met diagnostic criteria for three anxiety disorders: social anxiety, separation anxiety, and obsessive-compulsive disorder. He exhibited significant apprehension about social interactions and negative social evaluation. As a result, he avoided partaking in age-appropriate activities (e.g., class participation). Distress in social situations further compounded his ASD-related social deficits, preventing him from developing friendships at school. Also, he presented with significant anxiety towards separation from his mother, endorsing fears that either he or his mother would be harmed or "stolen." For example, Oliver exhibited excessive clinginess around his mother and engaged in co-sleeping with his parents. With regard to OCD, his obsessions included repeated and unwanted thoughts about the number six, the color red, contracting germs, and distressing images of a pony character from a cartoon he enjoyed watching. He experienced times when mental images of the pony became intrusive and distressing. Compulsions included repeated handwashing, hoarding trash, and a set of ritualistic behaviors he felt compelled to perform "just right" involving his stuffed animal. Consistent with ASD symptoms, Oliver demonstrated impairments with reciprocal social interactions (i.e., lack of shared enjoyment), communication (i.e., difficulty sustaining conversations), and stereotyped interests and behaviors (i.e., occasional hand flapping). His special interests were related to vehicles, science, and cartoons intended for a younger audience. Oliver's adaptive skills were below age expectations.

Sessions 1–3: building coping and independence skills. The general focus was on establishing rapport, providing psychoeducation on the nature of ASD and anxiety, collecting information on anxiety symptoms, and providing an overview of the CBT program. Oliver and his mother were taught core cognitive restructuring skills (recognizing anxiety feelings, identifying anxious thoughts, developing coping thoughts, and the concept of gradually facing fears). Rapport building focused on identifying Oliver's interests. A functional assessment of Oliver's ASD features and anxiety was conducted. His mother identified his current level of adaptive skills and selected age-appropriate target skills, focusing on private self-care tasks (self-grooming). She was taught key parenting communication strategies (providing choices, gradually fading assistance). Oliver's mother identified powerful rewards ranging from daily to longer term incentives to use throughout the program to increase his motivation for completing CBT assignments.

Oliver was encouraged to indicate his preference for labeling anxiety (he preferred the term "scared" and endorsed feeling "hot" when worried). Systematic Socratic questioning was employed to recognize bodily cues, challenge anxious cognitions ("The pony might get me"), develop adaptive coping thoughts ("The pony is a silly cartoon, so it can't harm me!"), and incrementally face feared situations with the aid of cartoon-based stories involving his special interests relevant to anxiety-provoking scenarios.

Sessions 4–5: development of the hierarchy and treatment plan. The focus was on providing an overview of exposure therapy, developing the exposure hierarchy, and implementing an incentive system. Oliver and his mother were presented with a list of fearful situations based on his diagnostic interview and information from the initial sessions. They provided ratings for each item on the hierarchy that included both anxiety and ASD-related symptoms (e.g., talking about his special interests). Coaching was provided to his mother to plan, negotiate, and complete exposures.

Sessions 6–15: comprehensive skill application in real world settings. The focus was on conducting in vivo and home-based exposures and monitoring the reward system. Concurrently, skills compromised by core ASD symptomology such as appropriate social entry behaviors (e.g., joining games) were targeted towards the middle of the treatment phase. Items rated as easier on his hierarchy were first attempted; steadily including several items from across anxiety domains. Cognitive restructuring was practiced both in session and at home to develop coping and parent communication skills. Home-based exposures served as extensions of in-session exposures. Given his interest in science, he was encouraged to think about exposures as a way to go about "busting myths." Homework gradually targeted multiple anxiety symptoms, self-care areas, and ASD-related deficits.

Social coaching intervention was provided to the parent and child. First, role-playing of typical social exchanges between Oliver and his peers was practiced in the session and at home (e.g., asking to join in a game). Then, his mother was encouraged to consider social coaching as a long-term strategy; look for naturalistic opportunities to practice positive social exchanges in the community (during school drop off and pick up); and provide him with positive feedback for practicing social and coping skills in real world situations.

Oliver was taught friendship skills of hosting playdates with peers. His mother was taught skills to foster Oliver's friendships and identify potential friends for playdates. Oliver was introduced to and practiced the rules of a good host (provide compliments, stay with the friend, and play flexibly by allowing the friend to choose the games). He was asked to select peers, make phone calls to invite them, and host playdates as part of his ongoing CBT homework.

Oliver's mother was taught strategies for increasing Oliver's age-appropriate activities. She was encouraged to raise Oliver's interest in age-appropriate TV shows enjoyed by most children his age. His interest in idiosyncratic topics and immature activities gradually diminished over time by rewarding him for increasing lengths of time in which he did not engage in these behaviors or engaged in more appropriate activities. He was gradually asked to refrain from watching preschool cartoons or talking about them for 1 day. He was rewarded for watching or discussing more age-appropriate topics.

The last phase of treatment involved a school observation, developing school-based exposures and home-school notes, and training relevant adults in the school setting. Social coaching was introduced to and implemented by his one-to-one aide during recess and lunch in the context of naturally occurring peer exchanges.

Session 16: termination. Treatment progress was reviewed with both parent and child during which they planned future home-based exposures to practice coping skills, self-care, and ASD-related skill development.

Session 17: follow-up. The purpose was to maintain Oliver's treatment gains and prevent symptom relapse through progress review and problem-solving to address new areas of anxiety in a collaborative manner.

Treatment outcome. Oliver no longer met diagnostic criteria for any of the three anxiety diagnoses. For example, obsessions related to cartoon characters remitted and he did not engage in compulsive hoarding. He made gains in friendships, as evidenced by an increased number of playdates in which he had the opportunity to play the role of host and guest. Overall, his anxiety and related ASD symptoms improved to the extent to which they became manageable, increasing his quality of life and functioning.

Conclusion and Future Directions

Anxiety at clinical levels is a phenomenon that occurs at high rates in youth with ASD and requires treatment. It is becoming increasingly evident that in order to accommodate the complexity of anxiety expression in ASD, CBT must be tailored to complement and meet the needs of youth in this population. Traditional CBT has provided solid foundations for the effective treatment of anxiety in typically developing children and those with ASD. Similar to the growing consideration in the field for more refined diagnostic methods to identify comorbid anxiety diagnoses, the development of CBT programs for anxiety in ASD is continuing to evolve with the growing fund of knowledge in the field. Children and adolescents with ASDs are susceptible to anxiety disorders and require and deserve appropriate treatment to promote their psychological well-being. The advancement of enhanced CBT interventions should be guided by research on the development of anxiety in both typically developing children and youth with ASD, paired with findings from both psychosocial and behavioral treatments for youth on the autism spectrum. In this way, CBT can have a lasting and meaningful impact on youth with ASD and concurrent anxiety.

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Treatment of Comorbid Anxiety and Disruptive Behavior in Youth

6

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Psychiatric comorbidity commonly occurs with childhood anxiety disorders (Geller, Biederman, Griffin, Jones, & Lefkowitz, 1996; Verduin & Kendall, 2003) and contributes to functional impairments beyond the influence of anxiety (Storch, Lewin, Geffken, Morgan, & Murphy, 2010; Sukhodolsky et al., 2005). The presence of comorbid anxiety and disruptive behavior disorders (DBD, e.g., conduct problems, oppositional/defiant behavior, impulsivity, hyperactivity) may be particularly problematic for children and families (Stringaris, Cohen, Pine, & Leibenluft, 2009). Comorbid disruptive behavior disorders (DBD) are relatively common (Loeber, Green, Lahey, Frick, & McBurnett, 2000), are functionally impairing (Stringaris et al., 2009), and are associated with lower treatment response rates in youth who receive evidence-based anxiety treatments (Storch et al., 2008). Additionally, anxiety and DBD symptoms tend to become even more impairing across the life span if they are not successfully treated in childhood (Kendall, Safford, Flannery-Schroeder, & Webb, 2004; Offord & Bennett, 1994). Because of the importance of increasing and improving treatment for youth with comorbid anxiety and DBD, this chapter reviews research on the phenomenology of comorbid childhood anxiety and DBD symptoms as well as the extant treatment approaches. Additionally, in an attempt to illustrate the application of interventions to treat comorbid anxiety and DBD symptoms, a case example is provided.

Childhood anxiety is associated with disruptions in academic, social, and family functioning (Ginsburg, Siqueland, Masia-Warner, & Hedtke, 2004; Langley, Bergman, McCracken, & Piacentini, 2004; Langley, Lewin, Bergman, Lee, & Piacentini, 2010; Woodward & Fergusson, 2001), the development of psychopathology in adulthood (e.g., anxiety, depression, substance abuse) (Aschenbrand, Kendall, Webb, Safford, & Flannery-Schroeder, 2003; Kendall et al., 2004; Woodward & Fergusson, 2001), and an increased risk for comorbid psychiatric disorders (Geller et al., 2000; Kendall et al., 2004; Langley et al., 2010; Verduin & Kendall, 2003). Although there often is variation in which the disorders are classified as DBD, we define DBD to include oppositional defiant disorder (ODD), conduct

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disorder (CD), and attention-deficit/hyperactivity disorder (ADHD). Furthermore, although categorically distinct, some overlapping phenomenological features exist between anxiety and DBD.

Studies comparing phenomenological characteristics of youth with comorbid anxiety and disruptive behavior symptoms to youth with anxiety symptoms alone are limited. One study found that 44% of children with pediatric obsessive–compulsive disorder (OCD) display comorbid disruptive behavior symptoms and a similar percentage (43%) displayed comorbid ADHD symptoms (Geller et al., 2000). Among non-OCD anxiety, estimates are similar: 28% of youth with GAD, 34% with separation anxiety disorder (SAD), and 15% with social phobia (SP) displayed disruptive behavior symptoms (Verduin & Kendall, 2003). In a meta-analytic review, Boylan, Vaillancourt, Boyle, and Szatmari (2007) found an average odds ratio estimate of having ODD with a comorbid anxiety disorder between 5.4 (community samples) and 8.9 (clinic-referred samples). Approximately 25–32% of youth with ADHD also display comorbid anxiety (MTA Cooperative Group, 1999; Spencer, 2006). Moreover, an interaction may exist between age and the development of anxiety in youth with DBD. For example, Biederman et al. (2006) found a 7% increase in the prevalence of co-occurring anxiety disorders from age 11 years to mid-adolescence in a sample of youth with ADHD. Although additional research is needed to establish the role of development in the expression of anxiety and disruptive behaviors, the increasing demands of adolescence may affect the development of both disorders (Guevremont & Dumas, 1994).

Although few studies have investigated this phenomenon, the presence of comorbid anxiety and DBD may result in greater psychosocial impairment than is produced by either type of disorder alone. A study by Storch et al. (2010) found that youth with comorbid DBD and OCD had greater OCD-related symptom severity, OCD-related impairment, overall anxiety levels, and other symptoms of internalizing psychopathology relative to youth with OCD but no significant DBD symptoms. Additionally, the presence of DBD symptoms was associated with greater family accommodation of symptoms and less symptom resistance. Similarly, a study by Drabick, Gadow, and Loney (2008) found that children with comorbid GAD and ODD displayed greater conflict with family members and had more co-occurring symptoms than children with either single diagnosis. Thus, comorbid anxiety and DBD symptoms have a negative impact on child and family functioning beyond the influence of either type of condition.

Other combinations of anxiety and DBD also are problematic. For example, children with ADHD and CD are at risk for developing anxiety and experiencing significant impairments in their psychosocial functioning (Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998). These youth often have difficulty adjusting to social environments, which can cause distress as they age and value social relationships (Guevremont & Dumas, 1994). Similarly, children with comorbid OCD and ADHD diagnoses display significant difficulties in social functioning, school problems, and an elevated risk for depression (Sukhodolsky et al., 2005). Overall, research suggests that the presence of disruptive behavior contributes to the development of additional psychopathology and exacerbates the impact of other disorders on children's functioning (Loeber et al., 2000; Storch et al., 2010).

Conceptualizing the Problem

Several hypotheses have been put forth to explain the co-occurrence of disruptive behavior and anxiety in youth (Jarrett & Ollendick, 2008; Lilienfeld, 2003). First, disruptive behavior may serve an operant function. For example, children may engage in reactive disruptive behavior that serves to reduce exposure to anxiety triggers. In this regard, Bubier and Drabick (2009) suggest that reactive aggression is an impulsive behavior that is learned over time and eventually becomes a typical pattern of responding. This behavior is often elicited when a child is in an anxiety-provoking or threatening situation, does not see a possibility of an easy escape, and experiences emotions that are difficult to control. Children with high levels of anxiety often are sensitive to

experiencing dysphoric emotions, which may cause them to be irritable, highly reactive, disruptive, and potentially aggressive (Walker et al., 1991). Additionally, recent evidence suggests that parental accommodation of anxiety symptoms may contribute to co-occurring disruptive behavior (Flessner et al., 2011; Storch et al., 2007, 2010). Parents of children with both OCD and disruptive behavior may respond differently to their child's "fearful" behaviors as compared to a "behavior problem," which can lead to inconsistent, ineffective parenting and increased disruptive behaviors (Lehmkuhl et al., 2009).

Second, although there is limited evidence for this phenomenon, disruptive behavior may precede anxiety in some cases. For example, co-occurring impairments in academic, family, and social functioning in youth with disruptive behavior disorders may contribute to the development of anxiety. In this regard, children with ADHD may experience increased anxiety related to academic struggles, problematic interactions with others, or negative consequences resulting from their disruptive behavior (e.g., losing privileges).

Finally, other studies have found no temporal relation between anxiety and disruptive behaviors (e.g., Baldwin & Dadds, 2008). Anxiety and DBD symptoms may co-occur due to shared polygenetic traits, neurological dysregulation, and the influence of various family factors (e.g., inconsistent caregiving) (Baumgaertel, Blaskey, & Antia, 2008; Jarrett & Ollendick, 2008).

Factors Contributing to Treatment Complexity

Given the complex and multi-determined relations of anxiety and DBD, clinicians should conduct a functional assessment to examine the antecedents and consequences of each behavior to identify which function the behaviors serve (see Haynes and O'Brien (2000) for a comprehensive review of functional assessment). Parents may not know how to respond to children who display concomitant anxiety and disruptive behavior. For example, children may scream to get their parents' attention or to avoid feared stimuli. Although the behavior may be topographically similar, understanding the function is imperative in determining appropriate treatment and in modifying parent–child interactions.

Behavioral parent training has been studied extensively for the treatment of DBD symptoms in preschool-aged children through adolescents (e.g., Barkley, 1997; Eyberg & Bussing, 2010; McMahon & Forehand, 2003). Many empirically based behavioral parent training protocols exist that include a variety of treatment components (e.g., positive attending, using time-out, giving effective commands). Based on operant conditioning principles (e.g., Skinner, 1953), parents essentially are taught to change their interactions with the child and their responses to the child's behavior using differential reinforcement of prosocial behaviors (see Herschell, Calzada, Eyberg, & McNiel, 2002; McMahon & Forehand, 2003; Patterson, 1971; Patterson, Reid, Jones, & Conger, 1975). Component analyses of behavioral parent training suggest that the inclusion of positive interactions with the child, the use of a time-out from positive reinforcement procedure, opportunities to practice new parenting skills with the child during therapy sessions, and consistent parental responding predicted moderate to large treatment effects on externalizing behavior (mean effect sizes = 0.36–0.69). There is some evidence that treating DBD symptoms also results in improvements in anxiety symptoms (Chase & Eyberg, 2008) or other internalizing symptoms (i.e., anxiety and/or depression; mean effect size = 0.40) (Kaminski, Valle, Filene, & Boyle, 2008).

Cognitive–behavioral therapy (CBT) is the first-line treatment for pediatric anxiety disorders and is associated with robust effects. For instance, Silverman, Pina, and Viswesvaran (2008) reported an average effect size for CBT for an anxiety disorder in youth as 0.99. Effect sizes for CBT for OCD are even higher. For example, Watson and Rees (2008) reported an average effect size of 1.45 for the efficacy of CBT to treat pediatric OCD. Furthermore, 50–80% of youth with anxiety achieve symptom remission when CBT is combined with selective serotonin reuptake inhibitor (SSRI) medication (Pediatric Obsessive Compulsive Disorder Treatment Study Team

2004; Walkup et al., 2008). In CBT, the treatment typically involves having a patient approach fear-evoking stimuli to extinguish his or her anxious response through repeated exposure (Gillan & Rachman, 1974; March, Frances, Carpenter, & Kahn, 1997; Silverman et al., 2008). This is accomplished in several steps. First, a patient-specific hierarchy of fears is developed with the help of the therapist. Second, the patient is encouraged to approach the fear-provoking stimuli (referred to as an exposure task) starting with less feared stimuli to ensure success and reinforce approach behavior. The exposure task is then repeated until the stimulus no longer triggers anxiety or triggers minimal anxiety. Third, exposure tasks are repeatedly conducted with increasingly more anxiety-provoking stimuli as the patient progresses in therapy. If a patient typically responds to anxiety with a compulsion or ritual (as in the case of OCD), he or she is asked to refrain from doing the ritual or avoidance behavior which allows escape from the anxiety-provoking stimuli and interferes with the autonomic habituation essential for extinguishing the fear response. The cognitive component of CBT involves teaching the patient to recognize irrational thoughts and challenge or externalize them (Kendall, 1992; March & Mulle, 1998).

Although most anxious children respond favorably to CBT, comorbid anxiety and DBD are associated with lowered treatment response. Storch et al. (2008) found youth with comorbid OCD and DBD symptoms to display lower CBT treatment response rates (46% remission) compared to youth with single OCD diagnoses or comorbid anxiety disorder diagnoses (92% remission). Similarly, children with comorbid anxiety and DBD symptoms displayed lower response rates to behavioral therapy and stimulant medication than did children with ADHD alone in the Multimodal Treatment Study of Children with ADHD (March et al., 2000). Further, attenuated treatment outcomes have been observed for youth with comorbid DBD and OCD symptoms in pharmacotherapy trials (Geller et al., 2003; Masi et al., 2005) and in combined medication and psychotherapy trials (Wever & Rey, 1997). Other studies of anxiety have found that comorbid DBD improves simply by treating the child's primary anxiety disorder (Flannery-Schroeder, Suveg, Safford, Kendall, & Webb, 2004). However, Rapee (2003) found that children with anxiety and DBD symptoms displayed worse DBD symptoms 12 months after treatment compared to baseline.

There are several reasons why children with comorbid DBD and anxiety may not respond to treatment. First, children with comorbid DBD symptoms may display defiance related to engaging in exposure tasks, completing homework assignments, or taking medication (Storch et al., 2007). Some children may even become aggressive when therapists and parents attempt to expose them to anxiety-provoking stimuli. This can then influence parents or therapists to reduce the magnitude of exposure tasks or may even make them reluctant to expose the child to anxiety-provoking situations altogether. Thus, disruptive behavior may decrease the level and frequency of behavioral exposures, which negatively impacts a patient's treatment response.

Second, hyperactivity, impulsivity, and inattention can interfere with engagement in therapy. Managing these behaviors during sessions can interfere with a therapist's ability to focus on therapeutic goals. In addition, youth with these comorbid symptoms may struggle to persist in therapeutic tasks or complete homework between sessions. In addition, these disruptive symptoms may interfere with the performance of other tasks (such as school homework or chores), which can cause them to take longer to complete and subsequently leave less time to engage in therapeutic tasks and homework. Thus, similar to how symptoms of ADHD interfere with academic engagement at school, therapists may find that children with comorbid ADHD and anxiety also struggle to engage in therapy.

Third, as suggested by Storch, Björgvinsson, Riemann, Lewin, Morales, & Murphy (2010), comorbid DBD symptoms can affect behavioral treatment of anxiety because of potential "secondary gains that make youth less motivated to reduce symptoms" (p.173). Building on the coercive parent–child interactions of DBD (Patterson, 1982), both parents and children are reinforced in some

ways when parents acquiesce to the child's disruptive behaviors. The child does not have to engage in the task, and the parents do not have to manage disruptive behavior. Thus, they are both rewarded by continued avoidance of feared stimuli (i.e., family accommodation) and escape from feared stimuli when the child becomes disruptive. Over time, the child may seek attention (e.g., parents rubbing back to calm down) and privileges (e.g., delayed bedtime) that have been associated with anxiety (i.e., secondary gains). These complex functional relations may make it more difficult to extinguish anxiety and likely affect family engagement in treatment and adherence to CBT homework.

Additionally, as a final feature that contributes to treatment complexity, research suggests that poor emotion regulation skills associated with the presence of ADHD may make children hypersensitive to anxiety (Kendall & Choudhury, 2003; Sukhodolsky et al., 2005). Therefore, the interaction of DBD symptoms and anxiety may be particularly challenging to manage in treatment as inattentive, oppositional, and defiant behavior directly impact mechanisms of change associated with anxiety treatment. Each one of the aforementioned factors complicate treatment, and in combination, they can interfere with children's habituation to anxiety, learning of adaptive ways to manage anxiety, improvements in family functioning, and generalization of skills outside of CBT sessions.

Treatment

Psychosocial Approaches. Although there is a need for tailored interventions to target anxiety and DBD symptoms, few studies have examined the effects of combined treatment on anxiety and DBD symptoms. In one such study, Levy, Hunt, and Heriot (2007) compared the effectiveness of group-delivered CBT for anxiety and group-delivered CBT tailored for children with anxiety and significant aggressive behaviors (i.e., scoring in the 90th percentile on both the Aggressive Behaviors and Externalizing scales of the Child Behavior Checklist). Both treatment arms consisted of nine sessions delivered over the course of 11 weeks, during which small parent groups and small child groups met separately for 1 h per week. In addition to standard CBT for anxiety components, the comorbid anxiety and aggression treatment incorporated anger management skills for the child (e.g., self-management, self-reflection, and self-monitoring skills, including self-talk; social problem-solving skills; behavior management; goal setting; and interpersonal group processes) and "education about aggression and a greater emphasis on behavior management techniques" for the parents (p. 1114). Results revealed that both interventions significantly reduced anxiety and aggressive behavior and that comorbidity did not affect treatment outcomes. Additionally, both treatment groups reported improvements in parenting practices and may have inadvertently involved the use of contingency management skills to engage children in treatment. Although the relatively small sample size precludes definitive findings, this study provides preliminary support for a tailored treatment approach for anxiety with comorbid aggression.

Several case studies offer preliminary support for the combined use of CBT for anxiety and behavioral parent training for DBD symptoms. One case study of a 10-year-old girl with OCD and disruptive behaviors incorporated four sessions of parent training prior to implementing CBT for OCD in order to address DBD symptoms and facilitate CBT (Lehmkuhl et al., 2009). After implementing behavioral parent training skills, CBT was implemented to treat the child's OCD symptoms, which resulted in reductions in both OCD and DBD symptoms. Another case study of a 6-year-old child with OCD and ODD involved working with the child's parents to differentiate between the functions of his problematic behaviors and implement behavioral parent training skills and CBT interventions (Ale & Krackow, 2011). Over the course of 23 sessions, they implemented positive attention, planned ignoring, time-out from positive reinforcement, and exposure tasks for a fear of accidentally swallowing and choking on buttons. Following treatment, the child exhibited mild OCD symptoms and no longer met criteria for ODD. These cases demonstrate the effects of working with parents

to incorporate a structured reward system to motivate compliance, differential reinforcement (i.e., provide attention for desired behaviors and ignore minor misbehavior and engagement in rituals) (Francis, 1988), and time-out from positive reinforcement for aggressive behaviors. Further, parent contingency management components can decrease aggressive behavior while increasing engagement in exposure therapy. Thus, family involvement is important in the treatment of children with disruptive behavior, and these case studies highlight the importance of including caregivers in anxiety treatment to address behavior problems and decrease family accommodation.

Results of the previous studies suggest that treatment to address anxiety and DBD (either concurrently or sequentially) may reduce both DBD symptoms and anxiety symptoms. With the exception of OCD, literature examining combined treatment for anxiety and DBD symptoms is limited. This may be due to the mixed findings which suggest that DBD symptoms may respond to standard CBT for anxiety (Flannery-Schroeder et al., 2004; Rapee, 2003). Moreover, it may be that OCD with comorbid DBD exhibits different clinical presentations and challenges than comorbid anxiety.

Treatment Strategies. Based on our clinical experience and on the evidence available, we suggest several strategies for treating comorbid anxiety and disruptive behavior. Initially, the clinician should gain a functional case conceptualization to better understand the relation of anxiety and DBD symptoms (i.e., which diagnosis is primary? how do the behaviors impact one another? what environmental variables maintain each behavior?). If the therapist notices that the disorders are co-primary and are interacting with each other and negatively affecting the family interactions, a concurrent approach to treatment will likely yield the strongest results. Therapists should work with parents to incorporate rewards (and possibly mild punishment) to encourage engagement in treatment and to reduce disruptive behavior. There should also be an effort to limit family accommodation and thus reduce negative reinforcement associated with escaping anxiety-provoking situations. This approach may be particularly useful with younger children, with children largely motivated by external rewards, and with those who engage in disruptive behavior to escape anxiety-provoking situations.

Alternatively, if functional analysis reveals that DBD symptoms primarily occur in the context of anxiety, it may be more effective to treat anxiety aggressively with CBT in lieu of the disruptive behavior, with the expectation that disruptive behaviors will decrease along with reductions in anxiety. However, this strategy may not be effective in cases where the disruptive behavior interferes with treatment implementation and compliance. In this case, continued assessment throughout treatment may indicate a need to incorporate specific behavioral techniques designed to increase engagement in treatment. In addition, for older adolescents, the use of motivational interviewing (see Erickson, Gerstle, & Feldstein, 2005 for review) may increase treatment engagement as well as reduce resistance.

For children with primary DBD symptoms and comorbid anxiety or for families who cannot engage in exposure tasks due to severely disruptive behavior, therapists should address disruptive behaviors first using parent training and contingency management techniques. Parent training interventions focused on increasing compliance and decreasing aggressive behaviors should first be introduced. As discussed above, key parent training skills include developmentally appropriate praising and rewarding desired behaviors, ignoring minor misbehaviors, and implementing time-out or loss of privileges for potentially dangerous misbehaviors. In our experience, addressing parenting skills can provide a stable foundation for children with comorbid DBD and anxiety. Children with primary DBD require parents and clinicians to have high-level contingency management skills while conducting exposure tasks; thus, shaping parent skills prior to addressing anxiety may be dually beneficial. Additionally, preliminary evidence suggests that children's anxiety may be reduced with the treatment of DBD symptoms (Chase & Eyberg, 2008). Furthermore, anxiety should be reassessed following behavioral parent training and addressed with CBT as necessary.

Pharmacological Approaches. In addition to the above strategies, pharmacotherapy also has been used to manage disruptive behavior in children with anxiety disorders. Haloperidol and chlorpromazine are neuroleptic medications that have treatment indications; however, these medications are associated with the presence of many untoward side effects (e.g., anticholinergic effects, extrapyramidal reactions, weight gain), so their use has declined with the advent of atypical antipsychotic medications that generally have safer side effect profiles. Although no FDA-approved pharmacological treatments exist for ODD or CD in typically developing youth, risperidone and aripiprazole (two atypical antipsychotic medications) have been approved for use with youth with comorbid autism spectrum disorders and disruptive and irritability symptoms (Food and Drug Administration, 2006). Additionally, these medications are often used off-label to treat disruptive behaviors in typically developing youth with anxiety disorders (Kutcher et al., 2004). In a multisite, double-blind placebo-controlled maintenance trial that included 436 children aged 5–17 years, Reyes, Buitelaar, Toren, Augustyns, and Eerdekens (2006) found that risperidone treatment was associated with reductions in disruptive behavior yet not "insecure/anxious" symptoms. Buspirone, an anxiolytic medication, also has been used off-label to treat comorbid anxiety and disruptive behavior. In an open-label trial, Pfeffer, Jiang, and Domeshek (1997) found that youth who were treated with buspirone displayed reductions in anxiety and aggression. However, treatment was discontinued for 25% of children due to increases in aggression or mania associated with treatment. In light of these findings and limited research on pharmacotherapeutic approaches for youth with comorbid anxiety and disruptive behavior, caution is warranted when using medication to treat this population.

Case Study

Background. Johnny Smith (pseudonym) was adopted when he was 2 days old. He was born one month premature and made limited eye contact with his parents as an infant. Johnny's verbal communication was delayed and a speech and language pathologist diagnosed him with speech apraxia when he was 2 years old. He has since been receiving regular speech therapy, with significant benefit. Johnny met all other developmental milestones within normal limits. Mrs. Smith, Johnny's adoptive mother, described his temperament as "difficult." Johnny has difficulty with social interactions and developing close friendships. After appropriately separating from his parents in early childhood (i.e., displaying no separation anxiety), Johnny started to display separation anxiety at age 8.

Johnny was brought to the clinic for evaluation when he was 9 years old and he was diagnosed with generalized anxiety disorder and separation anxiety disorder. In addition, he displayed disruptive behavior including explosive outbursts, noncompliance with adult requests, and physical aggression toward his parents and teachers. Johnny had numerous sources of anxiety. Examples included worrying about whether he would like the menu at a restaurant, whether the temperature in various places would be appropriate, if he would have a place to charge his electronics, etc. Johnny's generalized anxiety symptoms made it difficult for the family to go to restaurants and other public places. Johnny also had difficulty separating from his mother, which contributed to his reluctance to attend school. His parents described him as an "exact" child with a need to keep the same routine and order with tasks. For example, he refused to dress for school until after breakfast and he required his parents to serve him food following a specific routine. Any deviations from this routine, whether by Johnny or others, contributed to outbursts of disruptive and aggressive behavior. At the time of the evaluation, Johnny's parents accommodated his anxiety to prevent his aggressive behavior. They modified family routines such as avoiding restaurants, parties, and family gatherings. Additionally, they provided him with constant supervision in social settings and Mrs. Smith attended school with Johnny to help manage his classroom behavior. Johnny often spent a majority of the school day with his mother in a separate room away from his classmates. Despite these accommodations,

Johnny's academic performance suffered because he often refused to do schoolwork and homework.

Pharmacological Treatment. About a year before being evaluated by a psychologist, Johnny started taking escitalopram (5 mg), which resulted in immediate decrease in his anxiety and disruptive behavior. However, according to Mr. and Mrs. Smith, initial treatment effects associated with escitalopram waned over time and his dose was increased (up to 20 mg). With dosage increase, Johnny's rage attacks and separation anxiety symptoms also increased and he started to pick his skin compulsively, particularly on his legs. Subsequently, Johnny went through multiple medication trials that included oxcarbazepine, fluvoxamine, buspirone, clomipramine, and aripiprazole. Mrs. Smith reported that Johnny often initially responded well to a medication change, but the therapeutic effects gradually attenuated and he began to experience untoward side effects (e.g., sedation, agitation, and memory problems). At the time he presented for behavior therapy, Johnny was taking aripiprazole (15 mg), which was associated with modest reductions in disruptive behavior but also with significant weight gain.

Behavioral Treatment. Behavioral treatment for Johnny initially targeted his most disruptive and impairing behaviors including physical aggression. In the first session, Johnny refused to participate and became verbally and physically aggressive (pushing his parents and throwing objects) when his parents discussed his behavior problems with the therapist. He grew increasingly upset and began turning over chairs and struck his parents. These instances were dealt with loss of privileges, time-out, and, in one case, physical restraint.

Because Johnny's disruptive and anxiety-related behaviors were equally problematic and his disruptive behavior would likely prevent effective engagement in anxiety treatment (e.g., exposure therapy), the first several therapy sessions focused on parent training and behavior contingency management. Behavior management training included both parent-directed techniques (such as prompts, rewards, and consequences) and child-directed strategies (such as deep breathing, using a coping strategy [e.g., listening to a song], or expressing displeasure appropriately). Initially, concurrent with the behavior management session, issues related to Johnny's anxiety also were addressed (e.g., we attempted to talk to Johnny about ways to handle his anxiety); however, his disruptive behavior interfered with these efforts and he became aggressive when the discussion focused on his anxiety.

Over several sessions, with the use of specific rewards and consequences (usually related to loss of electronics for a specified time period), Johnny's behavior improved and engagement in therapy increased. However, Mr. and Mrs. Smith continued to accommodate Johnny's anxiety-driven behavior, which worked to sustain negative interactional patterns in the family. Thus, a plan was developed to address Johnny's anxiety that involved providing rewards for engaging in and practicing relevant anxiety-management techniques learned in therapy (e.g., not escaping the situation entirely or asking for help). At the same time, Mr. and Mrs. Smith were encouraged to reduce their accommodation of Johnny's anxiety-driven behavior gradually. For example, instead of letting Johnny escape an anxiety-provoking situation immediately, they allowed him to escape the situation after a few minutes.

Exposure to feared stimuli was initiated on the sixth therapy session. After a hierarchy of fears was constructed, Johnny's fear of elevators was targeted first using behavioral exposures. For instance, he stood near the elevators, then he put one foot inside, and so on. Each step was repeated until Johnny's habituated to the situation and the anxiety was manageable. After doing exposures in session, Johnny and his parents repeated them for homework. Using a combination of specific rewards and the implementation of strategies to reduce accommodation of Johnny's anxiety-driven behaviors, Mr. and Mrs. Smith were able to get Johnny to continue participating in behavioral exposures. Three weeks after initiating exposure therapy, Johnny was able to ride in elevators with an adult present. However, Mr. and Mrs. Smith were encouraged to continue exposures until Johnny was able to ride in an elevator alone. It is worth noting that Johnny often expressed a fear

that the elevator would become stuck. His parents had previously responded to this fear by providing him with reassurance, which had not been effective in reducing his anxiety. Therefore, Mr. and Mrs. Smith were encouraged to remain calm, neutral, and not indulge Johnny's reassurance-seeking behavior during exposures.

In addition to exposures, cognitive techniques were used to help Johnny cope with anxiety. These included having Johnny remember that anxious feelings are transient and decrease in intensity after several minutes. Johnny also was encouraged to affirm that he was able to manage anxiety in the past and would likely be able do it again in the future. However, it should be noted that cognitive techniques were not used to reassure the fear (e.g., "the elevator will not harm me") because of their potential to undermine the exposure process and their limited success in the home environment when provided by Mr. and Mrs. Smith.

As therapy progressed, Johnny began to develop more trust in his therapist, which allowed the therapist to begin challenging Johnny's "just right" symptoms using the exposure-based model. These included his need for strict adherence to routines, requirements for the food to be cooked a certain way, and clothes to feel right. Johnny was not able to express a feared consequence or a cognitive component underlying the anxiety driving these symptoms. Rather, he experienced more general distress and discomfort when his routines were not performed or accommodated. Exposure therapy for Johnny's "just right" symptoms involved graduated exposure to each anxiety-provoking situation until Johnny was able to withstand the situation with manageable anxiety.

Therapy concluded with addressing Johnny's symptoms of separation anxiety and refusing to attend school without his mother. A combination of strategies was used to address these problems. Specifically, gradual exposures were used in which Johnny was separated from his mother initially for short intervals that were steadily increased over a period of days. Johnny also was encouraged to use coping thoughts to manage his anxiety when separated from his mother (e.g., "I will see her after 30 minutes"). Engaging in exposures was reinforced by daily rewards. Any aggressive behavior during this process was addressed using consequences and rewards for demonstrating restraint.

Johnny's behavior and anxiety improved steadily during the course of treatment. He experienced fewer conflicts with others as his behavior improved and this contributed to improved adjustment at home and school. He also displayed less disruptive, defiant, and aggressive behavior following reductions in his anxiety. Although Johnny's anxiety and behavior were significantly improved by the end of 15 weekly therapy sessions, he still experienced occasional behavioral outbursts that were successfully maintained in monthly maintenance sessions.

Conclusions and Future Directions

Childhood anxiety and DBD are commonly comorbid, associated with significant impairment, and complicate treatment delivery and related outcomes. As discussed, the presence of disruptive behavior can interfere with children's engagement in treatment for anxiety and anxiety can contribute to children's resistance to engage in interventions to address disruptive behavior. Thus, this chapter highlights treatment strategies for youth with comorbid anxiety and disruptive behavior symptoms to introduce a treatment paradigm for this difficult to treat population. Some specific strategies involved treating anxiety as a primary indication, increasing motivation to engage in treatment, using contingency strategies to manage disruptive behavior while treating anxiety concurrently, and reducing disruptive behavior before initiating with anxiety treatment.

The treatment strategies discussed in this chapter are based on available research on childhood anxiety and DBD (alone and when comorbid), a very limited number of clinical trials, case studies, and our own clinical experience. One key factor in the treatment of youth with comorbid anxiety and disruptive behavior is that the treatment approach directly follows the case conceptualization. The specific strategy may depend on

whether a clinician views anxiety as primary and disruptive behavior a result of that anxiety, vice versa, or views both issues as comorbid without related causality. Regardless of which of these is the case, treatment is likely to be most effective when it is individualized for each person and is based on sound behavioral principles. The choice of whether to use sequential or concurrent treatment strategies should be based on the case conceptualization, the likely treatment response, and the current level of dysfunction caused by specific issues. Because of the sparse and sometimes conflicting nature of the extant literature, additional research is needed to support effective interventions for youth with comorbid anxiety and DBD. However, the available evidence suggests that combinations of cognitive and behavioral therapy are effective and components of extant treatment protocols can be modified to treat this population of youth.

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Diagnosis and Cognitive Behavioral Treatment of Anxiety Disorders in Young Children

7

Klaus Minde

Anxiety disorders are the most common form of psychopathology in children and adolescents with reported rates of 5–15% in the general child and adolescent population (Klein & Pine, 2002). This wide range of incidence may reflect variations in defining "a disorder" in children by different authors but may also be related to the varying peak onset times for individual anxiety disorders. Thus social phobia is more commonly seen during adolescence while the onset of separation anxiety disorder occurs more often during early childhood (Wittchen, Stein, & Kessler, 1999). This explanation is supported by Merikangas et al. (2010) who examined the lifetime prevalence of a mental disorder in 101,123 US adolescents. The group documented that 8% of their sample met the criteria for an anxiety disorder at age 4, going up to 14% at age 5 and 17% at age 6. Ten years later, 38% of girls and 26% of boys had experienced one or more anxiety disorders of which 8.3% were considered to be associated with severe impairment. The whole concept of giving preschool-aged children a psychiatric diagnosis based on categorical disorders is rather new. While there has long been interest in the normal and abnormal early development of children, academic investigators and clinicians talked about the "problems of preschool children" or focused on symptoms such as general behavior, sleep, or feeding problems (e.g., Richman & Lansdowne, 1988). In fact, in a 1995 volume of the Child and Adolescent Psychiatric Clinics of North America that dealt with the field of psychiatry of infants and preschoolers, there is no chapter on anxiety disorders, depression, or disruptive disorders (Minde, 1995).

A major reason for hesitating to "diagnose" preschoolers as suffering from categorical disorders has been the belief that young children need to be seen within the context of their families and that disorders in early childhood are best conceptualized as relational psychopathologies, that is, consequences of dysfunction in the parent–child environment system (Cicchetti, 1987). As a result, diagnostic assessments were usually based on observations of children and their caregivers, frequently documented by videotaped standardized interactional paradigms with detailed coding systems. This provided clinicians with relevant information and facilitated individually tailored treatment plans. Furthermore, by reviewing tapes with caregivers and asking them for their observations and comments, the clinician learned how parents interpreted their own and their child's behaviors, and any potentially distorted perceptions of the parents could then be addressed. There was also the general assumption that problem behaviors in the early years did not necessarily predict later psychopathology and that children "outgrow" these behaviors.

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Thus, the idea of distinct forms of psychopathology in the first years of life has not been easy to accept for some clinicians. However, there is now ample evidence that supports the presence of categorical psychiatric disorders in early childhood and in this chapter both categorical and dimensional approaches to psychopathology will be discussed. Furthermore, some of the research about the new understanding of anxiety disorders in preschool children and some recently developed and validated assessment tools will be described. Following that, the early clinical manifestations of these disorders will be discussed, based on data from developmental psychopathology and neuroscience. The emphasis will be on the clinical similarities to and differences from the anxiety disorders in older children and adolescents. Finally, some treatment programs that have been successful in helping young anxious children will be described and the importance of effective prevention strategies and early intervention programs for anxious children and their families are discussed.

The Diagnosis of Anxiety Disorders in Young Children

Traditionally, the diagnosis of psychiatric disorders in North America has been based on criteria defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM) of which there have been five versions so far, each reflecting the then current scientific understanding of psychiatric disorders. The last version, called DSM-IV TR (American Psychiatric Association, 2000), describes a number of disorders "first diagnosed in infancy, childhood, or adolescence." Among them are mental retardation, attention deficit hyperactivity disorder, and separation anxiety disorder. Some of these diagnostic categories ask to "specify early onset" which implies that the onset occurs before age 6 years. However, none of these diagnostic criteria allow qualifying statements about possible variations in, for example, the symptomatology of separation anxiety in toddlers and in adolescents. There are a number of reasons for failing to recognize the developmental aspects of a diagnosis. When DSM-IV was published 10 years ago, our knowledge about diagnosing infants and preschoolers was far less developed than it is today. Hence, the criteria for specific psychiatric conditions consisted of only a few cautionary remarks alerting the clinician to variations in the actual presentation of symptoms denoting anxiety in young vs. older children. This has led some clinicians to ignore that, for example, 3 out of a possible 8 symptoms are required by DSM-IV-TR to diagnose a 3-year-old toddler to suffer from a separation anxiety disorder, and instead have based their diagnosis on only 2 or even just one of the DSM-IV-TR required symptoms. This lack of fidelity to the required number of symptom partly reflects the reality that the cognitive development of most toddlers does not yet allow them to demonstrate at least 5 of the possible 8 criteria demanded for this diagnosis such as excessively worrying about possible harm befalling major attachment figures, having a history of nightmares, fear of getting lost or being kidnapped, or complaining of physical symptoms. Moreover, many anxious youngsters are not enrolled in out of home daycare programs as their caregivers do not think them appropriately ready for it—and hence they will not qualify for the item requiring "a persistent reluctance or refusal to go to school." Another example of difficulties clinicians have had with the DSM criteria for diagnosing young children is PTSD, a condition described in a significant number of preschool children who have experienced abuse or been exposed to violence. This is particularly relevant as there has long been concern about the potentially long-term impact violence or trauma have on children and an interest to determine whether there are developmentally defined age limits that may function as a shield against an actual memory of a trauma experienced during the first 5 years of life. This has led to nine studies over the past 15 years where investigators have examined the clinical presentations of traumatized young children and compared them with the DSM-based diagnostic criteria of PTSD. Four of these studies were by Scheeringa and his colleagues and in a recent review of the nine studies Scheeringa (2009) confirmed that (a)

PTSD can be reliably detected in children as young as 12 months; (b) it manifests most (but not all) of the items mentioned in DSM-IV and that; (c) an alternative criteria algorithm appears more developmentally sensitive than the DSM-IV-TR algorithm. Specifically, Scheeringa, Zeanah, Myers, and Putnam (2003) modified the wording of some items and changed the C criterion (numbing and avoidance items) by requiring only 1 rather than 3 items out of 7. This raised the rates of PTSD in clinic referred traumatized toddlers and children from 5 to 25%, equivalent to rates found in older populations.

While modifications in this specific diagnostic category has been helpful, it has also led to research examining to what extent psychiatric disorders during the preschool age are stable despite otherwise rapid developmental change. This work has been well summarized by Angold and Egger in a chapter (2004) and a special journal issue on preschool mental health (2007). They conclude that (a) preschool externalizing and internalizing problems are both quite stable and predict negative outcome years later; (b) it is not relevant to locate psychopathology "into the child" vs. "in the child's relationship," but, to cite Bronfenbrenner (1974), "to understand how the characteristics of the child and its social context work to produce psychopathology." In the same book, Egger and Angold (2004) present a Preschool Age Psychiatric Assessment Instrument (PAPA) which they had developed and used to assess 307 children aged 2–5 years, recruited from the Duke Children's Primary Care Clinic. Based on parental reports, the authors could identify eight specific diagnostic clusters (e.g., anxiety disorders) in these children which in turn were based on 25 modules (e.g., PTSD as a part of types of the anxiety disorder cluster). The anxiety disorder cluster included 9.5% of all children. In a later paper the authors showed that the PAPA had good 2-months test–retest reliability (kappas between 0.50 and 0.75, Egger et al., 2006). The assessment also includes detailed questions about the psychosocial environment of the child and his family and requires several hours to complete.

As far as anxiety disorders are concerned, clinical studies based on the criteria of developmental psychopathology demonstrate that infants who react negatively to novelty tend to show the same reaction as toddlers (Fox, Henderson, Marshall, Nichols, & Ghera, 2005). Likewise, toddlers labeled "behaviorally inhibited" by Kagan, Snidmen, Arcus, and Reznik (1994) show a two-to-fourfold increased risk for anxiety disorders in later childhood. As anxious children are frequently born to anxious parents (Gregory et al., 2007), these continuities likely reflect both genetic and environmental effects as well as gene–environmental interactions. Complicating these findings are studies reporting that up to 30% of anxious children face elevated risks for depressive disorders during adolescence, especially among girls (Caspi, Moffitt, Newman, & Silva, 1996). This suggests a heterotypic continuity. Infants with undifferentiated reactions to novelty become anxious toddlers and children who may mature into adults with anxiety and/or depression. Yet, three important questions remain:

1. It is not possible to predict whether an anxious child will remain anxious as an adult or will become an adult suffering from a major depressive disorder (MDD).
2. Studies show that only a minority of "at-risk" children ultimately manifest persistent disorders (Gregory et al., 2007). This implies that treatment of at least some anxious children will be successful because of the natural history of the condition and not because the therapeutic modality was relevant for the disorder.
3. Some of the present DSM-IV-defined psychiatric anxiety disorders provide special challenges because the symptomatology of some of them, e.g., Obsessive compulsive behavior, selective mutism, or PTSD, is more consistent over time and children suffering from these conditions are often not even included in published treatment outcome studies of anxiety disorders (Kendall, 1992).

New Insights from Neuroscience

Recent research in neuroscience provides additional explanations for the brain–behavior associations suggested by the above mentioned

clinical observations. For example, there is now solid evidence that the amygdala is necessary for learning how to deal with threats by regulating attention allocation to stressful events (Davis & Whalen, 2001). However, there are many more ways in which brain circuitry may relate to observed anxious behavior. As Pine (2007) states in his review on this topic, there are different neuronal components engaged when a person is confronted by innate threats or threats emanating from outside sources or a separation. There is also a different circuitry engaged when we learn to minimize punishment versus learn which cues are associated with specific punishers. In addition, significant developmental changes that occur in an individual's threshold for avoidance may be partly related to contextual factors such as family support and education. Thus genetically at-risk but clinically unaffected individuals may become symptomatic only when they are repeatedly exposed to stress, leading to an increasing appraisal bias, i.e., the individual will perceive even lower stresses as increasingly threatening (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007). There is also research that documents that distinct threats engage distinct brain circuitries which in turn leads to different behaviors (Blair, Mitchell, & Blair, 2005). This could also serve as a template for understanding the presence of distinct subtypes of anxiety disorders, such as OCD and SAD. Finally, different threats may show distinct associations with risk factors but not with actual disorders, i.e., they may primarily affect children who have anxious relatives (Pine, 2007). Other threat circuits appear to be additive, so that at-risk/affected individuals show the highest level of threat appraisal bias, with at-risk but unaffected people scoring lower although still higher when compared with non-at-risk/unaffected individuals.

It is clear even from this superficial review that there are an increasing number of investigators who attempt to integrate clinical and neuroscience perspectives on anxiety disorders. Their work shows that anxiety disorders are primarily the result of developmental perturbations that lead to a relative failure to regulate threat-related information processing functions in children and adults alike.

While there is a great need to better understand the potential causes of these perturbations and how they relate to specific types of anxiety disorders, there is agreement about the profound emotional and cognitive burdens these disorders place on the lives of affected individuals. There is also agreement on the premise that these disorders move from an undifferentiated and plastic state of a fear circuitry early in life to a more rigid and resistant threat appraisal bias in later childhood or adolescence. This suggests that treatment should start as early as possible for at-risk or affected children and should deal with modifying their regulatory abilities regarding both their emotional and cognitive responses to potentially threatening life experiences. Moreover, any effective treatment will need to involve caregivers as their potential role in modulating early reactions to threats in their young children is powerful and can lead to a significantly better quality of their lives.

It is of interest that the authors of two other prominent psychiatric assessment tools have also developed separate versions of their instruments for children aged 18–60 months (Achenbach & Rescorla, 2004) and for those aged 3–4 years (Goodman, 2001). Achenbach's CBCL identifies seven syndromes in the 18–60 months age group compared to eight syndromes in the older children. Syndromes reflecting the same DSM-oriented diagnosis are given a different name for the preschool group, for example, Emotionally Reactive instead of Affective Disorder. Goodman's (1997) Strength and Difficulties Questionnaire (SDQ) has five items for each of its five classes of behavior but uses somewhat different wording to adapt them for each age group and syndrome. While the published validation of the SDQ is restricted to children older than 5 years, the validity for 3 and 4 year olds has also been established (Goodman, 2011, Personal communication). However, neither questionnaire attempts to diagnose specific subtypes of anxiety disorders and the authors consider their instruments primarily as screening tools and advise that high scoring children should have a more

detailed clinical assessment although Goodman and Goodman (2011) have reported that in 5–16-year-old British children the SDQ scores predict the actual prevalence of clinician rated child mental health disorders within 1–2% ($R^2 = 0.89–0.95$).

Cultural Impact on Anxiety Disorders

Another issue complicating the assessment of psychiatric conditions in young children is the impact culture has on child development and psychopathology. While there is general agreement that cultural traditions shape behavior and the clinical presentation of psychopathology, few studies have addressed this issue. This seems even more important today as there is increasing evidence that epigenetic forces powerfully modulate the clinical expression of genetically determined medical and psychiatric conditions. Since caregivers transmit cultural narratives that will impact on possible gene expressions of psychopathology in their children, it would be helpful to have assessment tools that are sensitive to cultural values. There is currently no instrument available that addresses this issue specifically in preschoolers. However, Achenbach (2010) recently presented data that may provide the structure for such an endeavor. In contrast to DSM-IV-TR which represents a top-down approach to psychopathology where criteria have been formulated on the basis of the opinions of experts, Achenbach used data from evaluations of 47,987 children by their caretakers from 24 societies, using the CBCL and computed an "omnicultural mean score" for each syndrome. This then allowed him to rank the total scores of the 24 individual countries or societies and divide them into those whose mean score was one or more standard deviation above or below the omnicultural mean score with a third group containing ratings of societies within one SD of the omnicultural mean. That permitted him to assess to what extent, e.g., gender or SES differences determined the ratings of cases above the 96th percentile, suggesting clinical difficulties in respective groups of societies. It also facilitated the presentation of comparative data on groups of societies whose children fell in the suggested clinical range based on much lower or higher scores than was suggested by the respective omnicultural mean. Achenbach calls this approach the "bottom-up" strategy and sees it as one way to learn more about cultural values in specific cultures and their impact on psychopathology. It appears reasonable to make use of Achenbach's empirical data when considering the long-term association between similarly "abnormal" behaviors in preschoolers of immigrant families from, e.g., Vietnam and South America and their validity in predicting later psychiatric disorders within the North American context.

Finally, it is important to point out that neither the PAPA, nor the suggested modified diagnostic criteria of DSM-IV-TR nor screening instruments developed by Achenbach and Goodman claim to be relevant for classifying children younger than 24 months because of the developmental plasticity of this period of life. However, a number of clinicians working with infants and their caregivers felt that a different approach to diagnosing infants would overcome this challenge and developed the DC: 0–3 classification system in 1994, followed by an updated version DC: 0-3R (2005). This classification was created especially for the diagnosis of infants and toddlers, avoiding the pitfalls inherent in DSM-IV. Unfortunately, the system uses criteria that are primarily operationalized on clinical experience because of a missing research base. This led to some new diagnostic categories such as regulatory disorders and parent–child relationship disorders which have been helpful to some clinicians but do not reflect valid precursors for distinct later clinical entities.

Treatment

As has been documented in the previous sections, data from neuroscience, developmental psychopathology, and culturally relevant situational factors suggest that treating anxiety disorders in young children would be useful. All these lines of investigation demonstrate the relative malleability of anxious behaviors in early childhood and stress that potentially rapid remediation can be

expected. There is also evidence that it is possible to obtain an accurate and valid early diagnosis of a range of conditions such as SAD, OCD, specific phobias, general anxiety disorder (GAD), and PTSD. While parental counseling and play therapy have been the primary clinical interventions for young children despite limited supporting empirical data, the efficacy of CBT in helping older children with anxiety disorder and preliminary data in younger children, e.g., Freeman et al. (2008) has raised the question whether treatments using cognitive behavioral strategies can be helpful in this population. It has long been taken for granted that preschool-aged children function at a concrete, egocentric, prelogical, or preoperational cognitive level whereas CBT is based on a rationalist paradigm that expects the child to use a concrete operational way of thinking. Specifically, CBT requires patients to have a certain linguistic ability, self-reflection, perspective taking abilities, and an understanding of causality in order to recognize cognitive threat biases. According to Piaget, these qualities develop only after age 8 (Grave & Blissett, 2004) which would therefore exclude CBT as a valid treatment option for such young children.

However, these assumptions warrant revision. Some recent studies suggest that Piaget may have underestimated the cognitive competence of preoperational children, since it appears that by using familiar contextual information they can indeed understand causality and engage in hypothetical thinking (Meadows, 1993). According to Robinson and Beck (2000), preschoolers can also engage in hypothetical thinking of the future but not the past. Moreover, they prefer therapeutic strategies that are active, concrete, and outward focused (Harter, 1988). In practice, this means that young children can fight distorted cognitions quite readily if they are given an age appropriate narrative that is forward looking as has been so successfully demonstrated by March and Mulle (1998) in their treatment of OCD where children are encouraged to "run OCD off my land." Yet standard textbooks and researchers continue to ignore these findings and the clinical efficacy of cognitive behavioral treatment approaches for preschool-aged children is still to be properly acknowledged. For example, there are as yet no follow up reports on the outcome of preschool-aged anxious children since authors studying the long-term outcome of child samples had not included subjects below the age of 8 years (Kendall, Safford, Flannery-Schroeder, & Webb, 2004). This is especially regrettable as Saavedra, Silverman, Morgan-Lopez, and Kurtines (2010) in a recent report indicated that a CBT-based program of 10–12 weeks for 106 children aged 6–16 years ($M = 9.64$ years) at the time of intake had beneficial effects 9–13 years later when the children were between 16 and 26 years old ($M = 19.4$ years). The children had a wide range of initial diagnoses, such as agora and social phobias as well as specific phobias, and separation and GADs. Many had a comorbid second anxiety disorder but also ADHD, and all were randomized into either a group- or individual-based cognitive behavioral treatment group. The authors were able to locate 82 of the 106 initial sample (77%) and 67 finally participated in the follow-up study. A surprisingly high number of adolescents and adults did not meet criteria for any DSM-IV anxiety disorder (86.5%) and for DSM-IV major depression (91%) anymore. There was no difference in the outcome between the individually and group treated children. Unfortunately, the authors do not provide separate data for the children who were less than 8 years old when they were diagnosed and treated, making it impossible to see whether they responded differently from the rest of the sample. The study does suggest, however, that even young school-aged anxious children can benefit from CBT.

In the last 2 years three papers have been published that focus specifically on treatment outcome in preschool samples (Minde, Roy, Bezonsky, & Hashemi, 2010; Monga, Young, & Owens, 2009; and Scheeringa, Weems, Cohen, Amaya-Jackson, & Guthrie, 2011). As this may mark the beginning of evidence-based research into the possibilities of treating young anxious children using CBT, they will be discussed in more detail. Scheeringa et al. suggested specific modifications of the DSM-IV criteria defining PTSD (Scheeringa et al., 2003), and recently published the first randomly designed CBT-based

treatment study of young children with PTSD who were recruited through three battered women's programs in the New Orleans metropolitan area (Scheeringa et al., 2011). Their sample consisted of 75 children aged 36–83 months ($M = 63.5$ months). 64 children were randomized, 40 of them received "immediate treatment," and 24 were placed on the waiting list (WL). At baseline, 18 had a sufficient number of symptoms to satisfy the regular DSM-IV diagnostic criteria (24.0%) and 54 (72%) satisfied the modified PTSD criteria. Overall diagnoses were derived from five modules of the PAPA described by Egger et al. (2006). In addition to the two versions of PTSD, they included the PAPA version of MDD, SAD, oppositional defiant disorder (ODD), and ADHD. Treatment consisted of 12 highly structured individual sessions, using techniques adapted from a manual used with sexually abused preschool children (Cohen & Mannarino, 1996). The primary maternal caretakers were in the room with the children at all times, and all sessions included significant time for psychoeducation of the mothers. The therapists also rated the cognitive understanding the children had of specific concepts associated with PTSD and the aims of the treatment. They reported that at session 1, none of the 8 children aged 3 understood the concept of posttraumatic stress disorder from verbal discussion but 62.5% understood it from cartoons. However, more than half of the 4 year olds (7 of 13), understood it from verbal discussion and all of them from cartoons at session 1. Almost all the 5- and 6-year-old children understood the concept using either way of presentation. By session 8 almost all children of the total sample could differentiate moderate from worst anxiety provoking stimuli (92.6%) and self-ratings of their anxiety level could be obtained during sessions 6–10. Overall, the children were judged to understand and complete 83.5% of the possible 1,793 items rated in the 388 treatment sessions performed during the study. The 3 year olds had difficulties in gradations of emotion states but were successful at doing exposure exercises. They were also successful in doing homework assignments although they had difficulties verbalizing their understanding to their therapists. The changes of behaviors between pre- and posttreatment assessments had a large effect size for PTSD (1.01; $p < 0.0001$) and substantial ones for MDD, SAD, and ODD (from 0.72 to 0.92, $p < 0.0005$). Moreover, at a 6-month follow-up evaluation, PTSD symptoms as well as MDD-, SAD-, and ODD-associated difficulties were very significantly improved relative to baseline values ($p < 0.0005$). Not surprisingly, there was no change in their rate of ADHD.

These are very impressive results, especially since they were based on work with a rather disadvantaged population. The mothers' relatively low level of education, together with the interruption of the study by Hurricane Katrina, may explain why out of the 40 immediately treated and 24 waitlisted families who were treated after the initial cohort had terminated the study, only 26 children completed all 12 treatment sessions and not more than 19 were available for the 6-month follow-up session.

A group program study with a patient population of 5–7-year-old children attending a university-based anxiety clinic was reported by Monga et al. (2009). In this pilot study, 32 children were enrolled in a 12-week manualized CBT group program and a subset of 11 children were placed on a waitlist for an average of 3.5 months as a control. Groups consisted of 5–8 children and parents were mostly seen separately during the times of the group meetings. The children had various anxiety disorders, including social anxiety disorder, GAD, and selective mutism. This is of interest as social anxiety disorder is considered rare in preschoolers and selectively mute children usually require more than 12 sessions of treatment. Yet 43.8% of the children did not meet criteria for any Axis-I anxiety disorder at the end of the group treatment program. It is not clear from the data presented whether the children with SM were actually able to talk to nonfamily members after the treatment or whether changes were related only to their level of anxiety. The primary novelty of this paper is that Monga et al. developed a treatment manual for this age group which was not just a modification of other programs but used stories and games intrinsically appealing to this age group. The manual was tested on the

initially treated subgroup and thought to be appropriate for the children. In addition, mothers were taught relaxation exercises and desensitization strategies in the hope that they would then teach these techniques to their children. It is possible to imagine that some children may prefer a group treatment format to individual sessions as seeing others with similar difficulties would decrease their emotional isolation. On the other hand, few practitioners have the resources to deal with the organizational challenges associated with running groups for anxious preschool-aged children and their parents.

Finally, Minde et al. (2010) published outcome data on 37 children aged 37–89 months (average 71 months) who had attended a university-based child psychiatry anxiety specialty clinic and were treated with CBT. They were the youngest subgroup of 250 children who were consecutively referred to this clinic by their respective physicians during a 4-year period. The clinic accepted only children younger than 12 years and the sample presented all the children who were younger than 8 years at the time of referral except those with a primary diagnosis of selective mutism because this condition is inappropriate for a short-term CBT-based treatment program. All children were English speaking and attended some type of daycare or preschool program at least on a part time basis. The majority came from middle class families. All had been symptomatic for more than 6 months with 20 being considered clinically anxious for more than 1 year. Eleven had been seen by community-based psychologists or counselors in the past and their parents had received advice about appropriate management techniques, but that had not been successful. The waiting period between the initial phone call to the clinic and the first scheduled appointment with the director of the clinic and his team was between 10 and 12 weeks, further extending the period between onset of the symptoms and the treatment. While the team participating in the initial assessment usually consisted of medical students, psychiatric residents, master level students in art and drama therapy, a social worker, and a child therapist, the primary author provided all treatment sessions to the children and their parents. Before the assessment, the parents and the child's teacher were requested to fill in the Strengths and Difficulties Questionnaire (SDQ) (Goodman, 1997) and fill in a standard form asking for a set of family and developmental data.

The initial assessment included all family members and led to a diagnosis based on DSM-IV-TR criteria, SDQ ratings, interview findings, and a consensus rating on the Children's Global Assessment Scale (CGAS) score (Shaffer et al., 1983) by the primary investigator, social worker, and psychiatric resident. Nineteen percent of the children had only one anxiety disorder (SAD, GAD, OCD, or phobias), 43% had more than 1 anxiety disorder, 27% showed various comorbidities (ADHD, ADD, and ODD), and 11% had an associated delay in their language development. The treatment offered to the children included a modified CBT model, consisting of exposure only or exposure and response prevention, and learning how to "talk back to the brain," as well as psycho-education for the parents. In the treatment, special emphasis was placed on concrete ways to overcome fears and emotional vulnerabilities, using games, drawings, or stories to keep the children interested. One or both of their parents came in for the last 20 min of each session, allowing the parents to report on the progress made during the past week and help in planning the subsequent "home work." If no improvement was observed after 4–5 weeks, Fluoxetine was added, using the liquid form of 4–6 mg/day. Ten children (27%) required this additional help. There was one follow-up session 4–6 weeks after the last regular appointment when SDQ and CGAS scores were again obtained. No family discontinued the treatment of their children prematurely.

Results revealed that almost 50% of available parents qualified for one or more psychiatric diagnoses, in 60% consisting of anxiety and/or depression. SDQ ratings by parents and teachers as well as CGAS ratings showed statistically significant improvements after an average of 8.3 treatment sessions. The treatment effect was not associated with the age of the children. Interestingly, of nine potentially high risk back-

ground variables, only the presence of a past parental psychiatric diagnosis significantly predicted a positive treatment outcome ($r = 0.64$), as if the personal experience of living with mental health problems had made these parents into especially committed partners in the therapeutic process. Treatment had no effect on symptoms of ADHD, confirming findings of Scheeringa and his group (Scheeringa et al., 2011). Parent ratings on the SDQ also revealed a significant decrease in the burden of caring for their children after the treatment. The 10 children (27%) who had received medication in conjunction with CBT showed significantly higher baseline sum SDQ scores by both parents and teachers and significantly lower CGAS ratings by the psychiatrist. The medicated children also received significantly more treatment sessions.

In summary, this study shows that offering CBT-based interventions within the context of a regular outpatient clinic is well accepted by families of preschool-aged children and can be helpful in decreasing the anxiety of the children and improve the overall quality of life for their families following relatively few sessions. The study also confirms that many young anxious children already show comorbid conditions such as an ODD or an additional anxiety disorder. As many of their parents appeared to have suffered from anxiety now or in the past, they were ready to become solid partners in the clinical work when invited to do so.

The purely clinical venue of the study also explains some of the study's shortcomings. For example, there was no control group of untreated or differently treated children and we also did not use a fully manualized treatment format as we felt that this would not allow us the necessary flexibility in establishing a therapeutic alliance with these young children. Our decision to add a psychopharmacological component for children not responding to cognitive strategies presents another methodological challenge although authors studying older children have reported this to be the best strategy for dealing with serious manifestations of anxiety (Walkup et al., 2008) within a clinical setting. In fact, many parents were reluctant to accept medication for their children although they were grateful for the additional relief that provided in most cases. Another problem of this study is that all children and their families were treated by the primary author who was also responsible for obtaining the follow-up information and scoring the CGAS ratings. However, the rapid turnover of team members such as the psychiatric residents and the groups of other students made it practically impossible to create a group of seasoned clinical practitioners for these young children. This makes it essential to replicate this study on larger samples of children of more diverse backgrounds, and using more than one experienced therapist.

Two Cases

Case 1

Sara was 42 months old when she was referred by her paediatrician because of severe anxiety combined/associated with controlling and wilful behaviors for more than 2 years. She had an older sister aged 10. Her parents, both in their 40s, were from Italian immigrant families; their respective parents were factory or restaurant workers who had little time for them as children. Sara's mother, who was also worried about her own mother, had not worked outside the house since the birth of her first child. Sara's anxieties had shown themselves in various ways since birth. She had always been afraid to be separated from her mother and, even when with her, had never been able to visit anyone except her maternal grandmother. While she reached her milestones at a normal age, she had always been an extremely sensitive child, e.g., during her first 2 years, she would at times vomit when she was exposed to loud noises; she would hide in the basement when her sister invited another child for a play date; and had never had a birthday party because she could not tolerate sharing her mom's attention with anybody else. She ate only very few foods and would just sit at the table with her eyes closed and eat nothing if a different food was offered. She had also never been able to fall asleep alone. In fact, she slept on her mother's abdomen for the first 18 months of her life. At the time of the intake she would fall asleep around

8.30 PM with father being in bed with her. She woke up three or four times a night always demanding one of her parents to be with her. She would also lie down on the sidewalk and cry when her father attempted to bring her to a park just across the street.

Mother's pregnancy with Sara was uneventful. However, she had lost two babies during previous pregnancies because of an undiagnosed clotting disorder and was extremely worried that she would lose Sara as well and therefore spent almost 6 months in bed.

The parents scored Sara in the abnormal range on the emotional and conduct disorder and ADHD axes of the SDQ (Goodman, 2001) but felt that she had good pro-social abilities.

During our assessment interview, Sara hid behind her mother and would not look at me. I left her alone for about 1 h but then addressed her with a puppet in my hand, saying that the puppet thinks that Sara does not enjoy her worrying about so many things but does not know how to stop it. She responded by nodding. I then mentioned that I would try to help her chase these scary thoughts away and mom and dad would do so as well. We met 12 times during the subsequent 7 months where we played with farm animals that were scared but overcame their fears and were proud of themselves. The parents were asked to institute very gradual changes in her daily routine. For example, father moved from lying down with her at night to sitting on the bed, then to a chair besides the bed, etc.

Sara started drawing more positive pictures and after six sessions agreed to try a daycare program once per week for 3 h. While she did not eat and drink anything at the daycare and did not use the toilet, she allowed her mother to leave her there after 4 weeks. We all had ice cream during our last session and she sent me a Christmas card 6 months later with a photo of herself smiling. Twelve months later, she slept and ate well, had two friends and was looking forward to enter a regular kindergarten program.

Case 2

Marian, aged 44 months, the second born of a nonidentical pair of twins, was referred by her paediatrician because she had developed obsessive behavior patterns during the preceding 8 months. For example, she had very elaborate bedtime rituals where she required her bed sheets to be precisely positioned across her chest. She could not tolerate to have her toes covered by her blanket and would scream if her parents did not do it right. In her daycare, she would line up the shoes of all the attending children before joining them in play. She was also very particular that no piece of clothing was ever exchanged with her twin sister. She also would not permit anyone to touch any of her new toys or pieces of clothing. However, she slept and ate well, enjoyed her daycare and was a popular child overall.

Marian's mother comes from a family without psychiatric difficulties and works at a medical department in a local hospital. Because of her experiences at work and personal acquaintance with an autistic youngster she became very worried about Marian's symptoms. In addition, Marian's father, an IT specialist, washed his hands more than ten times per day. He also demanded that the house was spotless and parked his car at least 15 m away from the next car at the local shopping mall to prevent potential scratches from other drivers. He defended these peculiarities forcefully. Marian, during the initial interview, came across as a curious youngster who showed her love for her family by insisting that her parents also each got a cookie when I offered one to her. However, she was not interested in changing any of her habits as she felt well accepted by everybody. Marian's birth weight had been 3 lbs 19 oz and both twins had remained in the hospital for 3 weeks after birth. Both slept through the night by 3 months and reached their milestones at the expected ages, but were diagnosed to have prematurely closed sutures of their skull, requiring a corrective operation at 13 months. Both parents also rated Marian within the normal range of all 5 axes of the SDQ.

We discussed to what extent Marian's rituals were truly necessary and scheduled another appointment 4 weeks later. At that time, all her rituals had disappeared and Marian had mentioned to her parents that these habits were "not important anymore."

These cases document that anxieties and obsessional behavior patterns, as shown by Marian, can be part of normal development during the preschool period. However, the clinical features in Sara's case had a far more pervasive flavor, significantly impacted her overall social development and impaired her relationship with her family and peer group. Her profound need to control others is commonly seen in anxious children and best understood as the child's attempts to gain control over his or her anxieties. Psychological treatment requires a true partnership between therapist, child, and family and is most effective when the therapist respects the child's challenges and gently assures him or her that one can change and talk back to the brain when it tries to convince us about unnecessary fears and worries.

Prevention

The final part of this chapter deals with the possibility to create early prevention and intervention programs for anxious children and their families. One can argue that any clinical intervention involving young children with well-documented anxiety problems could be interpreted as representing a "secondary" type prevention since it may modify the natural course of the illness. Thus in the report by Minde et al. (2010), mentioned previously, the great majority of patients had displayed clinically meaningful anxiety symptoms for more than one year and their parents or other direct family members had often shown a lifelong history of battling anxious thoughts and behaviors. In fact, many parents did not want their children to suffer for 30 or more years from anxiety as they had done and they saw our clinic as an opportunity to prevent this fateful intergenerational continuity. Moreover, only 2 of our initial cohort of 37 children have come back for additional help during the subsequent 3–7 years, and their "relapse" required no more than two booster sessions to regain control. However gratifying, this does not provide any proof for the longer term prevention of anxiety problems in our sample as there was no randomly selected control group and no regularly scheduled follow-up assessments of all children.

Nevertheless, there have been increasing efforts and some interesting results reported for interventions provided to selected groups of children under the age of 3 years. For example, Wallace and Rogers (2010) recently summarized the implications of intervening in infancy for children with autism spectrum disorder (ASD) and those born with developmental delays and very prematurely. These groups can be reliably diagnosed at 18 months and interventions can start early. The mean effect size (SE) of the 12 type 1 and 2 studies, i.e., reporting on randomly prospectively designed trials, blind assessments, adequate samples and treatment manuals, encompassing families who had children with ASD, was 0.56. The 19 studies examining interventions for premature infants had a mean effect size of 0.44 and the four studies with infants at risk for intellectual disability had a mean SE of 1.26. These very significant changes confirm the potential plasticity of early abnormal child behaviors. An additional interesting aspect of these studies is that the most efficacious interventions for these three groups of children used a combination of four specific intervention procedures. These were: (1) active parent involvement in the intervention, including ongoing parent coaching that focused on parental responsivity and sensitivity to child cues and on teaching families to increasingly provide the infant interventions, (2) individualizing each infant's developmental profile and address it accordingly, (3) focusing on a broad rather than narrow range of learning targets, and (4) begin interventions as soon as the risk is detected and do so intensively and systematically for an extended time. Some of these intervention procedures are the same that have been found effective by clinical investigators in their work with anxious preschoolers. Thus all stressed the crucial role parents play in facilitating and supporting their children's growing cognitive and emotional understanding of their behavior. They also support early treatment and the need to address broad learning targets. It is not clear whether strictly manualized treatment programs, especially when employed with groups

of children and parents, meet the anxious child's individual needs and how long such interventions have to be to assure the best possible outcome.

There are two interesting published group intervention programs that focus directly on prevention of anxiety disorders in young children. One of them, designed by Rapee, Kennedy, Ingram, Edwards, and Sweeney (2010), is based on the observation by Kagan (1994) that young children who show a temperamental profile dominated by behavioral inhibition in infancy tend to remain shy and anxious over time and frequently develop an anxiety disorder in later childhood. This relative stability of anxious symptomatology was chosen by the authors as a worthwhile target for an early intervention. They sent special screening packets to more than 5,600 families of 3-year-old children attending 95 preschools in Sydney, Australia and received 1,720 responses. A total of 146 children from this group were selected because they scored high on withdrawal on a temperament questionnaire (approximately 1.15 standard deviations above the mean). The children also passed a laboratory assessment to elicit shy and inhibited behaviors and subsequently were randomly allocated to either a parent intervention group or a monitor group of 73 each. Treatment consisted of six 90-min group sessions with the parents discussing the nature of anxiety, principles of parent management techniques, highlighting the effects of overprotection. Later sessions dealt with the application of exposure techniques and of cognitive restructuring. There was no direct therapeutic contact with the children. Diagnostic interviews and questionnaire measures were repeated at 12, 24, and 36 months. Results showed a significant group-by-time effect in the number of anxiety disorder diagnoses from baseline to 36 months ($p = 0.008$) but none from baseline to 12 and 24 months. The same was true when measuring the average severity of anxiety disorders. Children in the parent education group also reported themselves to be less anxious at age 6 on an anxiety scale. The investigators suggest that the group meetings altered the trajectory of anxiety in these children because of the widening gap between the children in the treatment and control groups over time. Moreover, all children in both the intervention and monitoring group showed a reduction of their temperamental inhibition scores between age of 3 and 6 years, suggesting that the children's shy predisposition had remained equal in both groups, but had not led to anxiety in those whose mothers had attended the treatment group.

These follow-up results are very encouraging because they are associated with a brief parent-based intervention 3 years earlier and are well documented by validated instruments. This intervention, however, benefitted a specific subgroup of young children that can be identified early in life, much as children with ASD or other developmental disorders, and needs to be replicated with other populations.

More evidence that community-based group intervention programs targeting parents work comes from another recent Australian study where Havighurst, Wilson, Harley, Prior, and Kehoe (2010) reported on a program called "Tuning in to Kids" that aims to improve emotion socialization practices in preschool children. They randomized 216 parents of a target child aged 46–68 months and offered them 6 weekly 2 h sessions. One hundred and ninety parents finished the study. The program, based on a structured manual (Havighurst & Harley, 2007) encouraged changes in parenting beliefs and behaviors while increasing the emotional connection between parent and child. Parents were encouraged to become aware of their own as well as their children's emotions and how to empathize with them. One session dealt specifically with anxiety and problem solving. Parent and child ratings were obtained before and after parental group training and at 6-month follow-up. Parents in the treatment group reported being less dismissive, more emotion coaching and empathic at follow-up, whereas control parents did not change. Children whose parents were in the treatment group showed better emotional knowledge, less intensity in responding to stresses and a significant reduction in behavior problems reported by parents and teachers in comparison to their control peers.

While the assessment instruments used in this study did not allow to arrive at DSM-IV-based psychiatric diagnoses of the children, and there

were no follow-up assessments beyond 6 months, one could nevertheless see this study, together with the one by Rapee et al., as a promising addition to available parenting programs. In future work, it would also be of interest to explore whether a program like "Tune in to Kids," when provided to early childhood educators, would improve their mentalizing ability and assist them in helping preschool-aged children with or without validated anxiety disorders.

Summary

The present chapter attempts to summarize our present understanding of anxiety disorders in young children within the context of developmental psychopathology and neuroscience. Data support an individual predisposition to threat sensitivity in very young children that is open to epigenetic modulation by environmental events but shows significant stability after the age of 2 years. They also suggest the possible transformation of anxiety into depressive symptoms during adolescence but document the powerful impact parental care as well as other interpersonal experiences can have on the clinical presentations of anxiety disorders, especially in young children. Treatments addressing the cognitive and emotional distortions typical for anxiety disorders have been found to be helpful for school-aged children for some time but were considered unsuitable for preschoolers because of their alleged immature cognitive structures. Recent data have challenged this assumption and this chapter provides examples of creative treatment approaches by clinician researchers that have modified seriously incapacitating PTSD and other anxiety symptoms in preschool-aged children by using CBT-based treatment modalities. These authors educated the children's parents and provided the children with age appropriate narratives for fighting their unnecessary fears and concerns. The ready acceptance by parents of this form of treatment will undoubtedly lead to further refinements in evidence-based cognitive behavioral treatment programs of young children and lessen the burden of anxiety-based disorders for both the children and their families.

The ultimate aim of any treatment is the long-term prevention of an anxiety disorder or at least a modification of its course. Here again, the very recent literature presents hopeful signs. A comparatively brief exposure to well designed education programs appears to allow parents to significantly modify their interactions with their children and to bring about behavioral change of up to 3 years. One would hope that similar educational efforts could be directed at teachers of preschoolers in the future so that a wider range of anxious children could face life with hope and self-confidence.

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Treating Obsessive-Compulsive Disorder in the Very Young Child

8

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Obsessive-compulsive disorder (OCD) is a complex psychiatric condition. The empirical literature has documented at least two, age-related subtypes of the disorder, child- and adult-onset. As may be inferred, child-onset OCD is characterized by an onset of OCD symptoms prior to 18 years of age. Compared to adult-onset OCD, children with OCD are more likely to have at least one first-degree relative with the disorder (Nestadt et al., 2000). In combination with a growing body of corroborating evidence (e.g., the role of parental accommodation of a child's symptoms), this data suggests that understanding the family environment may be important for advancing science's knowledge regarding the pathogenesis and treatment of child-onset OCD. Even within the child-onset subtype, however, there are important developmental differences to consider. Unfortunately, children under 7 years of age are often left out of many clinical and treatment outcome studies (Barrett, Healy-Farrell, & March, 2004; Piacentini, Bergman, Jacobs, McCracken, & Kretchman, 2002; Storch, Geffken, & Merlo, 2007; Storch et al., 2004). Failure to include these younger children in empirical research may inaccurately imply that the presentation of OCD is identical from early childhood to late adolescence and adulthood. Our group has begun to more rigorously examine the experience of and efficacious psychosocial interventions for children with OCD who fall within the very young end of this developmental spectrum (e.g., 4–8 years of age). The aim of this chapter is to provide an overview regarding the nature of OCD in very young children, factors that may contribute to the disorder's complexity at this age, and treatment approaches to address these factors. We conclude with a case study designed to provide an example of the complexities surrounding the assessment and treatment of OCD during early childhood and areas for future research.

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Age Appropriate vs. Potentially Disordered Behavior

A common theme throughout this chapter is the importance of understanding developmental consideration for very young children with OCD. In this vein, it is equally important to distinguish between what may be developmentally appropriate vs. potentially disordered behavior. For example, young children engage in a variety of superstitious games (e.g., crossing one's finger when telling a lie) and exhibit repetitive themes during solitary play (e.g., only using the blue blocks when building a tower; Francis & Gragg, 1996) yet very young children with and without OCD exhibit these behaviors. A useful approach for differentiating pathological (e.g., diagnostic

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of OCD) from "normal" child behavior is the degree to which modification of the child's routine(s) is possible. For example, most young children will become distressed if told they must use blue and red blocks yet it is the degree of distress exhibited that may best differentiate behavior as age appropriate or disordered. This will be important to keep in mind throughout the remainder of this chapter.

Nature of the Problem

OCD is characterized by intrusive thoughts, ideas or images and/or repetitive, intentional rituals that cause marked distress and/or interference in one's life (APA, 2000). The disorder affects 1.5–2.2 million children in the United States alone (Valleni-Basile et al., 1995; Zohar, 1999). Childhood OCD is also associated with significant impairment in day-to-day functioning (Adams, Waas, March, & Smith, 1994; Cooper, 1996; Leonard, Lenane, & Swedo, 1993; Piacentini, Bergman, Keller, & McCracken, 2003; Toro, Cervera, Osejo, & Salamero, 1992). The majority (e.g., 75–84%) of children with OCD are also frequently diagnosed with comorbid psychiatric conditions (Geller, 2006). Despite this growing empirical evidence, childhood OCD is still under-diagnosed and under-treated. Epidemiological findings indicate that less than 25%of a community sample of adolescents with OCD received any mental health services, and none received treatment specifically for OCD (Flament et al., 1988). These findings clearly suggest that childhood OCD constitutes a significant public health concern. Compared to disorders such as major depression, schizophrenia, or even many other anxiety disorders (e.g., generalized anxiety disorder, social phobia), scant research is available regarding the pathogenesis and treatment of OCD. This is particularly true of very young children with the disorder.

OCD has been documented in children as young as 3 years old and demonstrates an average age of onset at approximately 10 years (Hollingsworth, Tanguay, Grossman, & Pabst, 1980; Swedo, Rapoport, Leonard, Lenane, & Cheslow, 1989). Clinical researchers typically parse OCD into two, age-related subtypes (e.g., child- vs. adult-onset). Evidence suggests though that prepubertal (hereafter referred to as early childhood), pubertal (adolescent), and adult-onset OCD may be useful distinctions. Despite these distinctions, common core symptoms of OCD are observed across the life span (Rettew, Swedo, Leonard, Lenane, & Rapoport, 1992) suggesting that use of the same general diagnostic nomenclature from early childhood to late adulthood is useful. With that said, unique features of early childhood OCD exist including the gender distribution of those affected, rates of comorbidity, and symptom expression.

Gender distribution. It has generally been thought that children demonstrating a younger age at OCD onset are more likely to be male. (Geller, 2006) This gender difference is reversed in adults. (Craske, 2003) Many studies note a male predominance in children (3:2) with the gender distribution becoming more equal in adolescence (Geller et al., 1998; Swedo et al., 1989). However, Garcia et al. (2009) recently found that 60.3% ($n=35$) of their sample of 4–8 years olds with OCD was female. These recent findings suggest the need for further research to better elucidate the gender distribution in children with OCD and perhaps whether early childhood OCD in fact represents another unique subtype of the disorder.

Rates of comorbidity. Among very young children with OCD, comorbid diagnoses such as tic disorders, ADHD, and learning disabilities (Geller, Biederman, Griffin, Jones, & Lefkowitz, 1996; Pauls, Alsobrook, Goodman, Rasmussen, & Leckman, 1995) are more common. For example, family studies have established significantly elevated rates of comorbidity between OCD and tic disorders (Pauls & Leckman, 1986). This finding is particularly strong for children with onset of OCD before age 9 years (Pauls et al., 1995). In addition, a recent study found that 20.6%of children with OCD between 4 and 8 years of age met diagnostic criteria for a tic disorder (Garcia et al., 2009). Further examination of these data revealed similarly high rates of both attention-deficit hyperactivity disorder (22.4%) and generalized anxiety disorder (20.7%) in this sample. Perhaps

not surprisingly, these authors also found very low rates of both major depression (1.7%) and dysthymia (1.7%). Due to the limited literature in this area additional research is necessary to replicate and extend these findings.

Symptom expression. Child-onset OCD cases have been identified as having an atypical pattern of symptom expression (Geller et al., 1996, 1998). In early childhood OCD, compulsions without clearly defined obsessions are common. In fact, the compulsive behaviors themselves may be different than those observed in adolescents or adults (Swedo et al., 1989). For example, Garcia et al. (2009) found that 58% ($n=28$) of very young children with OCD exhibited aggressive or catastrophic (e.g., something bad happening to a parent if the child does not complete his/her ritual) obsessions while 68% ($n=34$) and 60% ($n=30$) exhibited checking (e.g., 68%, $n=34$) and/or rituals involving other person (60%, $n=30$). These results also highlight the frequency with which younger children often involve family members in their ritualistic behavior, often in the form of reassurance seeking (verbal checking; Flessner et al., 2009). This pattern of interaction is often referred to as family accommodation and is described in greater detail below (*Family Involvement*).

Many differences in the symptom picture between very young children with OCD and adolescents or adults are likely due to developmental factors. Early childhood cognitive development may make it less likely that obsessional thoughts are prominent features in the symptom picture. Further, children are more embedded in the family context contributing to family involvement in the disorder. As a result, both of these factors serve as key contributors to case complexity in early childhood OCD.

Factors That Contribute to Complexity

Few, if any, psychiatric conditions are homogeneous. Complications exist with the treatment of any form of psychopathology because it is extremely rare for all patients to exhibit identical backgrounds or symptom presentations. Early childhood OCD is no different. Important developmental considerations and the role of the family in their child's OCD-related symptoms are particularly important factors contributing to the complexity of this form of the disorder. Therefore, we provide a brief review of these two factors and the impact they may have on the assessment and treatment of very young children with this disorder.

Developmental considerations. From a developmental perspective, very young children may not yet posses the cognitive skills necessary to adequately describe obsessions or worries preceding their compulsive behavior. As a result, the assessment of early childhood OCD can be quite difficult. Adding to this trouble is the potential difficulty differentiating OCD-related concerns from (1) developmentally appropriate behavior (e.g., rigid following of rules associated with a favorite game); (2) a comorbid psychiatric condition marked by repetitive behaviors (e.g., tic disorders); and (3) repetitive behaviors characteristic of other psychiatric conditions entirely (e.g., stereotypies common to autism spectrum disorders). Differentiating these diagnoses may be markedly more challenging among very young children with OCD because of the child's difficulty in elucidating preceding thoughts or feelings or feared consequences associated with not engaging in their ritualized behavior. Because these disparate conceptualizations require different therapeutic interventions, the developmentally appropriate assessment of very young children with OCD-like behavior(s) is of the utmost importance.

The Children's Yale-Brown Obsessive-Compulsive Scale (CY-BOCS; Scahill et al., 1997) is largely viewed as the "gold standard" instrument for assessing OCD symptoms among adolescents. The methods and procedures validated for use in the assessment of older youths with OCD may not be as well suited for early childhood OCD. For example, younger children are less able to adequately describe their anxiety symptoms. Therefore, the developmentally appropriate assessment of younger children will place a greater reliance on parent(s)-report of symptoms. Descriptions of specific symptom dimensions (e.g., excessive concern regarding urine, feces, and saliva) must also be tailored to the child's developmental level (i.e., worry or grossed out by

pee, poop, or spit). Because very young children may be less likely to report obsessions or intrusive thoughts, it may be advantageous to assess for compulsions first. In the absence of obsessions, CY-BOCS total score may not be reflective of overall symptom severity. With the employment of these subtle modifications, the CY-BOCS has demonstrated adequate psychometric properties for the assessment of OCD symptoms in children 4–8 years of age (Freeman, Flessner, & Garcia, 2011). As is illustrated below (see section "Case Example: Aaron"), use of the CY-BOCS in combination with additional data designed to parse out potential differential diagnoses (e.g., age appropriate behavior, autism spectrum disorder, tics) can help refine the conceptualization of a child's presenting concerns and guide the selection of appropriate therapeutic intervention(s).

Family involvement. Increasing attention has been paid to the role of family factors in the development of psychopathology, and specifically to OCD; as well as to literature supporting the role of the family in understanding and treating childhood psychopathology. It is commonly accepted that OCD can result in a marked, negative effect on both the patient and their family (Waters & Barrett, 2000). Some researchers have suggested that, within the context of the family, OCD demonstrates a bidirectional relationship. That is, families have an affect on and are affected by OCD. Within the context of childhood OCD, this suggests that the interactions between the parent and child are of great importance (March, 1995). Young children are embedded in a family context in a way that is meaningfully different from that of adults. Parents are more likely to play an active role in young children's rituals (e.g., physically assisting with washing or checking; Garcia et al., 2009; Lenane, 1989). Therefore, the family's participation in their child's OCD-related rituals (e.g., accommodation) has received growing empirical investigation in recent years.

The term accommodation is most often operationally defined as the participation of family member(s) in the ritual(s) of a child with OCD. In practice, accommodation may take several forms, including aiding in completion of the ritual, facilitating avoidance of situations, events, or persons, or any other activity the family may perform in response to the individual's OCD symptoms (Amir, Freshman, & Foa, 2000; Calvocoressi et al., 1995, 1999; Storch, Geffken, Merlo, Jacob, et al., 2007). Recent evidence suggests that as many as 88%of parents may engage in at least mild accommodation of their child's OCD symptoms (Merlo, Lehmkuhl, Geffken, & Storch, 2009). Independent investigations have found similarly high rates of parental accommodation and suggest that accommodation is ubiquitous across the families of children with OCD (Storch et al., 2007; Peris et al., 2008).

Patterns of family behavior (e.g., accommodation), parent–child interactions, and parents' own interpretations of potentially anxiety provoking stimuli, are likely to affect their young children with OCD and impact treatment. Merlo et al. (2009) recently found that changes in parental accommodation (e.g., parents becoming less involved in their child's rituals) predicted treatment response to cognitive-behavioral therapy (CBT). One important caveat to the research described above, however, should be noted. The majority of these studies have failed to specifically examine parental accommodation among very young children. Accommodation is most often studied in relation to the families of children with OCD broadly defined (e.g., patients under 18 years of age). Given important developmental differences among very young children and adolescents or adults (see *Development Consideration* above) and the findings noted previously, family involvement in treatment may be of particular importance for very young children presenting with OCD. Consequently, it is imperative that treatment approaches for these children incorporate both developmentally sensitive approaches to treatment and the family.

Treatment Approaches to Address Complexity

Cognitive Behavior Therapy has consistently demonstrated efficacy for the treatment of children with OCD (de Haan, Hoogduin, Buitelaar,

& Keijsers, 1998; Franklin et al., 1998; POTS Team, 2004; Piacentini et al., 2002), and exposure with response prevention (ERP) is viewed by most experts as representing the key ingredient to CBT for the successful treatment of OCD. As we move further down the developmental spectrum, however, different factors become increasingly important for incorporation into treatment protocols (e.g., developmental considerations, family involvement). In this section, we provide an overview of important additions, modifications, or refinements to CBT-based treatment protocols that we believe are important for enhancing the efficacy of therapeutic interventions for very young children with OCD. We conclude this section with a brief discussion of empirical evidence supporting this approach to treatment.

Developmental differences between children and adolescents have important implications for treatment (Kazdin & Weisz, 1998). The cognitive component of CBT protocols applied to the treatment of OCD, has limited its utility at best during the early childhood period. Young children do not yet posses the skills necessary to fully comprehend and benefit from cognitive therapy techniques (e.g., abstract thinking, cause and effect, understanding probability). Further, current approaches are based on individual modality of treatment. While adolescents may be able to independently attend a therapy session, understand and retain weekly assignments, and complete between session homework—all integral steps in existing treatment protocols—young children cannot. In therapy with young children, caregiver involvement is essential, as they are often required to take on a supportive or even primary role in administering treatment. Thus, the individual therapy modality is not an optimal mode of treatment delivery for this age group.

Earlier in this chapter (see *Rate of Comorbidity* above), we explained that very young children with OCD are more likely to present with comorbid tic disorders, hyperactivity and learning disabilities. From a developmental perspective, these increased rates of comorbidity must also be taken under consideration when modifying existing treatment protocols. In order for OCD treatment to be effective, parent behavioral training may also be necessary. Therefore, teaching parents basic behavior management techniques, developing behavior modification plans, and teaching parents strategies to manage their child's anxiety and distress is important for this population.

Finally, young children with OCD are more embedded in their families than older children or adolescents. The dependence of children on their caregivers makes them vulnerable to multiple influences over which they have little control. Parental mental health, marital functioning, and family functioning are just a few of the contextual factors that affect nature and severity of impairment, treatment progress, and maintenance of treatment gains for children (Kazdin, 1995; Kazdin & Weisz, 1998; Tharp, 1991; Weisz & Weiss, 1991). Further, the family unit and subsystems are also affected by the child's symptoms of OCD (Freeman et al., 2003).

The presence of a child with OCD symptoms is likely to compromise the functioning of the family unit and/or specific subsystems (e.g., parent–child, marital relationship). Therefore, it has been suggested that therapy with very young children by necessity is "de facto family context therapy" regardless of the theoretical underpinnings (Kazdin & Weisz, 1998). However, existing treatment protocols incorporate parents at a cursory level only and include a minor focus on the role of parents in effecting child behavior change. This approach to treatment may be appropriate for older children, adolescents or adults but is insufficient for very young children as they are embedded in a unique way in their family context. Focusing on OCD symptomology alone in lieu of considering involving the family system in treatment, may be insufficient for symptom amelioration and long-term improvement. Although only a first step in the process, preliminary evidence suggests that this approach to treatment may be efficacious for the treatment of early childhood OCD.

Recently, research has begun to examine the efficacy of a family-based cognitive-behavioral approach to the treatment of very young children with OCD (Freeman et al., 2008). Freeman and

colleagues recruited 42 children between 5 and 8 years of age with a primary diagnosis of OCD. Children were randomized to either family-based CBT, utilizing the approach to treatment described in the preceding paragraphs, or family-based relaxation therapy (RT). Completer analysis (e.g., participants who completed all 12 sessions of treatment) revealed that family-based CBT demonstrated a large effect size ($d=0.85$) with a significant treatment group difference. In total, 69%of children receiving CBT achieved symptom remission compared to only 20%in the RT group. Clearly, a more rigorous, randomized controlled design is necessary to replicate and strengthen these findings. However, this study provides preliminary support for a developmentally sensitive, family-based approach to the treatment of early childhood OCD. What follows below is a case example utilizing this tailored approach to the assessment and treatment of very young children with OCD.

Case Example: Aaron

Referral. Aaron is a 74-month (6 years, 2 months) old boy referred to our clinic by an area pediatrician for assessment and possible treatment. The chief complaint upon referral was in regard to "doing things until they are 'just right'" and "washing his hands all the time." Aaron's biological mother attended his first visit to our clinic. During this visit, he and his mother met with a psychologist who administered the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS; Kaufman et al., 1997), several parent-report measures, and the CY-BOCS.

Background. A brief psychosocial history revealed that Aaron has one, older brother. His parents have been divorced for approximately 2 years and share custody. Aaron is presently in first grade, and he reports that he likes school. Family history is positive for Tourette's disorder (brother, maternal uncle), OCD (brother, father), "anxiety" (mother), and depression (maternal grandmother). Because of Aaron's age and his limited ability to describe his symptoms, a significant portion of the K-SADS interview was conducted with his mother while Aaron played with toys in the therapist's office.

Assessment. Administration of the K-SADS revealed that Aaron met diagnostic criteria for OCD, Tourette's disorder (e.g., head-jerking, throat clearing, facial grimacing), and separation anxiety disorder (e.g., worries about bad things happening to his mother when separated). With regard to OCD-related symptoms, Aaron's mother reported that he is very reluctant to touch "germy" objects (e.g., anything that he knows other people, besides himself or his mother, have touched) and needs to touch objects until it feels "just right." She also reports excessive hand washing. Aaron's second major OCD symptom involved his mother. Whenever he and his mother part, including at bedtime, she kisses him, he puts his head on her arm, and then he touches/hugs her until it feels "just right." Aaron must be the last one to touch his mother. In the event that his mother touches him last, Aaron feels the need to touch her again. Administration of the CY-BOCS revealed a score of 24 indicating moderate to severe OCD symptoms.

Aaron's mother also reported that he often lines up his toys when playing and becomes very angry if someone messes up the order he has established (e.g., places toy A in front of toy B). His mother reports that this behavior has been present for "a couple of years" with little fluctuation in frequency over time. At first, it was unclear whether Aaron's behavior was age appropriate or OCD-related. Upon further evaluation, it was revealed that Aaron's proclivity towards ordering and arranging objects in this manner occurred only during playtime. He and his mother denied that Aaron engaged in this behavior in relation to other objects (e.g., toys on his bed, other objects in his room or house). Teacher-report corroborated these findings. His teacher reports that although Aaron becomes more upset than some of his peers when others "mess with" his toys, she did not believe that his behavior was significantly out of the ordinary for his age. Collectively, this evidence led the treatment team to conceptualize Aaron's behavior as age appropriate and thereby not an immediate target for intervention.

Treatment. Aaron was treatment naïve prior to the start of family-based CBT for his OCD-related symptoms. Aaron's mother attended the first two CBT sessions alone. She was provided with education about OCD as a neuropsychiatric condition, common co-occurring diagnoses, and the rationale behind family-based CBT (e.g., parental modeling, scaffolding, ERP/habituation). The therapist also began to work on a hierarchy of Aaron's symptoms for exposure exercises later in the courses of treatment. Aaron and his mother attended sessions 3–12.

Aaron was eager to begin treatment. It was also evident that he exhibited some difficulty understanding more complex elements to the treatment protocol (e.g., fear thermometer, symptom hierarchy). As a result, modifications were made. Rather than utilizing a 10-point fear thermometer, as is typical of CBT for childhood OCD, a visual analog scale was employed which allowed Aaron to report his OCD symptoms (which he and his therapist referred to as "The OCD Monster") using faces that ranged from "happy" to "worried." After talking in greater detail about The OCD Monster, the therapist, with the help of both Aaron and his mother, was able to construct several potential exposure exercises to attempt in the coming weeks. However, Aaron demonstrated difficulty ordering these exercises from easiest to hardest. To remedy this problem, the therapist decided to write each of the exposure exercises on a piece of paper. Next, he found and cut a long piece of string and placed the string perpendicular to Aaron. One at a time, the therapist read the exercise that was written on each piece and asked Aaron to place them on the string with the hardest one as far away from him as possible. Although somewhat unusual, this approach worked well and resulted in Aaron, his mother, and the therapist agreeing on a hierarchy for Aaron's OCD symptoms. A reward program was also established in which Aaron received one point every time he completed both his in-session and out-of-session homework exercises. These points could be turned in to receive agreed upon prizes throughout the course of treatment.

Sessions 4–12 centered upon the therapist working closely with both Aaron and his mother to "boss back" The OCD Monster. Aaron completed exposure exercises in session. Initially, the therapist modeled "bossing back" OCD with Aaron (e.g., both Aaron, his mother, and the therapist engaged in the weeks' exposure exercise). Over the next several sessions, Aaron slowly became more capable of "bossing back" The OCD Monster on his own. Also during these sessions, Aaron's mother was provided with education regarding important parenting behaviors that play a significant role in the maintenance and effective treatment of OCD (e.g., modeling, differential reinforcement, and scaffolding). His mother was asked to practice these parenting strategies within the context of OCD-related behaviors (e.g., asking mom to "boss back" her own anxiety). As treatment progressed, the therapist played less and less a part in developing exposure exercises. Instead, Aaron's mother was asked to develop exercises in collaboration with Aaron. The goal of this strategy was both to slowly fade the importance of the therapist for successful treatment and to "practice" what the family would do if OCD symptoms returned in the future. By session 12, Aaron had successfully reached the apex of his symptoms hierarchy. The CY-BOCS was administered 1 week following the family's final session as a "wrap-up" session. Administration of the CY-BOCS revealed a score of 9 indicating only mild symptoms of OCD.

Conclusion and Future Directions

A small but growing body of empirical research has begun to examine the assessment and treatment of very young children with OCD. Science is beginning to develop a greater understanding of potentially important differences between early childhood, adolescent, and adult-onset forms of OCD. Though room for improvement exists, evidence has begun to suggest that modifications to existing measurement strategies may yield reliable and valid assessment of the disorder (Freeman et al., 2011). Perhaps most importantly, preliminary evidence has demonstrated that a developmentally sensitive, family-based approach to early childhood OCD may be

efficacious for the treatment of very young children with the disorder. Despite all of these advances, a plethora of areas have yet to be examined or are in need of stronger empirical support.

Future directions. Several areas of future research may help to advance's science understanding regarding the complexity of early childhood OCD.

Etiology. Science has begun to obtain a greater understanding regarding the etiology of OCD yet more research is necessary. Preliminary evidence suggests that early childhood OCD may represent a useful distinction in comparison to adolescent and adult-onset OCD. However, a more reliable body of empirical research has generally supported two, age-related subtypes of the disorder. If early childhood OCD does indeed represents a distinct subtype, more research is necessary. Researchers may wish to examine genetic, neurobiological, or environmental factors that are common or unique to these distinct ages at OCD onset. It is likely that research of this nature, however, will require strong collaborations among many researchers sharing a common goal of better elucidating the pathogenesis of OCD.

Longitudinal research. Remarkably little is known about the pathogenesis of childhood OCD and, in turn, very young children with the disorder. One way in which our scientific understanding of this disorder can be greatly enriched is to examine the developmental course of OCD. Research of this nature might include the recruitment of children at-risk for the disorder, those exhibiting subclinical symptoms, or those already meeting diagnostic criteria for OCD. A critical component to such a line of research would include the recruitment of both very young children and adolescents to more adequately examine differences in the progression of the disorder (e.g., wax and waning nature of the disorder, response to environmental stressors, symptom expression and progression over time).

Treatment. We provided a brief summary of one approach to the treatment of very young children with OCD that has demonstrated preliminary efficacy. Currently, a large multisite randomized controlled trial is being conducted to more adequately examine the efficacy of family-based CBT for the treatment of early childhood OCD. Regardless of the outcome of this ongoing trial, additional research by independent investigators will be necessary to further test the benefit of family-based CBT for very young children with OCD. In addition, researchers are advised to continue examining new treatments or modified versions of existing treatment protocols making use of data from basic laboratory or clinical studies. It is only through continued scientific investigation that clinical researchers will be able to understand more regarding the etiology, maintenance, and treatment of childhood OCD.

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Treatment of Childhood Tic Disorders with Comorbid OCD

9

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Introduction

We have been charged with the task of presenting readers with a logical, empirically grounded, and clinically informed approach to the treatment of TDs in children and adolescents when obsessive-compulsive disorder (OCD) is comorbid. Our review below highlights the fact that this comorbidity is quite common, and poses a significant challenge to treating clinicians; what is also evident from the literature is that there are empirically supported pharmacotherapies and cognitive behavioral therapies (CBT) for each disorder (e.g., Abramowitz, Whiteside, & Deacon, 2005; Cook & Blacher, 2007; Franklin et al., 2011; Piacentini et al., 2010). Moderator analyses of treatment response in the Pediatric OCD Treatment Study I (Pediatric OCD Treatment Study (POTS) Team, 2004) indicated that comorbid tic symptoms predicted poorer response to pharmacotherapy alone but not to CBT alone or to combined treatment in a trial in which OCD was classified as the primary disorder (March et al., 2007); this information needs to be considered when making selection of treatment choice for individuals with both disorders present. As yet, the converse (moderator analyses of the effect of OCD on treatment response in primary TDs) has not been explored in the context of a randomized treatment trial, so clinicians need to exercise their empirically informed judgment when considering treatment of primary TD when OCD is also present.

First we will provide a focused review of psychopathology for each of these conditions, followed by consideration of what is known when they are both present. A heuristic is then presented for arriving at judgments for managing both symptoms clinically when they co-occur. This discussion is then followed by presentation of a case composite that flows from the heuristic presented. Our view is that there is much reason for optimism that children who have TDs and co-occurring OCD can be successfully treated, but that the treating clinicians have much to keep in mind as they do so.

Tic Disorders and Tourette Syndrome

TDs (TDs) and Tourette syndrome (TS) are chronic neuropsychiatric disorders that are characterized by "sudden, rapid, recurrent, non-rhythmic, stereotyped motor movements or vocalizations" (American Psychiatric Association, 2000). To meet criteria for a diagnosis of Chronic Motor or Vocal TD, tics must occur multiple times a day most days or intermittently for at least a period of a year with onset occurring before the age of 18 years. Chronic TDs are classified as either motor or vocal and can be either simple or complex in

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nature. Motor tics are repetitive contractions of discrete muscle groups that can occur in any part of the body. Simple motor tics are brief, sudden contractions that typically affect only one muscle group (e.g., eye blinking or head-jerking). Complex motor tics are longer, sequenced, or more exaggerated movements that may present as jumping, touching, or squatting. Vocal tics are repetitive sounds, with simple phonic tics presenting as meaningless sounds such as humming, grunting, sniffing, or throat clearing. Complex vocal tics are longer in duration, more meaningful, and appear purposeful and might present as echoing a word or phrase of another or repeating one's own utterances (APA). To be classified as a TD, only vocal or motor tics can be present; when both are present, the diagnosis of TS is given. Tourette syndrome typically presents with multiple motor tics and at least one vocal tic that occur either simultaneously or at different periods during the course of illness (APA). Important to note is that understanding the *function* of the repetitive behavior is critical for accurate diagnosis: for example, a repeated head movement designed to neutralize unwanted thoughts and to reduce the likelihood of a feared outcome (e.g., physical injury or death of parents) would be accurately diagnosed as a compulsion rather than a tic. The clinician responsible for the assessment of youth with both conditions will have to make these kinds of fine-tuned distinctions frequently, since somewhat different treatment strategies would be used depending on whether the behavior was conceptualized as a tic or a compulsion.

In terms of prevalence, it was determined in a large community sample of 4,475 youth that 0.8% had chronic motor tics, 0.5% had chronic vocal tics, and 0.6% had TS (Khalifa & von Knorring, 2003). Studies report the range of tic onset from 5.6 to 7.6 years (Comings & Comings, 1985; Freeman et al., 2000; Janik, Kalbarczyk, & Sitek, 2007; Leckman et al., 1998; Lees, Robertson, Trimble, & Murray, 1984) with symptom severity commonly peaking at 10 years of age (Leckman et al., 1998). Typically, the course of the illness follows the pattern of symptom emergence in childhood, an ebb and flow in severity and frequency of symptoms throughout the illness (Leckman et al.; Lin et al., 2002; Robertson et al., 1999), and a diminishing in symptom severity by the age of 20 years, with less than 20% of individuals with TS continuing to bear moderate impairment past the second decade of life (Bloch et al., 2006). Although persistence of severe impairment into adulthood is uncommon, studies report a variety of percentages of slight to moderate symptoms continuing into adult years, ranging from 20 to 90% of individuals (Bloch et al.; Leckman et al., 1998; Pappert, Goetz, Louis, Blasucci, & Leurgans, 2003).

Tic Disorders can be difficult to distinguish from symptoms of hyperkinetic movement disorders, such as Parkinson's disease and Huntington's chorea (Kompoliti & Goetz, 1998) but the main distinguishing factor of TDs may be the volitional nature of the tic itself. In contrast to movement disorders, most individuals with TDs can suppress the urge, but experience a mounting tension that they then consciously choose to alleviate by performing a tic to relieve the tension. As a result of this partial control, tics are commonly described as semi-volitional, since they are typically executed voluntarily in response to uncomfortable, involuntary sensations, or premonitory urges. Similar to scratching an itch, performing a tic in response to a premonitory urge provides a temporary feeling of relief (Banaschewski, Woerner, & Rothenberger, 2003; Kwak, Vuong, & Jankovic, 2003). The prevalence of these urges is high; in a sample of 28 child and adult participants, 82% reported experiencing premonitory urges immediately preceding motor and vocal tics (Cohen & Leckman, 1992). Interestingly, 57% of this sample felt the urges were more vexing than the actual tics themselves. Another study found that 93% of 135 participants reported the existence of these premonitory urges, 82% felt the performance of the tic relieved the urges, and 92% divulged the tics were either wholly or partly a voluntary response to the urges (Leckman, Walker, & Cohen, 1993). As will become evident later in the chapter, these observations about tic phenomenology are of great relevance to the implementation of behavioral treatment of TDs.

Tic severity and frequency are sensitive to numerous factors, such as common, daily environmental occurrences (Conelea & Woods, 2008) as well as anxiety-provoking situations, heightened emotions, and fatigue (Findley et al., 2003; Hoekstra et al., 2004). Results from a study investigating the effects of nearly 30 environmental factors in a sample of 14 youth with TS indicated that common causes of tic increases were anxiety-provoking situations, social settings, fatigue, watching television, and isolation (Silva, Munoz, Barickman, & Friedhoff, 1995). It is important to note, however, that some individuals with tics report that some of these same environmental factors are associated with decreases in tics for them. Thus, the treating clinician should conduct a careful functional analysis with specific patients rather than making assumptions about the relationship between tic urges and environmental triggers that are based on aggregated data.

Tics and the premonitory urges that typically precede them can cause high levels of distress and impairment in individuals with TS and TDs. Studies show that youth and adults with TS typically report impairment in multiple areas of life, such as overall quality of life, social, academic, occupational, and family domains. Hindrance in overall quality of life due to TS was studied in a sample of 59 youth, where children with tics produced lower quality of life scores than a control group of healthy participants (Storch, Merlo, et al., 2007).

Although some tics affect muscle groups in less noticeable sections of the body (e.g., abdominal tensing), most tics are visible to observers and can produce great social discomfort, self-consciousness, shame, and sadness (American Psychiatric Association, 2000). Social hindrances are commonly experienced in individuals with TS, such as difficulty in creating and maintaining friendships, hardships in dating, rejection from peers, social withdrawal, teasing, aggression, low popularity, negative social perceptions, and lower social acceptability (Champion, Fulton, & Shady, 1988; Elstner, Selai, Trimble, & Robertson, 2001; Lin et al., 2007; Marcks, Woods, & Ridosko, 2005; Packer, 2005; Stokes, Bawden, Camfield, Backman, & Dooley, 1991; Storch, Lack, et al., 2007; Woods, Fuqua, & Outman, 1999). Such effects may well become treatment targets once the tics and more challenging comorbid symptoms are addressed.

Academic functioning also appears to be negatively affected by symptoms of TS. Storch, Lack, et al. (2007) found that in a sample of 59 children diagnosed with TS, 36% of youth reported their tics as the cause of diminished academic functioning, affecting their preparedness for class, their abilities to write, do homework, and their overall levels of concentration. Additionally, a survey found that in a sample of 71 parents or guardians of children with TS, 50% reported moderate to significant interference in academic functioning due to tics, which included trouble in reading and writing (Packer, 2005).

Not only do TS symptoms negatively impact the individuals who have the disorder, but also their caregivers, and can cause impaired family functioning. Studies have found that families with at least one member with TS report a heightened burden on caregivers, a diminished family cohesion, great difficulty in solving family issues, and increased interference in the daily functioning of family members (Bawden, Stokes, Camfield, Camfield, & Salisbury, 1998; Cooper, Robertson, & Livingston, 2003; Hubka, Fulton, Shady, Champion, & Wand, 1988; Storch, Lack, et al., 2007).

The data reported above indicate that TDs are a worthy target for treatment intervention in and of themselves, and may well suggest that the reduction of core symptoms could be important in improving the quality of life for affected youth and their families. Comorbidity with OCD presents an additional complication, and below we discuss OCD first in order to provide the readers with enough information to fully appreciate the complexity of their interrelationship, and the clinical conundrum that ensues when patients have prominent symptoms of both conditions.

Obsessive-Compulsive Disorder

The DSM-IV Text Revision (DSM-IV TR; American Psychiatric Association, 2000) defines OCD by the presence of recurrent obsessions

and/or compulsions that interfere substantially with daily functioning. Obsessions are "persistent ideas, thoughts, impulses, or images that are experienced as intrusive and inappropriate and cause marked anxiety or distress" (p. 457). Common obsessions are repeated thoughts about contamination, causing harm to others, and doubting whether one locked the front door. Compulsions are "repetitive behaviors or mental acts the goal of which is to prevent or reduce anxiety or distress" (p. 457). Common compulsions include hand washing, checking, and mental compulsions (e.g., repeated praying silently). A functional link between obsessions and compulsions is typically evident: for example, in the DSM-IV field trial on OCD, over 90% of participants reported that their compulsions aim to either prevent harm associated with their obsessions or to reduce obsessional distress (Foa et al., 1995). For example, the obsessional thought of an OCD patient that he or she might be responsible for harm befalling someone by having neglected to lock the door will likely give rise to anxiety or distress. Compulsively checking the door is a behavior that attempts to reduce distress and reassure the patient that the feared consequence will not occur. Therefore, if the patient does not demonstrate a clear relationship between the obsession and the compulsion (obsessions are distressing and compulsions aim at reducing this distress), another diagnosis should be considered. One of these diagnoses may well be a tic disorder if the repetitive behaviors observed serve the function of reducing discomfort at the site of the behavior (e.g., premonitory urge in the neck resulting in repetitive head jerking movements).

In order to distinguish diagnosable OCD from the virtually ubiquitous occasional and not terribly distressing phenomena of unwanted thoughts and repetitive behaviors reported by the vast majority of individuals without OCD (Crye, Laskey, & Cartwright-Hatton, 2010; Rachman & de Silva, 1978), obsessions and/or compulsions must be found to be of sufficient severity to cause marked distress, be time consuming, and interfere with daily functioning. If another Axis I disorder is present, the obsessions and compulsions cannot be restricted to the content of that disorder (e.g., preoccupation with food in the presence of eating disorders).

Epidemiological data concerning OCD varies across studies (Ruscio, Stein, Chiu, & Kessler, 2010). OCD affects up to 1 in 50 people (Ruscio et al.), is evident across development (Piacentini & Bergman, 2000), and is associated with substantial dysfunction and psychiatric comorbidity (Piacentini, Bergman, Keller, & McCracken, 2003; Swedo, Rapoport, Leonard, & Lenane, 1989). The National Comorbidity Survey Replication Study involving over 9,000 adult participants in the USA estimated that the 12 month prevalence rate of OCD was 1.0% (Kessler, Chiu, Demler, & Walters, 2005); epidemiological studies with children and adolescents suggest similar lifetime prevalence rates in these samples (e.g., Flament et al., 1988; Valleni-Basille, Garrison, & Jackson, 1994). Data concerning younger children suggest that approximately 1 in 200 young people has OCD, which in many cases severely disrupts academic, social, and vocational functioning (Flament et al., 1988; Piacentini et al., 2003). Among adults with OCD, one third to one half developed the disorder during childhood or adolescence (DeVeaugh-Geiss et al., 1992) which suggests that early intervention in childhood may prevent long-term morbidity in adulthood.

Development of OCD is typically gradual, but more rapid onset has been reported in some cases. The course of OCD is most often chronic with some waxing and waning of symptoms, with patients reporting some responsiveness to external stressors as well (Franklin & Foa, 2011). In rare pediatric cases, however, onset is very sudden (e.g., overnight) and associated with strep infection; treatment of the infection is then associated with substantial reduction of symptoms, but recurrence of infection is associated with symptom exacerbation (Pediatric Autoimmune Neuropsychiatric Disorders Associated with Strep, PANDAS; Swedo et al., 1998).

Among adults, OCD is ranked tenth among the leading causes of disability worldwide including heart disease, diabetes, and cancer (Murray & Lopez, 1996). Given what is known about the tendency for OCD symptoms to persist over time, it would be prudent for clinicians who encounter

OCD in their practices to be prepared to provide the CBT protocols of established efficacy for this condition, which appear to be effective both with and without concomitant pharmacotherapy (Abramowitz et al., 2005). Providing CBT in cases in which TD symptoms are also present has been specifically recommended as preferable to treatment with medication alone (March et al., 2007). The interplay between the clinical management of OCD and tics in practice is the primary focus of our clinical discussion below.

Comorbidity of Tics/Tourette and OCD

Although there are differences in symptom presentation that can be helpful in distinguishing the two disorders, such as the more prominent role of cognitive symptoms (e.g., compulsions) in OCD as opposed to in TDs, there are times when the symptoms of complex motor tics can be difficult to distinguish from compulsions. To make matters more confusing, comorbidity rates between OCD and tics are high, with studies reporting rates of OCD in samples of individuals with TS ranging between 22 and 41% (Freeman & the Tourette Syndrome International Database Consortium, 2007; King, Leckman, Scahill, & Cohen, 1998; Termine et al., 2006). Conversely, some 20–30% of individuals with OCD reported a current or past history of tics (Pauls, Towbin, Leckman, Zahner, & Cohen, 1986); the comorbidity rate for TDs in the recently completed POTS II study was approximately 22% (Franklin et al., 2011). The case composite we discuss below includes symptoms of both disorders, some which are easier to distinguish from one another, whereas some of the symptoms (e.g., "Not Just Right" feelings and associated "evening out" rituals) seem to fall right on the border of both conditions. What is important clinically is that patients are taught to use the proper techniques to address those symptoms that are clearly emanating from one disorder or the other, and that they become comfortable experimenting with different techniques for those symptoms that could be classified as either one. We will discuss this issue in detail in the case presentation.

In order to differentiate the stereotyped motor behaviors that characterize TS and TDs from compulsions, the functional relationship between these behaviors and any preceding obsessive thoughts must be examined. Like compulsions, complex tics may appear intentional and produce a sense of relief (Mansueto & Keuler, 2005). However, research suggests that there are phenomenological differences in the antecedents to the primary symptoms of the two disorders: sensory urges and vague somatic tension are associated with TS, while physiological arousal and specific cognitions are linked to obsessive-compulsive behavior (Miguel et al., 1995, 1997, 2000; Scahill, Leckman, & Marek, 1995; Shapiro & Shapiro, 1992). Further, while there is no conventional way of differentiating tics from "pure" compulsions, the discerning diagnostician should be aware that OCD with "pure" compulsions is extremely rare (Foa et al., 1995).

Common clinical correlates of both disorders include childhood onset, a chronic waxing and waning course, and familial occurrence (Coffey et al., 1998). Tic Disorders and OCD can also share similar clinical presentations including repetitive behaviors, intrusive sensations, and impairment in behavioral inhibition (Lewin, Chang, McCracken, McQueen, & Piacentini, 2010). The key point for the clinician to consider here is not simply to assess for the presence of comorbidity but rather to consider its treatment implications. Comorbidity of OCD in TDs may be present in many cases but, if the OCD is primary, then CBT targeting OCD will likely prove effective regardless. There is also some evidence with other comorbid conditions, such as depression, that targeting OCD in treatment can result in reductions of the nontargeted comorbid symptoms (Franklin, Abramowitz, Kozak, Levitt, & Foa, 2000). Although the assessment of OCD and TDs can be diagnostically tricky, the competent clinician should endeavor with the patient and parents to discern which disorder surfaced first and which is currently responsible for the most impairment. Once these questions have been carefully considered, the next step is to consider the implications of the comorbid symptoms for treatment. This task becomes more difficult,

however, when clinicians, parents, the child, and the child's school have different opinions as to where the greatest area of impairment lies. Indeed, it may even be the case that the context matters a great deal when it comes to symptom expression, severity, and impairment. For example, children may well be better able to suppress tic urges at school because they allocate their attention away from tic urges and towards the many activities that require increased attention in school, whereas higher demands on their attention are not nearly as prominent at home. When forming a treatment plan, these contextual factors should be taken into account when deciding where best to begin treatment, which treatment techniques to emphasize, and which treatment components would be most likely to generalize to the other areas.

Although the research reviewed above indicates that tics alone can cause impairment in functioning, several studies provide support for the hypothesis that comorbidities, rather than tics, are often responsible for functional impairment. In 98 adults with TS, Thibert, Day, and Sandor (1995) showed that those with TS and obsessive-compulsive symptoms had significantly lower self-concepts and greater social anxiety than subjects with TS alone. Likewise, Wilkinson et al. (2001) showed that families of children with TS and comorbid conditions experienced greater impairment than families having children only diagnosed with TS. In a study assessing tic persistence and associated impairment in 50 children and adolescents with TS, results showed that, from baseline to 2-year follow-up, the percentage of youth meeting criteria for tic persistence remained the same, while the percentage meeting criteria for tic impairment decreased significantly in proportion. This suggests that tic persistence and impairment may not be associated (Coffey et al., 2004). Research interested in determining the functional impairment for people with comorbid TDs and OCD has produced varying results. Lewin et al. (2010) did not find that having a diagnosis of both a TD and OCD increased the severity of either disorder in children when examining severity levels, comorbidity burden, emotional and behavioral problems, or global functioning. However, research in adults with both TDs and OCD suggests that having comorbid TDs and OCD does lead to increased symptom severity levels compared to those with TDs or OCD alone (Coffey et al., 1998). The difference in outcomes in these two studies could reflect developmental differences given the use of pediatric and adult samples.

In terms of expecting a "two birds with one stone" effect from treatment, there may be some support for the possibility that exposure treatment, which has been found efficacious for OCD across the developmental spectrum, may also have positive effects on tics and tic urges. Verdellen et al. found that tics were similarly responsive to an exposure plus response prevention (ERP) protocol when compared in a randomized study to habit reversal training where a competing response is used to substitute for the tic (Verdellen, Keijsers, Cath, & Hoogduin, 2004). However, it is important to note that an emphasis on imaginal exposure to obsessional content would be prominent in treating an individual who engages in repetitive tapping behavior to prevent a specific dreaded outcome (e.g., death of a parent in an accident); whereas, an individual who reports engaging in a nearly identical tapping behavior in order to reduce discomfort associated with premonitory urges would be unlikely to benefit from imaginal exposure (Woods et al., 2008). Thus, the degree to which the OCD and tic symptoms are formally similar may well assist the clinician in devising approaches that can be used for both phenomena, provided of course that the patient is able to carry out the relatively simple yet perhaps more effectively challenging task of permitting unpleasant emotions to go unaddressed while they habituate, which is essentially what exposure entails.

Clinical Decision-Making with Comorbid Problems

There are several potentially reasonable options to consider when treating a child or adolescent with an impulse control disorder, like TD or TS, who has comorbid OCD. Previous work concerning

comorbidity of impulse control disorders (in this case trichotillomania) has provided effective guidelines to aid in conceptualizing the clinical management of comorbid TDs and OCD (Franklin & Tolin, 2007). These guidelines are as follows: (1) continue the focus on the disorder classified as primary regardless of the presence of other symptoms; (2) attempt to incorporate some clinical procedures and session time to manage the symptoms of the co-occurring disorder but continue to focus most session time and effort on the primary disorder; (3) shift the focus of treatment to address the symptoms of the secondary disorder because their presence makes it difficult to treat the primary disorder successfully, but move back to the primary disorder as soon as possible; or (4) treat the primary disorder only after the symptoms of the secondary disorder are under better control. In the clinical circumstance we are considering here, treatment of TDs with co-occurring OCD, the data on OCD driving the functional impairment when both disorders are present makes us inclined to consider Option 1 likely to be the least effective approach, unless the OCD symptoms are very mild and are not of paramount importance in the eyes of the child. The conceptual overlap between the disorders and the procedural similarity between some of the core interventions for OCD and TDs also probably renders Option 4 a suboptimal choice, since differentiating so clearly between the two phenomena is difficult and in some cases may not even be necessary.

Thus, the options that we are most likely to consider with TDs and comorbid OCD is to incorporate procedures for both conditions into one treatment, or given the likelihood that OCD will drive the majority of the functional impairment, to address the OCD first while carefully examining the effects of the OCD treatment procedures on tic symptoms. In the case composite we present below, clinical circumstances necessitated a blend of these two approaches: we made a clinical decision with the patient and family to attempt the OCD treatment first, but when tic symptoms began to worsen in the context of the most difficult exposures, we incorporated habit reversal training procedures into the mix to help the patient remain focused on the exposure tasks at hand without becoming distracted by tic urges. The details of the case are presented in the following section.

Case Composite

The case composite we describe below represents a combination of cases we have treated in our clinic over the years which have presented with symptoms of both OCD and TDs. We have chosen to feature a case in which there were symptoms that were clearly attributable to OCD (contamination fears with specific feared consequences and associated washing compulsions), to a chronic motor tic disorder (repetitive head jerking in response to a premonitory urge that emanated from the shoulder and neck muscles), and to symptoms that appeared to rest squarely on the border of the two ("Not Just Right" experiences and associated "evening up" rituals). We do this for two reasons: (1) this sort of case complexity is common in children and adolescents with both disorders; and (2) it permits us to discuss the need to adjust the plan in response to clinical needs.

Susan was a 15-year-old sophomore at an academically challenging local high school who sought treatment for "repetitive movements that she does all the time and intense worries about getting sick." Her initial evaluation in our fee-for-service clinic included the interviewer-rated Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS; Scahill et al., 1997) and the Yale Global Tic Severity Scale (YGTSS; Leckman et al., 1989) to assess symptoms of OCD and TDs, respectively, as well as a broader diagnostic survey of other internalizing and externalizing conditions (KID-MINI, Sheehan et al., 1998). Given her age, the patient was interviewed alone; at the end of the intake, both parents were invited in to discuss the findings of the intake and to discuss treatment alternatives. Susan's CY-BOCS total score was a 23, which reflects symptoms of moderate severity. The CY-BOCS checklist revealed primary contamination fears with specific feared consequences (getting the flu and missing school), associated compulsions (excessive and ritualized

hand washing, use of hand sanitizer), and passive avoidances (e.g., waiting until someone else opened a door to pass through doorways at school rather than touching the contaminated door knob). Her YGTSS total score was a 19, which also reflected symptoms of moderate severity. Her primary tics involved neck and shoulder shrugging movements which were done in response to a premonitory urge emanating in those areas of the body. In addition, the patient reported frequent "Not Just Right" phenomena that frequently affected walking, sitting, and coming into contact with objects and people. Physical responses to those "Not Just Right" sensations, included engaging in stepping rituals, fidgeting in her seat until she felt it was correct, and performing "evening off" rituals that involved putting equal pressure on the right side of the body if the physical contact had occurred on the left and visa versa. Given the absence of a clear cognitive prompt, one could make a logical argument for placing these "Not Just Right" symptoms under the diagnostic umbrella of tics; however, they were instead classified as OCD symptoms (Coles, Frosty, Heimberg, & Rheaume, 2003) and included in the CY-BOCS total score. The broader diagnostic interview did not reveal any additional diagnoses or clinical problems other than OCD and a chronic motor tic disorder (which became the formal diagnosis given the absence of a history of vocal tics).

Discussion with the patient revealed that although both sets of symptoms were problematic, the patient was primarily concerned about the OCD symptoms since they were beginning to affect her functioning both in school and at lacrosse practice. These were of particular importance to her since she was a sophomore in high school and, despite her year, was already one of the league's best players. Concerns about showering after practice led her to avoid doing so, which was beginning to draw comment from teammates. In addition, there were times when she was unable to handle worksheets or books at home that she feared had been contaminated by someone at school (e.g., teacher, classmate) whom she suspected was already sick. Moreover, the consequences of catching the flu or a bad cold had to do with Susan's excessive fears of academic failure and preoccupation with performing perfectly in school. If she had to miss classes, she worried she would get behind on work and then would consequently be less prepared for exams. Susan's perfect 4.0 grade point average provided her little assurance that her academic goals (admission to the country's most prestigious colleges and universities) would eventually be realized, and thus her concerns about getting sick were linked to a long litany of undesirable outcomes: diminished academic performance, suboptimal transcripts, rejection letters from the colleges "that mattered," compromised career goals, and a diffuse sense that at the end of her life she would have failed to make the most of her talents, which would then affect her standing with God upon transition to the afterlife.

Susan's broad and highly specific worst-case scenario was influential as the therapist worked with the patient to determine whether to focus on OCD or on tic symptoms. Since tics were not associated with such dire consequences in the eyes of the patient, it seemed imperative to move towards the OCD and related beliefs first. Her parents agreed to this plan, and were encouraged to remain an active part of the treatment by providing Susan with emotional support as she worked on the difficult content area pertaining to contamination. It was also suggested that her parents do their best to create a "tic-neutral" environment at home (Woods et al., 2008). Strong negative reactions to her tics proved only to temporarily reduce them, and actually increased stress, and her vulnerability to stronger tic urges in the wake of this stress.

Given that the target of treatment began with contamination-related fears, associated rituals, and avoidance behaviors, the therapist began by following the outline detailed in March and Mulle's (1998) manual which served as the study manual for POTS I and POTS II. The first 4 h-long treatment sessions are conducted twice per week, and devoted to: (1) Psychoeducation about OCD and a description of the treatment procedures that would flow from this conceptual model; (2) cognitive training, which essentially involves teaching the patient to "talk back" to

OCD when it makes its demands about the patient's behavior; (3) development of a treatment hierarchy; and (4) a trial exposure in the area targeted for initial focus in ERP. Given what was already known about the tic symptoms—their phenomenology, relationship to premonitory urges, and responsiveness to stress—it was also agreed that in these early sessions we would "keep a watchful eye" on these symptoms to determine whether the stress of exposure itself would exacerbate them. If so, the therapist and patient agreed her tics would be addressed using a basic competing response procedure which is central to the more detailed tic treatment manual (Woods et al., 2008).

Early sessions proceeded as hoped—the patient worked diligently on grasping the conceptual model and even requested readings from the cognitive-behavioral literature to augment what she had learned in session. Though her enthusiasm was laudable, the therapist declined to do this out of concern that it would yield more difficulties than it could solve because given her perfectionistic symptoms, the patient might have been prone to worrying excessively whether she was implementing treatment "incorrectly." A hierarchy for contamination fears was created, ranging from low level exposures of indirect exposure to surfaces that might be contaminated (known in our lab as "the principle of the thing that touched the thing," which allows the therapist to create exposures that would be anxiety-provoking, but less so than direct contact would be) all the way up to the most feared items, which included surfaces in the bathroom and direct contact with individuals known to be sick. The trial exposure was conducted in session four and went according to plan: Susan was able to touch the therapist's office desk and file cabinets with some anxiety and with a relatively low urge to wash. In this exposure, the therapist encouraged the patient to maintain direct contact with these items until anxiety was substantially diminished, (which was defined as a 50% reduction on the fear rating scale of 0–10). Given the patient's maladaptive perfectionism to further the exposure, the therapist gave the patient specific instructions to use rough estimates and provide immediate and possibly even inaccurate responses to queries about her anxiety level. The therapist and patient both noted only a slight increase in tics when conducting the trial exposure.

Subsequent sessions were conducted weekly given the patient's relatively moderate symptom levels, busy academic and athletic schedules, demonstrated ability to understand the conceptual model that served as the foundation for treatment, and high between sessions compliance with exposure tasks and response prevention. These sessions were devoted to moving up the patient's contamination hierarchy from more anxiety-provoking objects in the therapist's office (e.g., inside door handle) and in the environment outside the office. The therapist and patient conducted these exposures in session, then decided together on how best to conduct exposures between sessions that would be challenging but not too difficult to complete without engaging in rituals.

It was during the eighth treatment session when the therapist and patient both observed significant increases in the neck and shoulder tics. In keeping with the overarching goal of confronting the most anxiety-provoking stimuli relatively early on in treatment, this session was conducted on the floor of the bathroom in the therapist's office suite. The patient and therapist both sat on the floor with their palms down at first, then moved their now-contaminated hands up to their faces so that the patient could confront her most prominent fear of ingesting germs that would lead her to become ill. The patient's agitation increased in this session to the point where it was difficult for her to complete the exposure properly, although she was eventually able to do so. The therapist sent Susan home with a "souvenir" of their work for the day, a paper towel contaminated by the same bathroom floor, which she was to use to contaminate her room and other parts of her home environment that she typically kept as pristine as possible (e.g., bookbag, kitchen counter where she made her lunches for school). The patient was able to complete these exposures between sessions 8 and 9, but noted continuing difficulty with tics while doing so. This increase was beginning to

demoralize Susan somewhat and leave her less confident that she would be able to successfully manage her symptoms As she reported at the beginning of session nine, "It's like playing that little kid game 'Whack – a – Mole.' I work on one thing only to see the other thing get worse. I'm not sure I can do this any more."

In response to her increasing distress and waning resolve to manage her OCD symptoms as planned, the therapist provided emotional support but also changed the focus of session 9 to competing response training. The overlap in the conceptual model for the maintenance of tics and compulsions was first presented to the patient. This model was described as following: both behaviors are engaged in intentionally in order to reduce anxiety and obsessional distress (in the case of OCD) or physical discomfort (in the case of tics), and completion of the repetitive behavior provides negative reinforcement, (i.e., the relief experienced strengthens the association between the unwanted, non-volitional thoughts or sensations and the completion of the volitional behavior designed to provide this relief). Susan was able to grasp this commonality relatively quickly, and then was given the rationale for engaging in a competing response. The competing response was described to her as a physical behavior that is incompatible with tic completion that the patient should implement for 1 min at the first sign of the premonitory urge or, if the urge is missed, at the sign of the first tic.

The therapist and patient practiced competing responses in session that were not associated with anxiety related to high-level exposures. This method was taken simply to ensure that the patient understood how to use the procedure. For homework the patient was given only the assignment of using competing response at home for 30 min per day after dinner, which was a time that the patient noted was "pretty free." No exposure homework was given, although Susan was given general encouragement to "do the best you can" in handling OCD-triggering situations without ritualizing or avoiding. Report of the success of the procedure and grasp on the theoretical rationale for its use was evident during the initial stages of session 10. This success then provided Susan with increased confidence that she could return to climbing her exposure hierarchy, which still involved mastering public bathrooms both in school and beyond. As the treatment for contamination fear moved forward she and the therapist planned for the use of competing response to address tic urges in the context of contamination-related exposures. Some initial "tests" were conducted to assure that focus on competing responses would not block anxiety in response to the contamination-related stimuli being addressed. These tests for Susan proved informative: she was able to better manage tic urges via competing response while also concentrating on the exposure at hand (pardon the pun), and became more and more successful at doing so as the next several sessions and weeks of exposure practice were completed. Not surprisingly, and without specific instruction to do so, Susan also began to implement the competing responses when she noticed that her tics were increasing in other settings, such as on the bench before lacrosse games or in the midst of taking tests in class.

By the end of session 12 Susan's contamination-related OCD symptoms were substantially reduced, as was the associated impairment. Notably, her use of competing response had increased her sense of control over tic urges when she felt that it was important to exercise this control. At this point in treatment the decision was reached to address residual symptoms pertaining to the "Not Just Right" phenomena that affected walking, sitting, and situations in which she was inadvertently contacted on one side of her body. Interestingly, she noted that she did not experience any of these symptoms while playing lacrosse, which often involves inadvertent contact; nevertheless, it was Susan's view that her concentration on game situations while playing distracted her from the sensations in this context. The therapist and Susan decided to approach these phenomena using an exposure paradigm: Susan was instructed to intentionally prompt the "Not Just Right" feelings by walking and sitting incorrectly, and by bumping into objects and other people on purpose. Susan found these exposures especially uncomfortable, to the point

where her neck and shoulder tics began to emerge again in session. Repeated practice proved unsuccessful in alleviating the distress, although her reintroduction of competing response did afford her some increased control over the emerging tics. Susan was encouraged to attempt these practices between sessions, but reported upon her return in session 13 that she had "given up on them" because her discomfort was coming down so slowly.

At this point the therapist decided to utilize a competing response to help with the discomfort associated with unevenness prompted by touching something on one side of her body. The therapist asked Susan to touch something against her right upper arm and then, rather than attempting to touch the object to the exact same spot with her left arm, she was encouraged to push her upper arms against her body and hold them steady there for 1 min. After several attempts, Susan reported that she had greater confidence that she could implement competing response successfully without engaging in evening off compulsions. To ensure this rise in confidence and skill, she was given this competing response to practice between sessions. Upon return to the next session, Susan reported that she had great success with this procedure for preventing herself from "evening off," and had even begun to use the procedure for her walking and sitting compulsions with similar success. We discussed the process of fading these competing responses when she noticed that the urges were consistently weaker, but Susan decided on her own that she would delay this process at least until she had completed her semester, which was several weeks away.

At the end of the acute treatment phase Susan's CY-BOCS and YGTSS total scores were reduced by greater than 50% (9 and 7, respectively) and she felt confident that she would be able to continue to function well without weekly sessions. Booster sessions were implemented biweekly, then monthly, then every other month, until such time that Susan felt they were no longer relevant. Her gains were maintained for over 2 years, and recent booster sessions were provided to help her through the stress of the college application and selection process. Although clearly anxious about these uncertainties and demands, her OCD, tic, and "Not Just Right" symptoms remained subclinical, and were not addressed at all in these subsequent sessions.

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Treatment of Childhood Anxiety in the Context of Limited Cognitive Functioning

10

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Anxiety disorders are the most common mental health problem in the United States (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). It is estimated that approximately 13% of children suffer from anxiety disorders that cause at least a mild level of functional impairment, making this the most prevalent psychiatric concern in youth (Costello et al., 1996). While the incidence of anxiety disorders in children with a concurrent intellectual disability (ID) has received little empirical attention (Ollendick, Oswald, & Ollendick, 1993), recent studies suggest that individuals with cognitive impairment are at higher risk for anxiety disorders than those without ID. Ramirez and Kratochwill (1997) found that children with ID were more likely to report specific fears and generalized anxiety than children without ID. Dekker and Koot (2003) also found that 22% of youth (ages 7–20 years) diagnosed with ID met DSM-IV-TR (American Psychiatric Association, 2000) criteria for at least one anxiety disorder. According to the World Health Organization (2007), the true prevalence rate of ID is estimated to be around 3%, suggesting that a substantial minority of individual youth may present for treatment with ID and comorbid anxiety disorders. Within the ID population, higher levels of anxiety have been associated with poorer performance on achievement tests, relative to those without anxiety concerns (Feinstein & Reiss, 1996), further supporting the need for efficacious interventions targeting childhood anxiety in this population.

Nevertheless, the lack of empirical evidence about the epidemiology of ID and comorbid anxiety disorders reflects a highly understudied domain as a whole. In fact, effective treatment modalities for anxiety have not been well studied in individuals with limited cognitive functioning (Hagopian & Jennett, 2008). A number of potential reasons for this lack of research on effective treatments exist. First, since evidence-based protocols for the treatment of anxiety disorders in youth have historically not included children or adolescents with ID in their samples (e.g., Kendall et al., 1997; Silverman et al., 1999; Walkup et al., 2008), systematic research is unavailable to substantiate whether such treatments are at all appropriate or what specific modifications might make them most useful for this population. Additionally, professionals who work with ID populations may be trained primarily to educate and teach their patients basic daily-living skills. This training may supersede the professional preparation necessary to diagnose and treat mental health-related concerns within this population (Tanguay & Szymanski, 1980). Diagnostic overshadowing, in which anxiety symptoms may be de-prioritized by clinicians and researchers due to a diagnosis of ID, may also play a role in the decreased attention to anxiety disorders within this population (McNally & Ascher, 1987).

Keeping such empirical limitations in mind, in this chapter we will review the existent literature

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on the phenomenology, diagnosis, and treatment of anxiety in youth with mild to moderate ID. We will then forward recommendations for the treatment of anxiety disorders in children with limited cognitive functioning based on both empirical findings regarding those with ID and evidence-based treatment for anxiety in children without ID. Finally, a brief case study is provided to further demonstrate such treatment recommendations and guide suggestions for further research in this area.

As noted, we will only be discussing youth who are identified as having a borderline IQ (IQ of 70–79; WHO, 1992), mild or moderate ID in this chapter. While we recognize that the assessment and treatment of anxiety in children with severe and profound ID also merit discussion, unfortunately, there has been no documented research on anxiety in this more severe population (Crabbe, 2001). Additionally, anxiety disorders appear to be more prevalent in individuals with moderate ID, as opposed to those with severe and profound ID (Holden & Gitlesen, 2004), further supporting a focus on those with milder cognitive impairments within this chapter.

Phenomenology

While the research on child anxiety within the ID population is lacking, several risk factors and common symptom presentations have been identified. Identified risk factors for anxiety within the youth ID population include experiencing a greater number of stressful life events, including the presence of only one caregiver in the home (Emerson, 2003). As is the case with typically developing children, Stavrakaki and Mintsioulis (1997) also found that specific life events often precede the onset of anxiety-related symptoms. These life events include rape/sexual assault, physical assault, accidents, illness, move, or a loss of caregiver. As previously indicated, there is an increased risk for the development of an anxiety disorder among individuals with ID that have relatively higher general cognitive ability scores (Einfeld & Tonge, 1996). Similar to the typically developing population, depression is also highly comorbid with anxiety symptoms within the ID population (Glenn, Bihm, & Lammers, 2003).

In the literature on anxiety within the ID population, anxiety disorders have generally been treated as a single entity as opposed to separate disorders; however, there has been some research examining the presentation of individual anxiety disorders among those with ID. For example, generalized anxiety disorder (GAD) in adolescents and young adults with ID appears similar to the presentation of GAD within a non-ID population; with the exception that individuals with ID report decreased amounts of rumination, decreased sleep disturbance, and decreased somatic complaints compared to their non-ID counterparts (Masi, Favilla, & Mucci, 2000). In the same study, Masi and colleagues (2000) found that those with ID and concurrent GAD had higher rates of comorbid panic disorder, but equal rates of comorbid depression and other anxiety disorders as the group with GAD, without an ID diagnosis.

Specific phobias in youth with ID appear to be concentrated on similar fears to children without ID. However, children with ID are likely to report fears that are somewhat more concrete and tend to involve animals more frequently (Ramirez & Kratochwill, 1997). Additionally, specific phobias in adults with ID may more closely resemble the content of childhood phobias, as opposed to fears observed among typically developing individuals. For instance, some common fears reported among adults with ID may include fears of the dark and dogs (Stavrakaki & Lunsky, 2007). While there has been no research to date on the prevalence or presentation of social anxiety disorder within the ID population, there is reason to believe that individuals with ID are particularly vulnerable to this disorder, given the heightened potential for social exclusion and peer victimization in this population (McNally & Ascher, 1987).

Obsessive-compulsive disorder (OCD) is identified as frequently occurring in individuals with ID (Szymanski & King, 1999). While those with ID may have difficulty reporting on the content of obsessions, observation of compulsive behaviors may be used as an indicator of an

underlying OCD diagnosis. However, it is notable that such obsessions or compulsive behaviors may also be part of stereotypies or tic-like behaviors in this population (Bodfish & Madison, 1993), further complicating the diagnosis of OCD in this population. Posttraumatic stress disorder (PTSD) may also often be found in individuals with ID (Szymanski & King, 1999) and appears to be significantly under-diagnosed in this population (Ryan, 1994). The presentation of PTSD in this population typically involves violent or disruptive behavior and is frequently comorbid with depression (Ryan, 1994).

Assessment

When presented with a child or adolescent client exhibiting ID, issues immediately become apparent when endeavoring to conduct an effective clinical assessment of the child and family's concerns. Typically, youth anxiety disorders are diagnosed through a number of methods including self-report or parent-report questionnaires, diagnostic interviews, behavioral observations, and physiological assessment (Velting, Setzer, & Albano, 2004). While time is often limited for such comprehensive assessment, at least a degree of self-report measurement is typically recommended for use with children over the age of 7 (March & Albano, 1996). However, limited cognitive and communication skills may lead to challenges in the accurate completion of self-report measures with this population (Ollendick et al., 1993). It may also be difficult to discriminate between behavioral avoidance due to anxiety and avoidance that stems from personal preferences in this population (Hagopian & Jennett, 2008). In spite of these challenges, some modified diagnostic tools exist to aid in the identification of anxiety among those with ID.

Rating Scales

While individuals with ID may have some difficulty completing rating scales, there has been evidence suggesting that those with borderline IQ scores or mild ID can accurately provide responses to questions on a Likert-type scale or questions that require basic yes or no responses (Hartley & MacLean, 2006). The comprehensibility of self-report measures among those with ID may be further improved by using pictorial representations of items or constructs and limiting the number of words within the response choices (Hartley & MacLean, 2006). There have been multiple measures either designed specifically to assess anxiety within this population, or modified from other self-report rating scales to assess anxiety in the context of limited cognitive functioning. Unfortunately, none of these self-report measures are specifically designed for youth. A brief review of such measures, as indicated for adults with ID, is provided to serve as a basis from which clinicians and researchers may consider the potential utility of existent measures or the future development of similar scales for children and adolescents.

The *Glasgow Anxiety Scale for those with Intellectual Disability* (GAS-ID; Mindham & Espie, 2003) is a self-report measure designed specifically to assess anxiety in adults with ID. It uses a three-point Likert-type scale with visual representations of response options in order to assess the domains of worry, specific fears, and physiological symptoms of anxiety. The GAS-ID appears to have good psychometric properties when used with individuals in the mild to moderate range of ID (Mindham & Espie, 2003). Another self-report measure designed specifically to assess anxiety among adults with limited cognitive functioning is the *Fear Survey for Adults with Mental Retardation* (FSAMR; Ramirez & Lukenbill, 2007). This measure uses a yes or no response format to identify frequency and intensity of specific fears. Initial findings using the FSAMR indicate that the measure demonstrates good reliability, supporting its use among individuals in the mild to moderate ID range (Ramirez & Lukenbill, 2007). In addition to GAS-ID and FSAMR, which were designed specifically to assess anxiety in individuals with ID, the *Zung Self Rating Anxiety Scale* has been modified for use with adults exhibiting ID (Lindsay & Michie, 1988). Adaptations from the original version of

the Zung include the use of simplified language, verbal presentation of items, and the use of yes or no responses. This modified version of the Zung was found to be reliable in individuals with mild to moderate ID (Lindsay & Michie, 1988).

Given the difficulties in using self-report scales to assess anxiety in individuals with limited cognitive functioning, an observation-based informant rating scale has also been developed for this population. The *Anxiety*, *Depression*, *and Mood Scale* (ADAMS; Esbensen, Rojahn, Aman, & Ruedrich, 2003) measures symptoms related to anxiety, depression, and mania among adults with mild to profound range ID. The factor structure of the 55 items of the ADAMS evidenced a five factor model. The factors identified include "Manic/Hyperactive Behavior," "Depressed Mood," "Social Avoidance," "General Anxiety," and "Compulsive Behavior." A confirmatory factor analysis was conducted to verify these factors and the model fit was found to be acceptable. Additionally, internal consistency of the subscales and test-retest reliability for both the total scale and the subscales was high. Interrater reliability was acceptable. The validity of the ADAMS was measured by comparing responses from 129 adults with a diagnosis of ID and a concurrent psychiatric diagnosis vs. the responses of a control group of 323 individuals with ID, but no concurrent psychiatric diagnosis. The resultant data supports the use of the ADAMS to screen adults with ID for bipolar disorder, depression, and OCD. Unfortunately, the data is limited regarding validity for the general anxiety subscale, given the lesser number of individuals in this study with anxiety symptoms. Further research is warranted on the convergent and discriminant validity of this measure; however, these initial results were promising regarding the utility of the ADAMS in screening for anxiety and mood symptoms among individuals with ID (Esbensen et al., 2003).

Diagnostic Interviews and Behavioral Observation Tasks

As noted, assessments of youth anxiety often consist of structured interviews, such as the Anxiety Disorders Interview Schedule for the DSM-IV, Child Version (ADIS-IV-C/P; Silverman & Albano, 1996) or Diagnostic Interview Schedule for Children (DISC-IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), and behavioral observation paradigms (Dadds, Rapee, & Barrett, 1994). Particularly when working with children exhibiting limited cognitive functioning, parents and other care providers are instrumental in providing information through an interview format. Identification of anxious and avoidant behaviors, as well as attributions about the causality of such behaviors may be challenging for caregivers, particularly in the context of lower cognitive functioning; therefore, the diagnostic interview should be a starting point for the formation of hypotheses or case conceptualization within the assessment, and may not be ideal as a sole diagnostic tool in the identification of anxiety disorders. Furthermore, commonly used diagnostic interviews such as the ADIS-IV-C/P and DISC-IV do not have published data regarding their use or utility with the ID population, indicating that results, even those from a parent or caregiver portion of the interview only, should be interpreted with caution.

Hagopian and Jennett (2008) recommend the use of behavioral observation paradigms, such as the *Behavioral Activation Test* (BAT; Dadds et al. 1994), indicating that such paradigms may provide more confidence regarding the presence of anxiety and avoidance-related behaviors within this population. The BAT is a structured method of assessing avoidant behavior through progressive exposure to feared stimuli. Comprehension of the clinical severity and functional impairment associated with anxiety can be aided by identifying points at which the youth displays avoidance or escape-oriented behaviors. While the use of a BAT has not been studied for its clinical utility in individuals with ID per se, clinical case studies regarding the treatment of anxiety in this population frequently employ the BAT in the measurement of anxiety symptoms. For example, Erfanian and Miltenberger (1990) used a BAT to aid in the characterization and diagnosis of specific phobias of dogs and the formation of an appropriate fear hierarchy for two individuals with ID.

In addition to the use of a BAT, naturalistic observation can also be used to assess anxiety in individuals with ID. While, it may be difficult for the clinician to be present when a particular anxiety-evoking situation occurs, they may work with a child's parents and care providers to monitor anxious behavior, along with antecedent and consequent events using functional analytic techniques (Hagopian & Jennett, 2008).

Interventions for Anxiety in Individuals with Intellectual Disability

While treatments for anxiety within the general youth population have been well studied (Barrett, Farrell, Pina, Piacentini, & Peris, 2008; Silverman, Ortiz et al. 2008; Silverman, Pina, & Viswesvaran, 2008), there is little research regarding interventions for anxiety among children, adolescents or adults within the ID population. The literature that does exist detailing the treatment of anxiety and concurrent ID is limited to clinical case reports that focus on general symptom presentations and fail to include formal DSM diagnoses (Hagopian & Jennett, 2008).

From a review of such published case studies, Davis, Saeed, and Antonacci (2008) found that youth with developmental disorders, including ID, might benefit from modified versions of existing cognitive-behavioral interventions for anxiety. Modifications suggested by these authors include the presentation of treatment concepts in a more concrete manner, increased repetition of concepts over a greater number of sessions, greater use of behavioral reinforcement and modeling techniques, and increased parental involvement in the conduct of treatment (Davis et al., 2008). These recommendations suggest that evidence-based treatment packages with a strong cognitive-behavioral focus, particularly those with similar modifications for younger children (e.g., *Being Brave*; Hirshfeld-Becker et al., 2010; *Parent–Child Interaction Therapy for Separation Anxiety Disorder*; Pincus, Santucci, Ehrenreich, & Eyberg, 2008), those targeting school-aged children that include explicit involvement of parents in a "coaching" capacity (e.g., Silverman's "*Transfer of Control*" *model*; Silverman & Kurtines, 1996), and those developed with similar modifications for children with autism and anxiety disorders (e.g., Wood, Drahota, 2005) may be relevant models from which a clinician may work to tailor the intervention to the needs of specific youth with anxiety disorders and ID.

Bogacki, Newmark, and Gogineni (2006) also suggest that a combination of treatment approaches, including pharmacological, psychosocial, and behavioral may be appropriate for the treatment of anxiety disorders among those with ID, reflecting the potential for multiple service needs and complexity of case presentation among those with ID. A review of the existent literature in this domain suggests that certain behavioral, cognitive, and pharmacologic treatment components may have particular relevance when crafting a multicomponent intervention strategy for youth with ID and anxiety disorders. These components and treatment strategies are now reviewed in greater depth below (also see Fig. 10.1).

Behavioral Treatment Components

Classical conditioning, operant conditioning, and social learning theories have all contributed to the development of efficacious treatments for anxiety through the roles of paired association and avoidance learning in the development and maintenance of anxiety. Jennett and Hagopian (2008) identified selected behavioral procedures, including graduated exposure and reinforcement, as useful techniques for the treatment of phobic avoidance in the ID population. In another study by the same authors (Hagopian & Jennett, 2008), they recommend the use of a BAT to form a fear and avoidance hierarchy for those unable to verbalize experiences of anxiety, in addition to its usage as an initial assessment tool. These authors also suggest the use of a systematic preference assessment, based on nonverbal choice responses, to identify preferred reinforcers. Systematic preference assessments are performed by methodically exposing individuals to varying stimuli while recording their responses and can either take the form of an

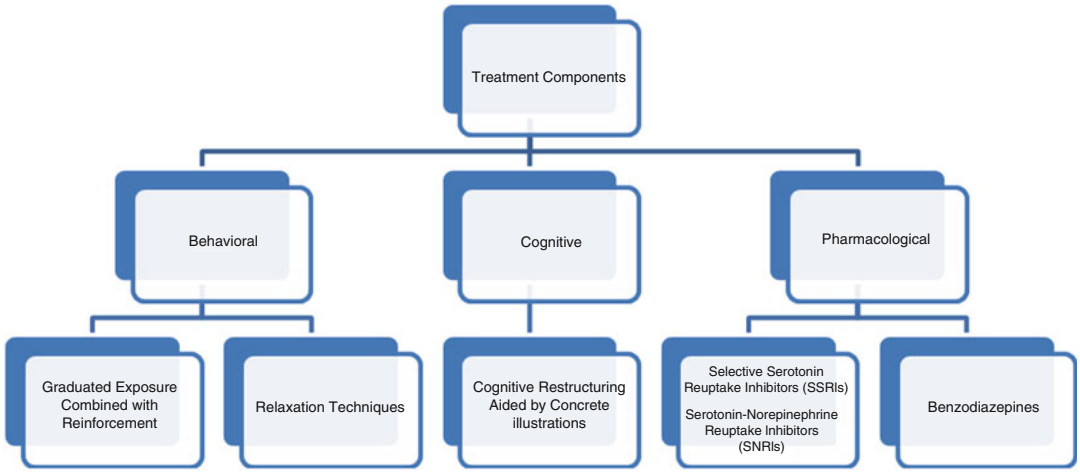

Fig 10.1 Various intervention strategies used for the treatment of anxiety in youth with ID

approach-based or an engagement-based assessment (Hagopian, Long, & Rush, 2004). In addition to the use of the BAT and a systematic preference assessment, Hagopian and Jennett (2008) also emphasized the heightened importance of not pairing feared stimuli with aversive stimuli during exposures, given the potential negative conditioning effect of this pairing.

In addition to these specific recommendations, Hagopian and Jennett (2008) further identified behavioral treatment components that may be used in combination with graduated exposure and reinforcement in the treatment of anxiety in individuals with ID. These additional components include prompting, response prevention, and the use of distracting stimuli. The use of a "least-to-most" prompting hierarchy when assisting an individual to comply with the steps of an exposure hierarchy is also recommended. In this model, the clinician first uses a participant model, then a verbal prompt, and finally a physical prompt to expose the individual to the feared stimuli. For a case study previously mentioned, Erfanian and Miltenberger (1990) used such a "least-to-most" prompting hierarchy during treatment for specific phobia of dogs in two individuals with moderate to profound ID. In the first session of treatment, a small dog and its owner were positioned on the opposite side of the room from the participant, while the participant engaged in rewarding activities and was prompted to watch the owner engage with his dog. Over the next few sessions, the participants were told to move one foot closer to the dog and then they were physically guided to move closer to the dog, while engaging in reinforcing activities. With each approach, the trainer brought the participant's awareness to the dog and praised them for their efforts. Once the participant could comfortably approach within three feet of the dog, a larger dog was used.

In addition to other behavioral techniques, relaxation training, including the use of muscle relaxation and breathing exercises, have also been investigated with ID samples. Commonly used relaxation training procedures include *Progressive Relaxation* (Jacobsen, 1938) and *Abbreviated Progressive Relaxation* (APR; Bernstein & Borkovec, 1973). These techniques have been applied to adults with ID to treat a range of behavioral and cognitive difficulties, including phobic symptoms (Guralnick, 1973; Peck, 1977). In these studies, relaxation exercises were combined with other behavioral techniques to systematically desensitize individuals to feared stimuli. However, APR alone has also been shown to reduce anxiety in individuals with mild ID, although less so among individuals with moderate to severe ID (Rickard, Thrasher, & Elkins, 1984).

Alternative relaxation techniques may be used when working with individuals exhibiting moderate to severe ID. Schilling and Poppen (1983)

developed Behavioral Relaxation Training (BRT) after discovering that APR was ineffective with boys exhibiting learning disabilities. In BRT, the instructor models the unrelaxed and relaxed states in different body areas and then the patient is asked to imitate the relaxed states. BRT has demonstrated enhanced efficacy vs. APR for adult patients with moderate and severe ID (Lindsay, Baty, Michie, & Richardson, 1989).

Ethical considerations in the use of behavioral treatment components for youth with ID. While behavioral treatment elements, such as exposure techniques, are clearly vital treatment components for youth anxiety disorders, it is important to consider the ethical implications of their usage when treating anxiety in children with limited cognitive functioning. Exposure exercises may be distressing for some children, even in the hands of a master clinician. However, in treating individuals without ID, the rationale for the exposure and corresponding distress can often be effectively communicated before the child initially confronts feared stimuli in the presence of a helpful and guiding clinician. This psychoeducation allows time for the child to generally assent to the exposure or at least comprehend its rationale, in spite of the distress that may result. However, in treatment of youth with ID, it may not be possible for the child to fully gain this understanding before they engage in exposure exercises. In these cases, extra care and consideration should be taken to slowly and gradually move along a fear hierarchy when exposing the patient to a feared stimulus, carefully reiterating the rationale and benefit of the process repeatedly. Although not ideal in most exposure scenarios, the alternate usage of systematic desensitization paradigms vs. graduated exposure may be considered to maintain rapport and motivation for treatment.

Cognitive Treatment Components

Cognitive theories emphasize the role of thought in influencing behavior and posit that maladaptive behaviors ultimately stem from dysfunctional thought processes. Cognitive therapy techniques for anxiety are often aimed at teaching the patient to evaluate and modify distorted cognitions or threatening appraisals regarding anxiety-provoking situations. While there has been some support for the use of cognitive components in the treatment of anxiety in ID populations (Dagnan & Lindsay, 2004), research in this area is also lacking. Dagnan and Chadwick (1997) identified two distinct approaches to cognitive therapy used in interventions for adults with ID. One approach is based on a cognitive distortion model, in which anxiety is seen as being caused and maintained by the individual's misinterpretations of feared stimuli. The second, and more widely used approach, is based on a deficit model, which assumes that emotional and behavioral difficulties are due to a lack of cognitive skills and processes among those with ID.

Unfortunately, no research exists on the efficacy or usage of cognitive components with youth exhibiting ID and anxiety disorders. Although implications of the deficit model (e.g., a need for cognitive skill-building and enhancement of positive social interactions) may also be useful for children and adolescents with anxiety disorders and ID, the use of cognitive techniques alone seems unlikely to be beneficial for such youth, unless substantially aided by concrete illustrations, visual depictions of complex constructs, and other modifications similar to those suggested by Davis and colleagues (2008). For example, children with ID and social anxiety may benefit from viewing depictions of appropriate social skills or responses to the evocation of social anxiety in the popular media (e.g., viewing a clip from a movie such as "Mean Girls"), discussion of the appropriateness of the skill exhibited and its relevance for the individual client, rather than relying on self-generated examples in session alone.

Pharmacotherapy

Medications often recommended for the treatment of adults and youth with anxiety disorders include selective serotonin reuptake inhibitors

(SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), benzodiazepines, and buspirone, among others (Vanin & Helsley, 2008). When administered to youth, SSRIs have demonstrated efficacy for the acute treatment of social anxiety disorder, separation anxiety disorder, OCD, and GAD (Vitiello & Waslick, 2010), although rates of remission and long-term improvements vary widely by disorder. Despite a black box warning regarding suicidal ideation (Food and Drug Administration [FDA], 2007), a recent review concluded that SSRIs are generally safe when administered to children; however, side effects including insomnia, nervousness, restlessness, fatigue, dizziness, sedation, nausea, and headaches may be reported in some children (Vitiello & Waslick, 2010).

Like most research on youth with ID and anxiety, evidence regarding the use of pharmacotherapy to treat youth with ID and anxiety disorders is extremely scarce. Davis et al. (2008) identified three studies that have tested the utility of SSRIs on the reduction of anxiety in children with a variety of pervasive developmental disorders (PDDs). While all of these studies were limited methodologically, some reduction of anxiety symptoms was seen, providing preliminary support for the use of SSRIs with this population. One of these studies consisted of a retrospective chart review assessing the benefits and negative side effects of citalopram in youth, ages 4–15 years, with PDD (Couturier & Nicolson, 2002). Eight of these 17 subjects had a concurrent ID diagnosis. Ten of these patients (59%) were rated as much or very much improved on the Clinical Global Impression scale (CGI; Guy, 1976) in regard to their target symptoms following citalopram usage. While citalopram was prescribed to address a variety of target symptoms in this study, anxiety and aggression were the most likely to improve. These findings did not differ across levels of cognitive ability. In regard to negative side effects, citalopram was well tolerated by most child patients. However, four families discontinued their child's medication within the first 2 months of treatment due to adverse responses, including increased agitation, insomnia, and possible tics.

There have been no systematically controlled trials conducted to assess the efficacy of anxiolytic medication in individuals with ID (Crabbe, 2001). However, anxiolytics are commonly prescribed in this population to control disruptive behavior and for symptoms related to GAD (Aman, Collier-Crespin, & Lindsay, 2000). Due to the lack of research on the effects of anxiolytics on children with ID, these authors advised using caution when considering whether to prescribe this class of medication to youth (Aman et al., 2000).

Case Study

Katrina M.[1] is a 9-year-old girl of Cuban-American descent that presented for treatment, along with her mother and father, to an anxiety research clinic situated in an academic psychology department. During an initial assessment session, Mrs. M provided a copy of a recent psychoeducational evaluation indicating that Katrina currently had a full-scale IQ of 72, with no significant discrepancies between her factor scores on the Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV; Wechsler, 2004). Katrina was currently enrolled full-time in a special education classroom at her school that utilized a curriculum suitable for children and young adolescents with a variety of developmental and intellectual disabilities. Katrina was able to speak to clinicians clearly in English and Spanish, but primarily spoke English using brief, clearly distinguishable statements in sessions. Using the ADIS-IV-C/P (Silverman & Albano, 1996), the initial examiner indicated that Katrina was currently experiencing clinically significant symptoms of Specific Phobia, Animal Type (Dogs) with a Clinical Severity Rating (CSR) at a six (range = 0–8). This interview and additional questionnaire measures completed by Mrs. and Mr. M indicated that no other emotional disorder or behavioral symptoms were currently present at

[1] Katrina's case information is a composite of several prior cases. No identifying or descriptive data from any prior case is used in this case presentation.

a clinical level. However, Katrina's parents did report subclinical symptoms of social anxiety and depression that did not currently result in any noticeable impairment.

During the diagnostic interview, Katrina and her parents discussed the functional impairments that Katrina experienced reportedly in response to her fears about interacting with dogs of any breed or size. No significant phobic behaviors regarding dogs were noted for Katrina prior to age 5. At age 5, Mrs. M reported that Katrina was playing with her older brother at a local field when a large, off-leash dog came ran over to them unexpectedly. Mrs. M reported that Katrina did not see the dog approach and when she turned, the dog was very near to her and immediately jumped up and licked her face. Katrina was reportedly startled and began crying uncontrollably. She was immediately separated from the dog by nearby relatives and removed from the area. However, she apparently was unable to calm herself sufficiently for several hours following the incident. Mrs. M indicated that although they knew it was "not the right thing to say", the only thing that seemed to sufficiently reduce Katrina's distress at that time was to repeatedly state that Katrina would not have to interact with a dog "ever again."

During the intervening 4 years, Katrina developed a systematic line of questioning related to dogs that she "required" family members to answer prior to entering most new situations, including unfamiliar homes, parks, and other situations where a dog may be present. These questions related to the likelihood of a dog being present and the plan for removing Katrina from the situation immediately, if one were present. If Katrina were entering the home of a familiar person with a dog, she would require that the dog be placed in a room with the door closed before entering the house. Prior to the onset of the current course of treatment, Katrina received 27 sessions of "supportive psychotherapy and family therapy" from a clinical social worker that subsequently referred the family to the anxiety research clinic. The referring clinician indicated that she believed Katrina was now in need of "exposures" and may be amenable to receiving such at this time. Katrina's parents concurred that Katrina recently made a friend with a dog that she wished to see more often and was motivated to try and work more directly on her fears.

Treatment consisted of 12 sessions, inclusive of an initial rapport building and psychoeducation-oriented session, a baseline BAT and reinforcer assessment session, nine in vivo exposure sessions, and a concluding BAT and relapse prevention-focused session. During the initial rapport building session, the clinician developed her relationship with Katrina through the use of a "memory game" adapted from Wood and colleagues (2005), in which Katrina was prompted to state five "fun" facts about herself and the clinician did the same, then both restated as many of these facts about each other as possible at intervals throughout the session. Katrina responded very positively to this game and was able to discuss her feeling that dogs were "very scary" and her concurrent desire to play regularly at her classmate's home. Much of this initial session was also spent providing psychoeducation about the nature of fear to Katrina's parents, with them guiding the clinician regarding how best to share this information with Katrina in a concrete manner. Before ending session, the clinician presented Katrina and her parents with the rationale for the BAT and exposure as a means to reduce Katrina's anxious and avoidant behaviors. The family agreed to a BAT session with a "small, friendly dog" to aid the clinician in creating a fear and avoidance hierarchy that would eventually facilitate graduated exposures.

Although the initial BAT made use of only one dog (a small, Chihuahua mix), it was effective in assessing spontaneous statements made by Katrina that were suggestive of specific fears about being licked and jumped on by the dog. Prior to the BAT, a reinforcer preference assessment suggested that having a small amount of soda, juice, or candy during session would be useful as Katrina took steps toward a dog or engaged in new behaviors (e.g., touching a dog, allowing a dog to lick her, giving a dog a treat) and these were used throughout the BAT and most subsequent exposure sessions.

During the nine exposure sessions, a "least-to-most" prompting hierarchy was utilized, similar to that recommended by Erfanian and Miltenberger

(1990). Katrina was able to steadily progress from smaller and more familiar dogs to those that were larger and more overtly intimidating to her. She was also prompted to use a set of questions when encountering a new dog owner (e.g., Is your dog friendly? Do you think it would be safe for me to pet your dog? Can you show me how your dog likes to be pet?) and then engage with the dog as directed by the owner. Memory for these questions was prompted by her parents and by allowing Katrina to carry a laminated card with her that included what she referred to as her "brave puppy questions." At home, Mrs. and Mr. M aided in the generalization of these new, approach-oriented behaviors by ensuring that Katrina was given opportunities to practice acquired skills with new dogs and those that she had avoided in the past. They were asked to initially follow the same exposure procedure as they observed and participated in with their clinician. By session eight, they and the clinician were able to fade the provision of a reinforcer to the end of Katrina's interactions with a dog, rather than throughout the exposure, with an equivalent amount of success.

At the concluding BAT session, Katrina was able to easily approach the same small dog she initially demonstrated much fear and reticence in approaching during session two. She indicated that dogs were still "scary" to her, but that she now knew how to use her "brave puppy questions" and approach skills to manage such interactions across a range of dog sizes and activity levels. Her parents indicated high levels of satisfaction with the treatment. Finally, at a posttreatment assessment, the examining clinician rated Katrina's specific phobia at a CSR of three, indicating that she still possessed and vocalized some fears about dogs, but was no longer demonstrating clinically significant levels of such or notable functional impairment.

Conclusions and Future Directions

When discussing the presentation of anxiety disorders in the context of a child with ID, inevitably the cognitive limitations of the child client come to mind as a barrier to effective treatment. As this chapter reflects, numerous treatment strategies may be attempted with such youth in hopes of alleviating their anxiety-related distress and behavioral avoidance. Furthermore, the usage of modified cognitive-behavioral treatment (CBT) packages that have demonstrable efficacy among typically developing youth with anxiety disorders may hold potential to benefit children with ID. Nonetheless, it is fair to suggest that the state of the literature on youth with anxiety disorders and ID is poor. Further research is clearly indicated regarding the etiology, epidemiology, assessment, and treatment of this population.

In terms of treatment research, although interesting modifications and suggested applications of behavioral and pharmacologic treatment approaches have been proposed for children with ID and anxiety, it is unclear how well such treatments might apply to the greater population at hand, given that the available data is limited to a handful of case examples and limited open trial research on pharmacologic approaches. Clearly, the further study of such approaches using carefully controlled designs and randomization paradigms is needed. Although such treatments appear quite plausible for children with ID, these interventions are likely to be presented to families in the context of a necessarily limited assessment process that may fail to fully identify the function and reinforcement of anxious and avoidant behaviors, particularly if time constraints limit use of techniques such as the BAT or other observational methods that appear particularly important to the assessment of youth with ID and anxiety. Therefore, the matching of a particular treatment approach to the functional impairments of a given child may be challenging to achieve.

Acceptability and satisfaction data regarding potentially efficacious approaches, including behavioral techniques and modified CBT protocols, also appears vital to future treatment research. For instance, even families of typically developing children may have a difficult time comprehending the value and relative safety of exposure techniques. However, as noted in this chapter, families of children with ID may be in

particular need of support to systematically and supportively move through fear and avoidance hierarchies effectively. Overall, such research questions are ripe for systematic evaluation. Given the commonality of ID and its frequent co-occurrence with anxiety disorders in youth, future engagement in research on these topic areas appears crucial.

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Special Considerations in Treating Anxiety Disorders in Adolescents

11

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Nature of the Problem

Anxiety disorders affect 6–7% of children and adolescents (Cartwright-Hatton, Roberts, Chisabesan, Fothergill, & Harrington, 2004; Compton et al., 2004) and are associated with wide-ranging personal and social consequences including poor school performance, disrupted relationships with peers and adults, as well as diminished participation in the typical activities of youth. Adolescence is often a time when the consequences of untreated anxiety become particularly damaging (Silverman, Pina, & Viswesvaran, 2008), the frequency of comorbid disorders increases (Carey & Oxman, 2007), and maladaptive coping styles and family interaction patterns become entrenched.

Cognitive behavioral therapy (CBT) is the most established evidence-based treatment for anxiety disorders in youth and is considered probably efficacious based on meta-analytic reviews (Silverman et al., 2008; Canadian Psychiatric Association, 2006; Connolly, Bernstein, & the Work Group on Quality Issues, 2007; National Institute for Health and Clinical Excellence, 2007). The addition of medication targeting serotonin may produce a more robust therapeutic effect than either intervention alone (Compton et al., 2010). Nevertheless, a substantial number of children and youth fail to respond to treatment in general and CBT in particular.

Despite the vast literature on the treatment of adult and childhood anxiety disorders, the treatment of adolescents with anxiety disorders has received limited research attention (Bennett et al., 2010; Masia-Warner, Fisher, & Reigada, 2008). Furthermore, anxious adolescents who are willing and able to complete research protocols may not be representative of those typically seen in community settings (Manassis, 2009). Thus, the effective treatment of adolescents with anxiety disorders continues to be a challenge that merits further research and careful consideration. This chapter reviews the developmental and social factors that may account for this challenge, key research evidence pertaining to the treatment of anxious adolescents, and treatment approaches that may improve outcomes for anxious adolescents. These approaches are illustrated using a case example.

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Factors Contributing to Complexity

Empirical Evidence

Interpreting the evidence for the treatment of adolescents with anxiety disorders is complicated by the fact that few treatment studies have focused

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exclusively on this age group. The existing studies reviewed have all employed various forms of CBT.

Adolescent Social Phobia has received somewhat more research attention than other adolescent anxiety disorders. Several authors have reported symptomatic improvement following group CBT for adolescents with this disorder in both open and waitlist-controlled trials (Albano, Marten, Holt, Heimberg, & Barlow, 1995; Baer & Garland, 2005; Hayward et al., 2000; Herbert et al., 2009), with maintenance of gains at 1-year follow-up (Albano et al., 1995; Hayward et al., 2000). Group therapy generally included some social skills training, in addition to cognitive strategies and behavioral exercises (e.g., exposure to social situations). Interestingly, a recent study which included an active control condition (i.e., educational/supportive psychotherapy) found symptomatic improvement and improved functioning following both the CBT and active control conditions, except with greater behavioral gains following CBT (Herbert et al., 2009).

Apart from Social Phobia, few other disorder-specific treatments have been evaluated in studies focused exclusively on adolescents. CBT to address school refusal has been researched with and without the concurrent use of the antidepressant imipramine (Layne, Bernstein, Egan, & Kushner, 2003). Results have favored the combination of CBT and imipramine over CBT alone. Higher baseline attendance and the absence of Separation Anxiety Disorder or Avoidant Disorder (an older diagnosis, similar to generalized Social Phobia) also predicted more favorable outcomes (Layne et al.). CBT combined with interpersonal skills training was studied in a small group of adolescent girls with Generalized Anxiety Disorder in an open trial (Waters, Donaldson, & Zimmer-Gembeck, 2008). Improved interpersonal functioning and reductions in anxious and depressive symptoms were found with treatment.

Treatments have also been developed and evaluated for adolescents with various anxiety disorders (usually, one or more of Generalized Anxiety Disorder, Social Phobia, or Separation Anxiety Disorder) in academic settings and (in one study) an urban school setting. In a small randomized controlled trial ($n = 12$), Ginsburg and Drake (2002) reported symptomatic improvement and 75% remission of primary anxiety disorder following school-based CBT for urban, African-American adolescents. By contrast, only 20% of adolescents remitted following an attention-support control condition. Siqueland, Rynn, and Diamond (2005) randomly assigned 11 adolescents to CBT alone or CBT plus an attachment-based family treatment. Both groups showed decreases in anxious and depressive symptoms, without a significant group difference. Legerstee et al. (2008) examined the role of parental psychopathology in moderating individual CBT results for 51 adolescents with various anxiety disorders. Maternal anxiety predicted more favorable treatment outcomes.

Most CBT treatment studies have included some adolescents with anxiety disorders in samples that span a broad age range and are predominantly comprised of younger children. Only a few of these studies have examined age as a potential moderator of treatment effect, with equivocal results. While some studies found no age effects (Berman, Weems, Silverman, & Kurtines, 2000; Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008), others found better outcomes in younger children (Bodden et al., 2008; Southam-Gerow, Kendall, & Weersing, 2001), and only one study found better outcomes in older children (Cobham, Dadds, & Spence, 1998). A recent meta-analysis suggests that anxious adolescents benefit from CBT to the same extent than younger children (Bennett et al., 2010). However, most of the research was developed in academic settings, where therapists are generally well-trained in developmentally appropriate adaptations of CBT. Results might differ in community settings. Results may be further confounded by the tendency of most researchers to report only on treatment completers. Focusing on treatment completers obscures possible age-related differences in treatment participation and dropout rates. Furthermore, age is not always a good proxy for developmental level, as adolescents of the same age can vary widely in their physical, cognitive, emotional, and social level of maturity.

Developmental Factors

Cognitive, psychosocial, and physical changes as well as the nature of anxiety disorders in adolescence can both help and hinder successful treatment. Cognitively, adolescents have a greater capacity for abstract reasoning than younger children, including self-reflection and insight which are the capacities deemed most relevant to CBT (Sauter, Heyne, & Westenberg, 2009). Therefore, they can often more readily recognize and challenge maladaptive, anxious thoughts compared to younger children. Due to their increasing cognitive abilities, however, adolescents can also generate more complex worries than younger children. Similarly, "the imaginary audience" (a belief that everyone is watching the adolescent) is a mild cognitive distortion that can be considered normative in adolescence (Kingery et al., 2006) yet can also contribute to social anxiety. Fear of social evaluation can affect treatment motivation, as many adolescents worry what their peers will say if they confess to seeing a "shrink." Further, cognitive development is quite variable in adolescents, and some never acquire the highest levels of reflective thought. Therefore, therapists must evaluate cognitive capacities relevant to CBT rather than assuming that all adolescents possess these necessary skills.

Psychosocial development in adolescence is characterized by an increased need for autonomy (Stallard, 2002). Autonomous behavior can aid therapy, such as allowing an adolescent to travel to and from appointments independently or organize exposures to feared situations without parental help. Unfortunately, a desire for autonomy can also interfere with engagement in therapy, particularly if the therapist is seen as authoritarian (rather than collaborative), being allied with the adolescent's parents, or coercing the adolescent into attending therapy. Once in therapy, a desire to assert his or her autonomy can also result in the adolescent avoiding exposure tasks or completing homework between sessions. Appropriately, involving parents in therapy can also be difficult in some adolescents, who may assert their autonomy by trying to prevent parental contact with the therapist. Parents of anxious adolescents may feel helpless to encourage age-appropriate independent behaviors (Foa & Andrews, 2006), as adolescents often argue with parents in an attempt to assert their autonomy while their physical size reduces the parents' ability to control them.

Physical development can also affect adolescent anxiety and its treatment. For example, children who appear physically mature yet lack cognitive or emotional maturity may face unrealistically high expectations from others (who may assume they are older than they really are), contributing to anxiety. Likewise, therapists can also overestimate these adolescents' abilities, adversely affecting the adolescent-therapist alliance and therapeutic outcomes. Additionally, response to medication can be affected by physical development (see Labellarte, Ginsburg, Walkup, & Riddle, 1999). For example, rapid liver metabolism may result in a need for higher doses of certain medications for adolescents compared to adults of similar size, while other medications may still need to be provided at doses similar to those given to children. Some side effects (such as sexual ones in serotonin-specific medications) may be more concerning to adolescents than to younger children. The risk of weight gain is another side effect that tends to be of great concern to teenagers, even though this risk may actually be greater for prepubertal children than adolescents (Jerrell, 2010).

The nature of anxiety disorders is also different in adolescents than in younger children, often affecting treatment. For example, comorbid depression and substance abuse are more common in anxious adolescents than children (Carey & Oxman, 2007), which may undermine anxiety treatment if undiagnosed. If they are detected, therapy may need to be tailored to address these conditions in addition to the adolescent's anxiety disorder. In children who have suffered with anxiety disorders for years, maladaptive coping styles and patterns of family interaction may become entrenched in adolescence. For example, families may accommodate some manifestations of their child's anxiety by tolerating immature behavior, avoiding discussions that might be anxiety-provoking for the child, or allowing the child

to avoid age-appropriate activities. Maladaptive coping styles can make CBT more challenging, as adolescents are sometimes more resistant to change than younger children or have little hope for change. Maladaptive patterns of family interaction may require additional family therapy or targeting these issues in treatment.

Social Factors

Children are expected to function at an increasingly independent level as they progress through adolescence. School work completion, transportation, and social conduct increasingly become the adolescent's responsibility rather than that of his or her parents. Subjectively, anxious adolescents may find these expectations stressful, especially if their families have been overly protective in the past, limiting their experience with independent behavior. The cognitive distortions commonly associated with anxiety (e.g., perfectionistic beliefs or biases toward threat perception) may contribute to this stress, as they may result in anxious adolescents perceiving societal expectations as being higher than they actually are (Lonigan, Vasey, Phillips, & Hazen, 2004).

As a result of these developmental changes, anxiety disorders often result in greater actual and perceived impairment at this age than in younger children. For example, Silverman et al. (2008) have argued that social anxiety symptoms that may cause minor impairment in an elementary school child could be catastrophic for a high school student who was unable to pass an oral examination required for graduation. Societal expectations can also place anxious adolescents at a disadvantage relative to their peers. In particular, anxious adolescents are expected to function more independently from their families than anxious children. The inability to do so (e.g., due to Separation Anxiety or Social Phobia) can single out these youth among their peer group. When a young person is less independent and socially competent than average, there can be detrimental effects on self-esteem and social functioning. Gender role expectations also change at adolescence. Gender-specific expectations have been associated with anxiety symptoms and anxiety disorders in children and youth, possibly contributing to a higher rate of anxiety disorders and anxiety-related impairment among adolescent girls than adolescent boys (Ginsburg & Silverman, 2000). For all of these reasons, limiting anxiety-related impairment is often an important therapeutic focus in adolescents.

Key transition points where the adolescent faces new challenges may be particularly difficult for someone with an anxiety disorder yet may also represent a therapeutic opportunity. For example, the need to cope with increased academic work, multiple teachers and classrooms, and a larger peer group can make the start of high school a stressful change for many anxious youngsters. Adolescents with anxiety disorders are vulnerable to increased distress and deterioration in functioning in response to this change. However, as a result, their willingness to engage in treatment may also be heightened.

Treatment Approaches to Address Complexity

An approach to treatment must address some of the complexities encountered in adolescents with anxiety disorders is shown in Fig. 11.1. Prior to treatment, it is important to do a developmentally sensitive assessment. Assessments with adolescents differ from those with younger children mainly in that extra attention is devoted to engaging the young person in a developmentally appropriate manner. Such engagement is essential for obtaining complete and accurate information and subsequently for successfully initiating psychotherapy.

Engaging the Adolescent in Therapy

Due to their desire for autonomy, concern about social stigma, and greater identification with peers rather than adults, adolescents can be difficult to engage in therapy. Although various strategies to improve engagement have been described, the most consistently advocated strategy is that the therapist adopts a collaborative rather than author-

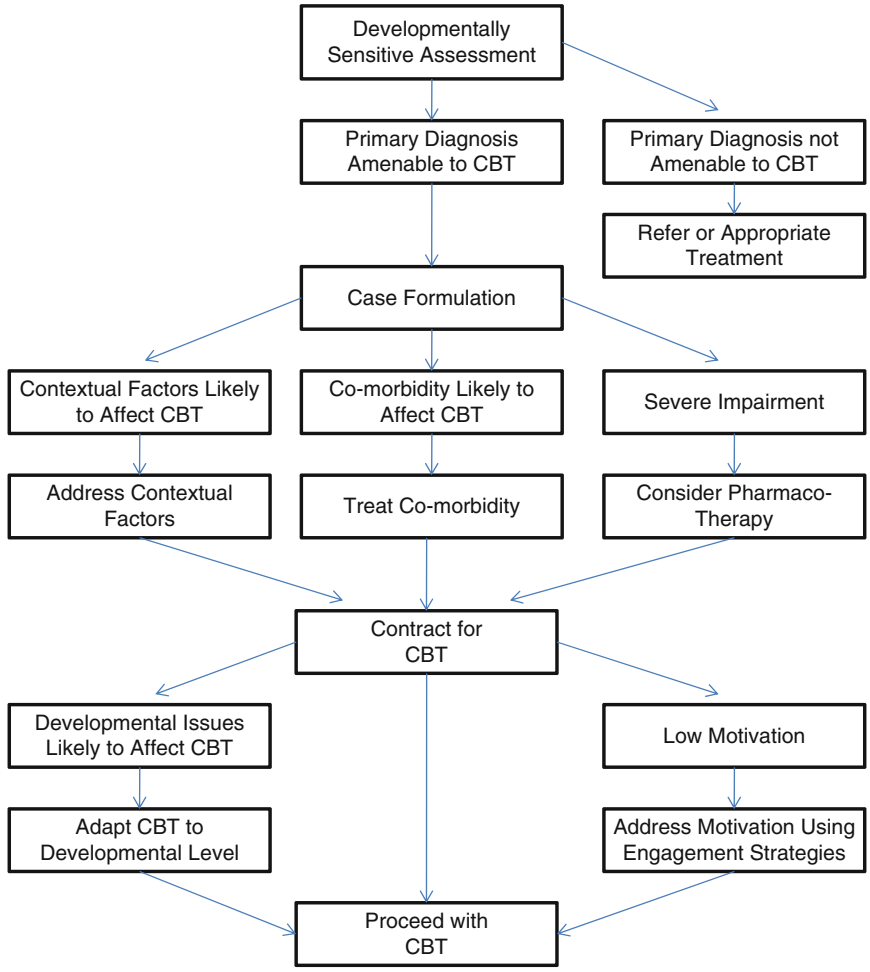

Fig. 11.1 Flow Chart of Treatment Approach

itarian stance toward the adolescent. By eliciting and considering the adolescent's ideas throughout therapy, the therapist shows respect for those ideas and for the adolescent's emerging autonomy. If a younger adolescent often looks to the therapist for guidance, a more directive stance may be warranted, especially if parents report that the adolescent is not yet asserting his or her autonomy at home. In most cases though, adolescents appreciate the opportunity to have their opinions heard. Therapist language should also be adjusted to avoid "talking down" to teens. For example, asking "How do you get along with people at school?" may be more appropriate than asking "How do you get along with kids at school?" as "kids" is a term usually associated with younger children. A benign but frank attitude is also helpful, as adolescents generally react negatively if they perceive the therapist as insincere (Sauter et al., 2009).

Engaging adolescents in therapy may also require one or more initial sessions to build motivation and define their therapeutic goals. Motivational interviewing questions (e.g., "How would your life be different if this problem were solved?" or "What do you hope to get from therapy?") may be useful. These questions may also help distinguish the adolescent's goals from those of his or her parents or of the therapist, allowing the development of a mutually agreed upon agenda for therapy.

Other engagement strategies are designed to make CBT seem relevant to adolescents' developmental needs. Adolescents may appreciate more detailed psychoeducational information on anxiety, emphasis on coping strategies that are respectful of their grasp of formal operational thought (e.g., use of the evidence to test hypotheses about certain cognitions), being given choices about the therapy (e.g., choosing a name for it; Kendall, Choudhury, Hudson, & Webb, 2002), relating CBT principles to their individual interests, and use of age-appropriate rewards (e.g., extra time with friends, "screen" time (e.g., computer, videogames), and gift certificates; Kingery et al., 2006). An emphasis on peer-related and other interpersonal situations can also increase an adolescent's interest in CBT (Scapillato & Manassis, 2002), as can the use of computers and other interactive media. By contrast, assigning written homework between sessions is sometimes perceived by adolescents as alienating. Given that some practice of CBT strategies outside sessions is essential, therapists working with youth may prefer to place greater emphasis on experiential exercises between sessions as opposed to written homework.

The Role of Context and Comorbidity

A thorough case formulation needs to include contextual factors, comorbidities, and developmental factors so that these can be addressed. Because transparency is a hallmark of CBT, therapists are advised to share the formulation with adolescents, to the extent that they are able to understand it (Sauter et al., 2009), and their parents.

Anxious adolescents' family, school, and social environment must be understood in detail, as these environments provide the context for therapy. Parental expectations of the adolescent and of therapy may need to be adjusted, as some parents' expectations are unrealistically high (e.g., the idea that the anxious teen should immediately participate in all age-appropriate activities) or unrealistically low (e.g., an anxious parent who feels that their teen could not manage the stress of a graduated exposure exercise; Kingery et al., 2006). Some authors have also cautioned against meeting with parents without the teen (as teens may resent "feeling talked about"), yet this problem does not occur in all families of anxious adolescents. It may be helpful to give the teen a choice such as "Would you like to be here when I talk to your parents?" and respect his or her wishes on the matter.

The optimal role of parents in CBT with adolescents is unclear. In younger children, parents often "coach" the implementation of new coping strategies outside the office, but most adolescents would consider this condescending. It has been suggested that parents are used more as "consultants" by teens in therapy or sometimes merely as "chauffeurs" (i.e., providing transportation to facilitate exposure and attendance of appointments). A sensible approach may be to assess the adolescent's need for autonomy at the start of therapy and involve parents accordingly (Sauter et al., 2009). Moreover, despite the fact that parents often feel helpless to change their teens' behavior, they can still model adaptive coping strategies, assist with problem-solving when the teen wants or needs this, encourage and support exposure to anxious situations, and set helpful behavioral limits for the teen when needed. Educational materials for parents of anxious adolescents (e.g., Foa & Andrews, 2006) can further enhance their ability to support therapeutic progress.

As mentioned, the transition to high school can pose new challenges for many anxious youth, and many anxieties manifest in the school setting. Unfortunately, high schools are often more difficult to involve in treatment than elementary schools, given that students typically rotate among multiple teachers and classrooms. Smaller high schools are sometimes less overwhelming for adolescents with anxiety disorders, but the willingness of school leadership to support the adolescent's therapeutic goals may be a more critical factor. School avoidance can be a particularly challenging issue in adolescence and usually requires an individualized treatment plan (see Layne et al., 2003). On the other hand, offering school-based CBT programs may be helpful

for adolescents with mild anxiety disorders or subclinical anxiety symptoms (Christensen, Pallister, Smale, Hickie, & Calear, 2010) and less stigmatizing than treatment in a clinic setting.

Although adolescents' identification with peers can heighten their sensitivity to peer criticism, it can also be useful in therapy. Sometimes, a close peer can be engaged in accompanying the adolescent to certain exposures, if the adolescent feels comfortable acknowledging his or her anxiety to the peer. CBT treatment groups involving peers can sometimes engage adolescents in treatment that would otherwise be difficult to engage in individual therapy. The universality of groups (members knowing that they are not alone) is also appreciated by adolescents, and members are often more receptive to suggestions from other members than from a therapist, facilitating therapeutic progress (Scapillato & Manassis, 2002).

Optimally addressing comorbid diagnoses may require some planning. Sometimes, a comorbid condition must be treated before the teen can benefit from CBT. For example, severe substance abuse may interfere with a teen's ability to attend appointments consistently and may be associated with cognitive impairment that interferes with use of CBT strategies. Significant family turmoil can also interfere with consistent treatment and may therefore need to be prioritized. Often however, comorbid conditions can be addressed concurrently. This can be done by combining different treatments (e.g., combining CBT for an anxiety disorder with stimulant medication for attention deficit hyperactivity disorder), by using a CBT program relevant to both conditions (e.g., programs that address both anxious and depressive symptoms; Manassis, Wilansky-Traynor, Farzan, Kleiman, Parker, & Sanford, 2010), or by providing CBT modules that address key elements of both conditions (e.g., activity scheduling, cognitive restructuring, relaxation, and exposure modules for anxiety disorder with comorbid depression). A modular approach to anxiety disorders in children and adolescents is further described by Chorpita (2007). Sample CBT modules are provided, with emphasis on careful assessment to determine which modules are appropriate in a given case and how they should be incorporated into the overall treatment plan (Chorpita).

Adapting Therapy to Developmental Level

Most adolescent CBT programs are based on child CBT programs that have been adapted "upward" or adult CBT programs that have been adapted "downward." Adolescent programs are generally less complex than adult programs. For example, they often avoid multicolumn recording of anxious and adaptive responses to situations found in adult programs. However, these programs often contain wording and strategies that are more sophisticated than in child-focused ones. Sauter et al. (2009) and Kingery et al. (2006) have described some adolescent-specific adaptations of CBT in more detail than outlined here.

Affective, behavioral, and cognitive adaptations are all important factors in creating CBT program for adolescents. Accurate recognition of affect in oneself and others is a basic requirement to do CBT. Many adolescents already have some ability to do this before starting therapy, so the time spent on affect recognition exercises is often shorter than in child-focused programs. Relaxation strategies to address anxious affect, however, may be helpful at all ages. Adolescents can often understand the rationale for these exercises in more detail than younger children. For example, they can be helped to understand sympathetic and parasympathetic nervous system responses rather than just agreeing to do "belly breathing." They may also be amenable to more complex emotion regulation strategies than younger children (Kingery et al., 2006), such as mindfulness-based strategies. For example, an adolescent with good metacognitive skills can often identify "worried thinking" and deliberately disengage from it, instead of challenging specific worries. In addition to relaxation/mindfulness strategies, adolescents can also learn other aspects of self-soothing, such as regularly engaging in physical exercise, talking to friends, listening to calming music, and avoiding self-

medication with illicit substances. These strategies may be particularly helpful for those with concurrent depressive symptoms.

Exposure exercises in adolescents require personal motivation, as parents are generally not able to "force" adolescents to engage in them. Explaining the rationale for exposure in adult language (e.g., using terms such as "hierarchy" or "habituation"; Kingery et al., 2006) and collaboratively developing the specific exercises to be tried show respect for the adolescent's emerging autonomy and can sometimes enhance motivation. Then, the therapist can ask the adolescent "How could your parents help with this?" and discuss possible parental involvement rather than unilaterally assigning a role to parents. Peers can sometimes be involved as helpers too if the adolescent is comfortable confiding in them. Despite adolescents' greater maturity, their anxieties may require that they start with tasks ordinarily required by younger children. For example, a socially anxious adolescent may need to start with ordering food at a restaurant rather than asking someone out on a date. The adolescent's baseline functioning is used as a guide to the first few exposure tasks such that the first task should be the consistent practice of a situation that the adolescent is already managing occasionally.

Cognitive techniques that are most suitable for a given adolescent depend on an accurate assessment of cognitive capacity. Sauter et al. (2009) recommend using a standardized scale such as the Self-reflection and Insight Scale for Youth (Sauter et al.) and matching the cognitive techniques used to the result. They caution, however, that techniques are context-dependent, so sophisticated techniques should not be used when the adolescent is in a highly emotional or challenging situation. Other authors have used cartoon bubbles or questions such as "What went through your mind?" to gage the adolescent's capacity for reflection and access to his or her thoughts (Kendall et al., 2002). Another option is to test the adolescent's capacity for CBT by demonstrating a sample CBT exercise and asking him or her if it makes sense. If the adolescent can understand the sample exercise when modeled by the therapist, he or she is likely to be able to engage in CBT successfully. In general, less cognitively sophisticated adolescents will require more behavioral and fewer cognitive strategies and concrete reminders in order to use cognitive strategies consistently. More cognitively sophisticated adolescents can benefit from both cognitive and behavioral strategies.

As with all aspects of CBT, a collaborative, respectful approach is needed to engage adolescents in cognitive work. In recognition of the adolescent's cognitive maturation, the therapist must avoid simplistic language, use age-appropriate materials, and encourage the adolescent's own ideas. Use of culturally sensitive materials can show respect for the adolescent's heritage and his or her emerging identity. Examination of the evidence for or against a particular thought or assumption may be helpful, as adolescents are often skeptical and welcome such an empirical approach. Complex thought challenges (e.g., examining the pros and cons of several perspectives) may also be possible (Kingery et al., 2006). Use of computer-based learning and other technology is a common part of adolescents' school and social experience and may therefore be welcome when used in CBT.

The Role of Medication

Selective serotonin reuptake inhibitor medications (SSRIs) are often used in combination with CBT in adolescents with anxiety disorders. Although studies suggest therapeutic benefits in using SSRIs and in combining CBT and SSRIs in young people with anxiety disorders, they encompassed a broad age range that included children in addition to adolescents (Compton et al., 2010; RUPP, 2001). Accordingly, adolescent-specific studies are needed. Given the generally higher levels of impairment in adolescents with anxiety disorders, adolescents are more likely to receive SSRIs than younger children.

As with CBT, a collaborative, respectful approach is needed with respect to medication in adolescents. Adolescents will usually take medication only if they feel they have been

consulted in medication-related decisions. They often want more detailed information on potential benefits and potential side effects than younger children and often look up information (or misinformation) about various medications on the Internet. Prescribing physicians may need to help them evaluate the quality of various types of evidence so that therapeutic decisions are based on accurate information. If CBT and medication are provided by the same professional, there may be some value in stabilizing the medication before starting CBT so that the adolescent can see the benefit of his or her efforts in CBT, not just the benefit of medication. If two professionals are involved (one prescribing medication, the other providing CBT), close collaboration between them is essential so that the two therapeutic modalities can complement each other. Such collaboration also ensures that the adolescent does not receive mixed messages or pit one professional against the other (as some are inclined to do).

Case Study

To illustrate some of the above concepts related to anxiety disorders in adolescents, we now consider the case of Carlos. Carlos was a 14-year-old boy who presented to our clinic because he missed several weeks of school after suffering a panic attack in class.

Carlos had been seen previously in our clinic at age 10. At that time, he was diagnosed with generalized anxiety disorder and a specific learning disability. He had already been started on a serotonin-specific medication (fluoxetine) by his pediatrician, which had resulted in some decrease in anxiety symptoms. We recommended participation in an individual CBT program based on "Coping Cat" (Kendall, 2006), with minor modification in view of his learning disability. Academic modifications and supports were recommended to the school. Carlos responded well to the CBT program and showed a further decrease in anxiety symptoms. He also became more confident and more engaged in his school program.

Carlos' current symptoms began shortly after the start of high school, an important transition point for many adolescents. He attended a large public school (over 2,000 students), which he found quite overwhelming. His parents had made sure that appropriate academic supports were available to Carlos at the new school, but he was not making use of these. He said he felt embarrassed about leaving class or staying behind after school to go for extra help. He reported that, in his peer group, it was more socially acceptable to be "not very smart" than to go for extra help. Consequently, his grades were poor, and his teachers considered him unmotivated.

At home, Carlos' parents had different opinions about their son. His mother understood his embarrassment about going for extra help but still encouraged him to go. She worried, however, that his panic attacks might become life-threatening or that he might become depressed or suicidal if forced to go to school despite his anxiety. Carlos' father, by contrast, considered his son undisciplined and didn't think his anxiety should be an excuse for school failure. "He just needs to take the initiative more" was his father's view. Both parents agreed, however, that apart from his school difficulties, Carlos was well-behaved, becoming more responsible (e.g., looking after his younger brother on occasion), and not associating with peers who were antisocial or abusing substances. He struggled to leave the house independently though, fearing he would have a panic attack on the street. His mood had also appeared more downcast in the previous couple of weeks.

Carlos himself thought that his anxiety occurred because his medication was no longer working. He had been prescribed 10 mg per day of fluoxetine at age 10 years by his pediatrician and had remained on this dose ever since. Carlos vaguely remembered his CBT program and continued to do some deep breathing when anxious, but did not practice any other CBT skills. He recalled "I used to just tell myself it would be OK, but that doesn't work anymore." He had also developed a habit of looking up possible causes for his physical symptoms of anxiety on the Internet, which usually made him more anxious.

He often worried about these symptoms, about his friends' opinions of him, and other subjects.

After a thorough diagnostic assessment, we concluded that Carlos suffered from Panic Disorder with agoraphobia and Generalized Anxiety Disorder. He continued to have a significant learning disability. He had some depressive symptoms related to his recent school problems, but these were not severe enough to warrant a diagnosis of Major Depression. His self-esteem was clearly deteriorating though, suggesting that he was at risk for depression if his struggles with anxiety continued.

Carlos' medication, although helpful to him at age 10 years, was now clearly inadequate in view of his physical development since then. He was at least six inches taller and 50 pounds heavier than he had been at age 10 years so definitely needed a higher dose. Carlos' CBT skills were also no longer adequate, as his cognitive development since age 10 years made vague, general reassurances like "It will be OK" seem silly now. He needed to learn more sophisticated coping strategies. He also needed to learn how to evaluate the quality of the evidence he found on the Internet, so that this did not become a further source of anxiety. His parents indicated that they had already tried to discourage Carlos from looking up symptoms online, but their efforts had been futile.

To address these issues, we agreed with Carlos that neither his medication nor his CBT strategies were working any more, and we offered to do something about that. Thus, by validating his own description of the problem, we were able to engage Carlos in further treatment. We increased Carlos' medication dose and scheduled a few sessions to "update" his coping strategies. After these sessions, Carlos liked and trusted his therapist and was then amenable to addressing his Internet habit, including learning how to evaluate evidence he found online.

Most urgently, Carlos needed to find a way to return to school and obtain the academic support he needed without fearing he would be socially ostracized. Realizing that this was unlikely to occur without parental support, we talked with Carlos' parents about his dilemma. His father needed to understand that his son could not simply "take the initiative" but needed treatment for his anxiety and encouragement to return to a place (i.e., school) where he had once experienced extreme fear. His mother needed to understand that Carlos' anxiety attacks were not life-threatening and that his risk of depression was actually lower if he faced his fears by going to school than if he remained at home. Once both parents were able to empathize with Carlos' difficulty but confidently encourage him to leave the house, he was willing to walk on the street again with a friend. By enlisting the help of a friend, we were able to use Carlos' adolescent focus on his peer group to help rather than hinder his progress. Parental accompaniment (often used in younger children) would have been embarrassing to Carlos, but he really valued his parents' encouragement and faith in him.

Returning to school was something Carlos clearly considered more difficult than walking down the street. Therefore, respecting his need to participate in therapeutic decisions, we agreed to have him desensitize to walking down the street first. Within a couple of weeks, he had mastered this task. His panic attacks were also subsiding in response to his increased medication dose and the psychological interventions with Carlos and his family. Carlos still hesitated to return to school, however, fearing his peers' reaction to his school absence and to his need for extra help. We role-played with Carlos some simple responses to questions his peers might ask about his absence, and he found this reassuring. Unfortunately, we were not successful in applying this approach to his fear of peer reactions when seeking extra help. Unable to overcome this obstacle with Carlos, we approached his parents about seeking private tutoring for their son outside of school. After this was arranged, Carlos was willing to try going back to school for part of the day. In consultation with his school, we were then able to gradually reintegrate Carlos into his school program. He attended consistently for the rest of the year. Avoiding Carlos' anxiety about embarrassment in this way might be considered suboptimal

from a CBT perspective, but we felt it was a reasonable compromise as it ensured timely return to school so Carlos could successfully complete his year. Once both his anxiety and his school problems decreased, Carlos became more optimistic and did not need any further intervention for depressive symptoms.

Future Directions

Future studies specific to the treatment of adolescents with anxiety disorders (rather than including adolescents in studies of children of various ages) have been advocated by numerous authors (Field, Cartwright-Hatton, Reynolds, & Creswell, 2008; Kendall & Choudoury, 2003; Silverman et al., 2008; Masia-Warner et al., 2008). Research recommendations that were made in several papers are reviewed below.

Consistent with the ideas in this chapter, the need to tailor CBT to address developmental considerations and to address specific disorders (rather than treating anxiety disorders as a group) was identified (Field et al., 2008; Kendall & Choudoury, 2003; Silverman et al., 2008; Masia-Warner et al., 2008). Furthermore, the influence of developmental factors on outcomes of adolescent-specific CBT may merit evaluation (Sauter et al., 2009). Timing intervention in relation to key points in development such as the beginning of high school (Kendall & Choudoury, 2003) and the use of age-appropriate materials and topics were suggested (Silverman et al., 2008). Group and individual treatment have generally been found equivalent (Silverman et al.); however, it is not clear whether subgroups of adolescents would benefit more from one or the other (Albano & Kendall, 2002).

Many authors advocated studies in community settings with treatments provided by a variety of less specialized clinicians (e.g., pediatric office staff, primary care providers, school personnel; Albano & Kendall, 2002; Ginsburg, Becker, Kingery, & Nichols, 2008; Kendall & Choudoury, 2003; Sherrill, 2008; Silverman et al., 2008; Masia-Warner et al., 2008). Ideally, treatment would be offered in naturalistic settings (e.g., school) potentially improving adolescent engagement and offer broad-based anxiety prevention/early intervention while consistently identifying those requiring more intensive treatment. Client, therapist, and environmental differences and different means of payment from specialized clinics were all cited as potential challenges. Computer-assisted CBT was suggested as an alternative means of increasing treatment availability to adolescents (Khanna & Kendall, 2008; Sherrill, 2008).

Reflecting the clientele in many community sites, studies that included youth with psychiatric comorbidity (Sherrill, 2008) and youth from minority groups (Ginsburg et al., 2008; Silverman et al., 2008) were advocated. Developing functionally equivalent strategies for various cultural groups was also suggested (Silverman et al.). Integrating psychopharmacology into CBT treatment studies was urged, as it would be informative and allow inclusion of a broader range of severity and comorbidity as well (Albano & Kendall, 2002; Kendall & Choudoury, 2003; Labellarte et al., 1999; Silverman et al., 2008).

Parental involvement was advocated, but defining the optimal nature of that involvement was cited as a challenge (Field et al., 2008; Kendall & Choudoury, 2003; Silverman et al., 2008). Examining processes whereby parents influence therapeutic progress was suggested (Field et al., 2008). The possibility that parental involvement may need to differ by age group or by specific problem was raised (Kendall & Choudoury, 2003; Silverman et al., 2008).

Methodologically, several authors recommended active rather than waitlist control groups and longer term follow-up (Adler-Nevo & Manassis, 2009; Albano & Kendall, 2002; Kendall & Choudoury, 2003; Silverman et al., 2008; Masia-Warner et al., 2008). Two groups (Kendall & Choudoury, 2003; Silverman et al., 2008) made more detailed methodological suggestions pertaining to various aspects of measuring outcome, factors moderating or mediating outcome, improved handling of non-completers and missing data, and broadening outcomes to include the sequelae of adolescent anxiety (e.g., substance abuse).

Conclusion

In summary, treatment of adolescents with anxiety disorders must be sensitive to the developmental and social influences in this age group. Factors to consider include level of physical, cognitive, and emotional maturity; the adolescent's need for autonomy; determining the appropriate role of parents and peers in treatment; presence of comorbid diagnoses; anxiety-related impairment; societal expectations; and challenges associated with developmental transitions. Given these complexities, a careful case formulation is essential in order to best tailor CBT to the adolescent's needs. Particular attention must be paid to strategies that enhance adolescent engagement in therapy and that are appropriate to the adolescent's level of cognitive and emotional development.

Many directions for further research have been suggested as have many methodological improvements relative to existing studies. Therefore, researchers must prioritize their goals for future studies. They face the challenge of designing studies that are comprehensive, methodologically rigorous, and feasible in community as well as academic settings. Such studies are important, however, if we are to reduce the burden of suffering in anxious adolescents and return them to a healthy developmental trajectory toward adulthood.

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Social Anxiety and Socialization Among Adolescents

12

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Social anxiety disorder is a condition characterized by abnormal fears of social situations and is one of the most prevalent psychological problems among adolescents (American Psychiatric Association [APA], 2000). The disorder typically emerges in adolescence, with average age of onset between 12 and 16 years of age (Rapee & Spence, 2004; Schneier, Johnson, Hornig, & Liebowitz, 1992; Silverman et al., 1999; Strauss & Last, 1993). Without treatment, social anxiety typically runs a chronic course (APA, 2000; Turner & Beidel, 1989; Wittchen, Stein, & Kessler, 1999).

The terms social anxiety disorder, social phobia, and social anxiety are often used interchangeably in research literature, even though they may have different connotations. Social anxiety disorder and social phobia refer to clinically significant features that meet specific diagnostic criteria set forth by the *Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition* (DSM-IV) (APA, 2000) and are defined as a "marked and persistent fear of one or more social or performance situations" (APA, 2000, pp. 450). The term social anxiety typically serves as a more general term for which the presentation of social anxiety may not meet diagnostic criteria for social anxiety disorder/social phobia, though fear of social situations and interference with daily functioning are still present. For the sake of conciseness, this chapter will refer to these phenomena as "social anxiety disorder" from this point forward (unless specified). Social anxiety disorder is categorized into two subgroups, generalized and non-generalized. Social anxiety disorder is specified as generalized when an individual's fears occur during most social situations (APA, 2000), whereas non-generalized (also known as performance-based, circumscribed, or specific social anxiety) denotes a fear of a single performance situation and/or some, but not most, social situations (APA, 2000). Music performance anxiety and reading aloud in front of a class are examples of these non-generalized, specific situations. The debate over the usefulness of these subgroups has raised questions as to whether social anxiety disorder can be understood as a continuum with different levels of severity and presentation or as a categorical perspective of either meeting criteria or not (Bögels et al., 2010; Marmorstein, 2006). Another controversial topic within the conceptualization of social anxiety is the potential classification of non-generalized (performance-based) social anxiety as a specific phobia rather than simply a subtype (Bögels et al., 2010).

Within the child and adolescent population, studies have suggested that 1% meet diagnostic criteria for social anxiety disorder at any time for males and females (Beidel, Turner, & Morris,

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1999; Kashani & Orvaschel, 1990). However, this percentage may underestimate the true prevalence of the disorder in youth because many studies are based on an outdated diagnostic criteria system of social anxiety disorder that specifically excludes public speaking from the diagnostic category. Speaking or reading in front of a group is one of the most common social fears in adolescents, with percentages of adolescents with social anxiety disorder endorsing this specific fear as high as 90% (Beidel et al., 1999). Subclinical rates (i.e., symptoms that fall short of meeting diagnostic criteria) of social anxiety disorder are significantly elevated. For example, one study found 22% of 8-year-olds, 46% of 12-year-olds, and 56% of 17-year-olds reported fears associated with social situations (Kashani & Orvaschel, 1990). Adolescence is a time of social comparison and beliefs that others are evaluating oneself (Piaget, 1958). During adolescence, self-awareness and self-consciousness continue to develop, and shyness and withdrawal often begin to be perceived as more problematic by peers (Hymel, Rubin, Rowden, & LeMare, 1990). As youth with social anxiety disorder continue to develop cognitively, they begin to increase their abilities to see others' perspectives and compare themselves with others, potentially increasing any preexisting social evaluative fears (Morris, Hirshfeld-Becker, Henin, & Storch, 2004). Avoidance of social interactions and anxious behaviors during social and school situations may adversely affect overall social functioning and development, and negative experiences may increase anxiety regarding future social interactions (Inderbitzen, Walters, & Bukowski, 1997; Rubin & Burgess, 2001). Thus, difficulties with socialization may serve as both a cause and a consequence of social anxiety disorder.

Beyond socialization difficulties, youth with social anxiety are likely to experience difficulties in academic and future occupational functioning, making this disorder a frequent impairment into and throughout adulthood. For example, children and adolescents with social anxiety often have poor academic performance that is coupled with difficulty attending school (Beidel & Turner, 2007; Kingery, Erdley, Marshall, Whitaker, & Reuter, 2010; Mychailyszyn, Mendez, & Kendall, 2010; Seipp, 1991). Symptoms of anxiety in childhood have been found to significantly predict poorer standardized achievement scores (Ialongo, Edelsohn, Werthamer-Larsson, Crockett, & Keliam, 1995), and teachers subjectively rate children with social anxiety children lower in academic performance than their peers (Strauss, Frame, & Forehand, 1987).

Additionally, academic problems associated with social anxiety often persist into adulthood, with approximately 90% of college students with social anxiety reporting academic difficulties such as poor grades, infrequent class participation, avoidance of classes with public speaking requirements, and decisions to not attend graduate school (Turner, Beidel, Borden, Stanley, & Jacob, 1991). Beyond academics, individuals with social anxiety also tend to have problems in other realms (e.g., occupational, addiction) that persist into adulthood. The small body of literature exploring the relationship between social anxiety and occupational functioning suggests that individuals with social anxiety exhibit occupational difficulties, including problems obtaining employment, accepting job offers, and receiving promotions (Stein, Torgrud, & Walker, 2000). Furthermore, adults with social anxiety are significantly less likely to initiate conversations and engage in interactions with coworkers and report greater hardships in work relationships (Yeganeh, 2006).

The complex relationship between socialization and social anxiety symptoms has implications for understanding the manifestation of social anxiety disorder, as well as the development of appropriate and effective interventions for youth presenting with its symptoms. This chapter will provide a brief overview of social anxiety disorder among adolescents. Next, we will discuss the relationships among social anxiety disorder, social development, and social experiences during adolescence. Finally, we will conclude with a discussion of the implications for treatment of this disorder among adolescents and the presentation of a case study to illustrate these concepts.

Physical, Cognitive, and Behavioral Symptoms

Symptoms of clinical and subclinical adolescent social anxiety disorder are usually classified into three categories: physical/somatic, cognitive, and behavioral. However, the boundary between clinical and subclinical presentations of social anxiety has been controversial in recent literature. The DSM-III-R and DSM-IV do not provide clear guidelines for distinguishing between clinical and subclinical presentations of social anxiety (Stein, 1995), often making diagnostic decisions difficult. Social anxiety is prevalent in the general population (Stein & Walker, 1994), and those with subclinical levels of social anxiety close to the diagnostic cutoff often present with equal levels of disability (Stein, 1995). Thus, due to the lack of specific diagnostic thresholds, clinicians are often forced to make a full diagnosis of social anxiety disorder based on other subjective factors. Some researchers have attempted to clarify the clinical versus subclinical distinction by viewing anxiety in children and adolescents on a continually changing trajectory over time (Weems, 2008; Weems & Stickle, 2005). One way to view this trajectory is to redefine how we diagnose social anxiety disorder according to the DSM-IV (Weems & Stickle, 2005). By casting what these colleagues refer to as a wider "nomological net," children experiencing social anxiety could have clinical diagnoses based both on symptoms and mechanisms of anxiety. This would allow for more precise classification of types of social anxiety among adolescents without a strict two-dimensional view of a child having either clinical or subclinical social anxiety. This view suggests that most youth have varying levels of anxiety throughout their development, which is likely to fluctuate in severity and impairment based on continually changing biological, social, environmental, and other factors (Weems, 2008). In other words, while some core characteristics of social anxiety may remain stable and continuous for anxious adolescents, other symptoms are likely to fluctuate in clinical severity across time. The clinical versus subclinical debate is likely to persist as researchers continue to more accurately define social anxiety among adolescents. Regardless, symptom presentation among youth with social anxiety disorder has important implications for case conceptualization and treatment, as the various types of symptoms may affect the social functioning of adolescents in different ways.

Social anxiety disorder can include a wide variety of somatic symptoms, including nausea, sweating, heart palpitations, choking, fainting, headaches, stomachaches, and panic attacks (Beidel et al., 1999; Beidel, Christ, & Long, 1991). Adolescents who experience somatic symptoms of anxiety may interpret threat in social situations (thus, linking with cognitive symptoms) or may perceive that they are sick and therefore leave/avoid the social situation (thus, linking with behavioral symptoms).

Cognitive symptoms of social anxiety disorder include expecting to perform poorly, negative appraisal of personal performance, negative self-talk, social pessimism, perceived low social acceptance and self-worth, increased levels of loneliness, low expectations for social performance, and overall, more negative thoughts and less positive thoughts (Alfano, Beidel, & Turner, 2006; Erath, Flanagan, & Bierman, 2007). Table 12.1 contains a review of various empirical studies investigating the cognitive domains associated with social anxiety disorder.

Finally, behavioral symptoms can be classified into three subcategories: social, school, and other behaviors. Social behavioral symptoms of social anxiety disorder include avoiding age-appropriate social behaviors, such as dating and partying; fear or avoidance of situations where scrutiny from others may occur; social withdrawal; social isolation; fewer friendships; and social impairment (Bögels et al., 2010; Ginsburg et al., 1998; La Greca & Lopez, 1998; Sutker & Adams, 2001; Vernberg, Abwender, Ewell, & Beery, 1992). School behaviors of social anxiety disorder include withdrawal, school refusal, and decreased participation in physical, team-based, and competitive activities (Beidel & Turner, 2007; Bögels et al., 2010; Van Roy, Kristensen, Groholt, & Clench-Aas, 2009). Other behavioral signs of social anxiety disorder include crying,

Table 12.1 Recent empirical literature concerning cognitive symptoms for social anxiety in children and adolescents

Study	Sample size (*N*)	Participants	Objectives	Key findings
Alfano et al. (2006)	80	Children ages 7–11 and adolescents ages 12–16	To examine different cognitive phenomena in children and adolescents who exhibit socially phobic tendencies	Socially anxious children and adolescents were more likely to expect to perform poorly and evaluate their performance as more inferior compared to the control groups. Socially phobic adolescents engaged in negative self-talk in social interactions.
Beidel, Turner, and Morris (1999)	72	Children ages 7–14	To depict the clinical syndrome of socially anxious children	Socially phobic children reported extreme loneliness. Compared to the control group, socially phobic children rated higher on neuroticism and lower on extroversion.
Chansky and Kendall (1997)	78	Children ages 9–15	To examine the link between social anxiety and negative social experiences	The anxiety disorder group perceived themselves as less socially competent compared to the control group and had negative expectancies about being accepted by peers.
Crick and Ladd (1993)	338	Children in third grade (M_{age} = 9.5) and fifth grade (M_{age} = 11.4)	Assessment of sociometric status in relation to loneliness, social anxiety, social avoidance, and attribution for social outcomes	Rejected children exhibit higher levels of social distress and loneliness than any other status group. Rejected children were more likely to view peers as the cause for their social difficulties compared to other status groups.
Erath et al. (2007)	84	Adolescents in sixth and seventh grades	Evaluate social anxiety with negative social performance, maladaptive coping skills, and social skill deficits	Socially anxious adolescents exhibited less prosocial behavior and more social withdrawal compared to the control group. The socially anxious adolescents were associated with negative expectations in social performance.
Ginsburg, La Greca, and Silverman (1998)	154	Children ages 6–11	Examine the relationship between social anxiety and children's emotional and social functioning	Highly socially anxious children reported low perceived social acceptance and global self-worth. They also reported more negative interactions with peers compared to lower socially anxious children.
La Greca and Lopez (1998)	250	Adolescents in grades 10 through 12	Measure social anxiety in relation with social support, perceived competence, and best friendships	Highly socially anxious adolescents perceived low general peer acceptance and felt less romantically attractive to others.
Spence, Donovan, and Brechman-Toussaint (1999)	54	Children ages 7–14	Measure social anxiety in relation to self-talk, self-evaluation of performance, and outcome expectancies	Socially phobic children had higher levels of negative cognitions in social tasks, anticipated negative outcomes in social tasks, and evaluated their own performance more negatively as compared to the control group.
Stopa and Clark (1993)	36	Adults	Measure social anxiety in relation to self-talk, self-evaluation to performance, and actual performance	Socially phobic participants reported more negative self-evaluative thoughts and had more negative thoughts on behaviors in social situations as compared to the control groups.

selective mutism, stuttering, limited eye contact, nail biting, and mumbling (Albano, DiBartolo, Heimberg, & Barlow, 1995; Ollendick & Ingman, 2001).

Although symptom presentation will likely vary by adolescent, each of the physical, cognitive, and behavioral symptoms may affect the social functioning of the individual (Langley, Bergman, McCracken, & Piacentini, 2004). The impact of social anxiety disorder on the social functioning and peer interactions in adolescence will be presented in detail in the following section.

Social Anxiety and Socialization

Peers play a critical role in influencing the development of self-concept, health behaviors and norms, feelings of belongingness in school, psychosocial adjustment, and social and risk-taking behaviors during adolescence through interactions, friendships, and romantic relationships (for a review of relevant studies, please see Table 12.2). In fact, imaging studies show that areas of the brain associated with social cognitions and processing continue to develop during adolescence and thus may be shaped, in part, by social experiences (Sebastian, Viding, Williams, & Blakemore, 2010). Thus, engagement in positive peer friendships, social activities, and romantic relationships is critical for psychosocial adjustment and healthy transition into adulthood (Simon, Aikins, & Prinstein, 2008; Waldrip, Malcolm, & Jensen-Campbell, 2008). Youth with social anxiety disorder may not experience the same quality or quantity of positive interactions with peers. That is, the somatic, cognitive, and behavioral symptoms may affect the frequency by which the adolescent interacts with peers, as well as place the individual at risk for future negative interactions with peers. An understanding of the complex interplay of social experiences, socialization, and social anxiety is critical to developing treatment strategies for adolescents with social anxiety disorder.

The relationship between social anxiety and withdrawal can be conceptualized as cyclical in nature (Inderbitzen et al., 1997; Rubin & Burgess, 2001). Behavioral symptoms of social anxiety disorder involve avoiding social situations, leading to a lack of peer interaction that limits opportunities for adolescents to develop and practice important social skills. Unfortunately, because socially anxious youth often have limited opportunities to develop and practice social skills with peers, their skills are likely to continue to lag behind their peers across development, limiting the experience of the benefits friendships can provide (Kingery et al., 2010; Siegel, La Greca, & Harrison, 2009). These deficits in social skills may place the adolescents at risk for being targets of bullying and other forms of peer victimization. Additionally, as overt signs of anxiety become more severe, adolescents experience increased risk for peer victimization (Ollendick & Hirshfeld-Becker, 2002; Siegel et al., 2009; Storch, Brassard, & Masia-Warner, 2003). Finally, negative social interactions may lead to increased social anxiety, lower expectations of social situation performance, and lower self-esteem (Rubin & Burgess, 2001). Figure 12.1 presents a proposed model depicting the cyclical nature of the relationships among social anxiety, social development, and social experiences. Each component of the model is described in detail in the following section.

Decreased Interactions with Peers

One early correlate with social anxiety that tends to be stable across time (often extending into adolescence) is behavioral inhibition (a pattern of withdrawal, avoidance, fear of the unfamiliar, and sympathetic nervous system hyperarousal; Morris et al., 2004). Children with behavioral inhibition tend to approach early school years (e.g., preschool, kindergarten) with reserve, reticence, and quiet watching behavior, particularly when they are with unfamiliar peers (Hirshfeld-Becker, 2010; Hirshfeld-Becker et al., 2008; Kagan, Reznick, & Gibbons, 1989). Although these children are likely unable to articulate fears of social evaluation, they at the least tend to demonstrate debilitating fears of adults and other children that prohibit them from talking to new

Table 12.2 Recent empirical literature exploring how peers influence various aspects of childhood and adolescent development

Study	Sample size (*N*)	Participants	Objectives	Key findings
Brendgen, Lamarche, Wanner, and Vitaro (2010)	201	Children ages 11–13	Examine how friendship experiences (i.e., having no friends, having nondepressed friends, and having depressed friends) relate to depressed mood trajectories in early adolescents	Friendless youth demonstrated a more elevated trajectory of depressed mood than youth who had reciprocated relationships with nondepressed friends. Friendless youth demonstrated a lower trajectory of depressed mood than youth who had depressed friends.
Mackey and La Greca (2008)	236	Females ages 13–18	Examine a model linking girls' peer crowd affiliations (e.g., Jocks, Populars) with weight concerns, perceived peer weight norms, and weight control behaviors	Girls' level of identification with certain peer crowds was associated with girls' self-reported concern and perceived peer concern with weight. Girls' own concern and peer norms were independently related to girls' weight control behaviors.
Masten, Juvonen, and Spatzier (2009)	364	Children in fourth, sixth, and eighth grades	Examine associations among school-based behaviors, perceptions of peer group norms for these behaviors, and inferences of parent values about these behaviors during adolescent onset (when parents and peers compete for influence)	Perceived parent values predicted academic and social behaviors at each grade level. Peer group norms predicted social behavior for all grades, but academic behavior was predicted by peers only for older students.
McIsaac, Connolly, McKenney, Pepler, and Craig (2008)	174	Adolescent couples ages 15–18	Explore associations between conflict negotiation and the expression of autonomy in adolescent romantic partners	Expressions of autonomy were associated with behavior of the self and behavior of the romantic partner. For facilitative and restrictive conflict responses, female autonomy was uniquely associated with her behavior; male autonomy reflected contributions from himself and his girlfriend.
Simon et al. (2008)	78	Children in sixth to eighth grades	Compare characteristics of participants' friends to those of potential romantic partners. Examine how degree of similarity within friend and romantic dyads explains the importance of general and relationship-specific peer selection criteria	Romantic partners' popularity, symptoms of depression, relational aggression, and relational victimization significantly predicted changes in functioning in these areas over time. Of these, only popularity and depressive symptoms were important to partner selection.
Vaquera (2009)	90,000	Adolescents in grades 7 through 12	Explore relationships between friendship formation (e.g., having best friend at same school), school engagement, and belonging among White and Hispanic students	Hispanic students were more likely to be friendless than White counterparts, and Hispanics were also less likely to form friends in school. Both Hispanic and White youth who reported having a best friend also reported lower engagement problems and a higher sense of school belonging. However, only students whose best friend attended their same school reported higher levels of school belonging, suggesting that school belonging is only promoted by friendships within the school.
Waldrip et al. (2008)	238	Adolescents in the fifth to eighth grades	Examine unique contributions of peer acceptance, friendship, and victimization to adjustment	Adolescents who had less peer acceptance, fewer friends, and lower friendship quality had greater teacher-reported maladjustment. Friendship quality buffered against adjustment problems when peer acceptance and number of friends were low.

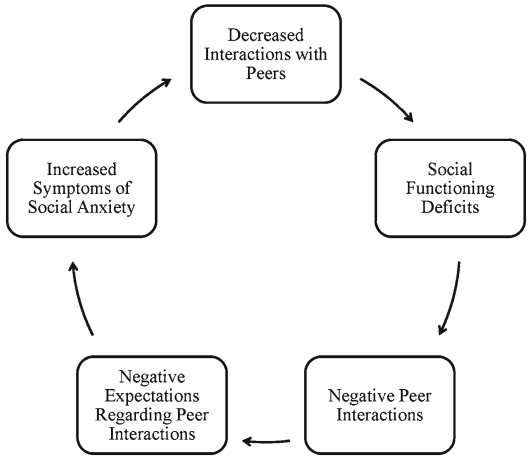

Fig. 12.1 Proposed cyclical model depicting relationships among social anxiety disorder, socialization, and social experiences

adults or peers, developing peer relationships, and going to places where new friends might be made (Morris et al., 2004). As early years are important in socialization, the presence of behavioral inhibition (an early risk factor for social anxiety; Biederman et al., 2001; Hirshfeld-Becker et al., 2008; Morris et al., 2004) could be at least one preexisting trait that leads to socialization problems and social skills deficits in early childhood years. Socialization problems, which may persist into adolescence, may include decreased likelihood of forming friendships that are important to overall development. However, it is important to note that behavioral inhibition does not necessarily lead to social anxiety and that social anxiety, negative appraisals, and social evaluative fears are not always preceded by behavioral inhibition (Morris et al., 2004). Regardless, any behavioral manifestations of social anxiety (e.g., extreme shyness, fear, withdrawal) are likely to interfere with normal social development in both childhood and adolescence. In fact, social skills deficits and negative social appraisals have often been cited as important childhood traits related to social anxiety disorder (Barrett, 2000; Hudson & Rapee, 2000; Ollendick & Hirshfeld-Becker, 2002).

During the adolescent years, behavioral symptoms of avoidance are often characteristic of youth with social anxiety disorder. These symptoms may include decreased classroom performance (e.g., avoiding speaking in class), refusing to attend school, and avoidance of participation in physical, team-based, and competitive activities (Beidel, Turner, & Young, 2006; Bögels et al., 2010; Van Roy et al., 2009). Additional social behavior symptoms include avoiding age-appropriate social behaviors such as dating and partying, fear or avoidance of situations where scrutiny from others can occur, social withdraw, social isolation, fewer friendships, and social impairment (Beidel et al., 2006; Bögels et al., 2010; Ginsburg et al., 1998; Ginsburg & Grover, 2005; La Greca & Lopez, 1998; Sutker & Adams, 2001; Vernberg et al., 1992). Decreased involvement in peer activities and avoidance of social interactions can inhibit friendship formation. Indeed, adolescent females with social anxiety disorder report having fewer best friends and having friendships that are lower in intimacy, companionship, and emotional support (La Greca & Lopez, 1998; Vernberg et al., 1992).

Social Functioning Deficits

Adolescents who are isolated from engaging in social activities show several difficulties with social development due to the lack of contact with peers, as they have fewer opportunities for corrective socialization experiences (Rubin & Stewart, 1996). Socially anxious children and adolescents demonstrate a range of social skills deficits, such as withdrawal and shyness and inappropriate assertiveness and aggression (Inderbitzen-Nolan, Anderson, & Johnson, 2007; Strauss, Lease, Kazdin, & Dulcan, 1989). Furthermore, longitudinal studies have demonstrated that adolescents with social skills deficits experience increased psychosocial problems (including social anxiety) when encountering new stress in their environments (Segrin & Flora, 2000), such as negative peer interactions (e.g., bullying).

Negative Peer Interactions

Behavioral and cognitive symptoms of social anxiety disorder and corresponding deficits in social functioning place adolescents at risk for

negative peer interactions, such as peer victimization (e.g., bullying) and peer rejection (Grills & Ollendick, 2002; Inderbitzen et al., 1997; La Greca & Lopez, 1998; Storch & Masia-Warner, 2004). Specifically, social avoidance and withdrawal, coupled with decreased friendships and deficits in social skills, make children with social anxiety disorder salient targets for aggressive peers. The link between social anxiety disorder and peer victimization may be particularly salient in the middle-school years when peer victimization is most prevalent (Nansel et al., 2001). During the early adolescent years especially, unskilled and withdrawn behavior is likely to invite harassment by peers who view youth with social anxiety as easy targets (Egan & Perry, 1998; Grills & Ollendick, 2002).

Adolescents who are repeatedly victimized by peers tend to report increased symptoms of social anxiety in adolescence and young adulthood (Dempsey & Storch, 2008; Grills & Ollendick, 2002; La Greca & Harrison, 2005; Siegel et al., 2009; Slee, 1994; Storch, Masia-Warner, Dent, Roberti, & Fisher, 2004; Storch, Nock, Masia-Warner, & Barlas, 2003), including fear of negative evaluation (a cognitive symptom of social anxiety; Slee, 1994; Storch, Brassard, et al., 2003; Storch & Masia-Warner, 2004).

Negative Expectations Regarding Peer Interactions

Negative peer experiences in the form of peer victimization and peer rejection may place adolescents at risk for the emergence or exacerbation of symptoms of social anxiety disorder. The mechanism for this link may be the cognitions of the adolescent. For example, negative peer experiences may result in reduced expectations regarding success in future peer interactions, decreased self-efficacy for social relating, and increased fear of negative evaluations (Flanagan, Erath, & Bierman, 2008; Grills & Ollendick, 2002; Inderbitzen et al., 1997). Additionally, peer rejection experienced by adolescents who relocated to a new school led to greater fears of negative evaluation and subsequent social avoidance (Vernberg et al., 1992). Thus, adolescents with social anxiety disorder and a history of negative social experiences may experience disruptions in healthy social processing, such as perceiving threat in social situations that most would interpret as benign and decreased perceptions of self-efficacy.

Treatment Approaches

Because there are several components to the proposed cyclical model linking social anxiety, social development, and peer interactions, treatment approaches for social anxiety disorder and socialization difficulties need to be directed to various parts of the relationship. Treatment approaches should incorporate strategies that directly target cognitive and behavioral symptoms of social anxiety, as well as behaviors and strategies to promote socialization and coping with negative peer experiences.

Cognitive-behavioral therapy approaches incorporate the multiple components into a comprehensive treatment plan for adolescents with social anxiety disorder. In addition, cognitive-behavioral interventions that include social skills training, exposure, and cognitive restructuring, such as the *Stand Up*, *Speak Out* program (Albano & DiBartolo, 2007), have been promising for implementation for youth with social anxiety disorder and a history of peer victimization at reducing symptoms of social anxiety and improving social interaction skills (Berry & Hunt, 2009; Chu & Harrison, 2007; Herbert et al., 2009). Figure 12.2 provides a summary of the various treatment strategies that should be included in comprehensive cognitive-behavioral interventions and depicts how they related to the proposed model of social anxiety disorder, socialization, and social experiences. Each strategy will be reviewed in the following section.

Exposure and Friendship Promotion

Treatment of socially anxious adolescents that experience negative social experiences has showed

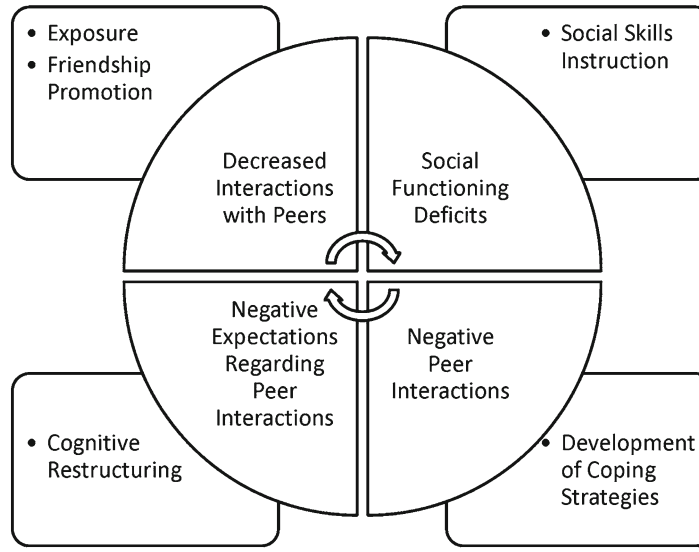

Fig. 12.2 Summary of treatment strategies related to the proposed model of social anxiety disorder, socialization, and social experiences

promising results when the treatment involves the identification and/or development of a social support system (La Greca & Harrison, 2005). Exposure to opportunities in which successful, positive peer interactions are likely (e.g., activities that involve prosocial peers and shared interests) will encourage the development of friendships with same-age peers and challenge the veracity of maladaptive and irrational beliefs (see section on "Cognitive Restructuring"). Thus, encouraging adolescents with social anxiety to identify target peers or activities in which successful peer interactions are likely is a critical component in the treatment of social anxiety disorder. This should include exposing adolescents to activities for which they previously exhibited avoidance behaviors and are likely to be successful with appropriate support and training (Chu & Harrison, 2007).

In addition to providing opportunities for positive interactions with peers via exposure, therapists should also work with adolescents with social anxiety disorder to develop close friendships, as the presence of close friendships may help reduce symptoms of social anxiety and provide a buffer against future negative peer interactions (Hall-Lande, Eisenberg, Christenson, & Neumark-Sztainer, 2007; La Greca & Lopez, 1998). In support of this idea, affiliation with a peer crowd, no matter the status, is associated with reduced symptoms of social anxiety in adolescence (La Greca & Harrison, 2005). That is, peer crowd affiliation and corresponding peer acceptance may provide adolescents with opportunities to develop companionship, which in turn will inhibit social anxiety disorder manifestation (La Greca & Harrison, 2005).

Additionally, close friendships serve as a buffer for adolescents who are exposed to repeated experiences of peer victimization and may actually decrease the likelihood that victimization will happen in the future (Bowker, Spencer, & Salvy, 2010; Davidson & Demaray, 2007; Hodges, Boivin, Vitaro, & Bukowski, 1999; Pellegrini, Bartini, & Brooks, 1999). As socially anxious youth are particularly at risk for being targets of aggressive peers, factors found to build resilience among victims of bullying and peer victimization may be especially important to include in a comprehensive treatment approach. In support of this, one research study indicated that adolescents with social anxiety who had close friendships were less likely to experience loneliness and peer victimization than those without close friendships (Erath, Flanagan, Bierman, & Tu, 2010). Additionally, adolescents with additional friendships (secondary friendships) also reported greater self-efficacy related to interacting with peers.

Social Skills Instruction

Simply presenting adolescents with opportunities for positive social interactions and friendship development may be insufficient for some adolescents with social anxiety disorder, as they may not have the social skills to facilitate positive interactions (Strauss et al., 1989). Existing social skills deficits may inhibit success in such interactions without adequate preparation. Therefore, a critical component of cognitive-behavioral treatment for many adolescents with this disorder is social skills training and rehearsal (Spence, Donovan, & Brechman-Toussaint, 2000). In nonclinical populations, social skills training leads to decreased symptoms of social anxiety and increased self-esteem (Bijstra & Jackson, 1998), though social skills instruction alone is not sufficient for monumental or lasting change. In fact, interventions including exposure are often noted as critical for the treatment of social anxiety disorder (Chu & Harrison, 2007; La Greca & Harrison, 2005).

Development of Coping Strategies

In addition to promoting positive, successful interactions with peers and developing close friendships, therapist should work with adolescents with social anxiety disorder to develop healthy coping with feelings of anxiety and negative peer interactions, such as bullying. Adolescents with social anxiety disorder are more likely to exhibit other comorbid psychosocial problems, such as alcohol and drug use (Amies, Gelder, & Shaw, 1983; DeWit, MacDonald, & Offord, 1999) and depression (Sterba et al., 2010). In addition, adolescents who are exposed to negative peer experiences are more likely to experience negative psychosocial outcomes (including depression and anxiety) when they employ maladaptive coping strategies (Hampel, Manhal, & Hayer, 2009), whereas adolescents who display problem-solving-oriented coping styles are less likely to experience psychosocial problems associated with bullying (Baldry & Farrington, 2005).

Cognitive Restructuring

A final critical component of cognitive-behavioral treatment strategies for adolescence with social anxiety disorder is cognitive restructuring to reduce negative cognitions associated with social anxiety (e.g., fear of negative evaluation, low self-efficacy, and social competence). In a meta-analysis examining the effectiveness of cognitive-behavioral therapy for adolescents with social anxiety disorder, Chu and Harrison (2007) noted that treatment should include modifications of maladaptive thinking and attitudes, identifying thinking errors, Socratic questioning, and developing coping thoughts. Therapists may work with adolescents to challenge automatic and irrational perceptions of social situations as threatening and to instead use self-talk to train themselves to use more adaptive cognitions.

Illustrative Case Study

The problem of social anxiety disorder as it relates to socialization is clearly complex, and treatment must be multifaceted to address multiple issues in the relationship. The following case study describes an adolescent who presented for therapy with one of the authors. Care has been taken to alter details of the case to protect the anonymity of the individual.

Lauren was a 16-year-old student who moved to a new school at the start of her 11th grade year. Her mother referred her for therapy midway through the school year due to difficult interactions Lauren was experiencing with her peers, including social exclusion, rumor spreading, teasing, and mild physical aggression (e.g., pushing her in the hallways). Her mother noted concerns that Lauren begged and cried most mornings (particularly on Mondays) to be excused from school. Lauren's mother allowed her to stay home approximately once per week.

During the first few therapy sessions, it quickly became apparent that Lauren had a history of social isolation. She had only one close friend who had lived in a different city from her for several years. Lauren saw her best friend approximately

once each summer and talked to her intermittently via email. She was not involved in any clubs, sports, or group activities, though did regularly attend private violin lessons and attended a 1-week music camp two summers before with her best friend.

When moving to the new school, Lauren initially received invitations from peers to join them on social activities. However, Lauren told her mother she did not want to go to such activities because she did not really know the other girls and would feel awkward because she did not know what they would talk about. After several declines, the invitations stopped and bullying at school began. Lauren's peers reportedly teased her about her clothes and hairstyle, called her names, did not talk with her at lunch, and threw bits of paper at her during class.

Lauren revealed that she hated to attend school because she expected that her peers would tease her. During class, she did not speak for fear that she would say something wrong that would target her for further bullying. Lunch was particularly difficult for Lauren. Her school had assigned seating at lunch, and Lauren did not talk to her peers sitting near her. She reported feeling so upset at lunch that she would do or say something wrong that she often did not eat and would ask to go to the nurse's office due to nausea. Prior to presenting for therapy, Lauren's distress had become so severe that she was showing signs of depression, including frequent crying, loss of interest in playing her violin, and indicating suicidal ideation.

Lauren exhibited a number of symptoms of social anxiety that were functionally related to her difficulties with social interactions. Behavioral symptoms of Lauren's social anxiety included a clear pattern of withdrawal and avoidance of social interactions, indicative of a generalized subtype of social anxiety disorder. Although it was not possible to determine whether social skills deficits preceded the social avoidance, it was evident that Lauren lacked certain social skills necessary for successful peer interaction (e.g., not accepting social interactions because she did not know what to talk about with peers). Furthermore, her difficulty interacting with peers increased her avoidance of social situations, as evidenced by her refusal to accept social invitations, and prevented her from forming friendships with her fellow students. Thus, instead of befriending her, students at school selected Lauren as a target for bullying. Her ability to cope with the bullying was diminished, as she did not have a strong social support network and her existing coping strategies were insufficient for handling the high level of stress. Finally, the bullying contributed to an exacerbation of her social anxiety symptoms, as she felt even more fearful that she would behave in a way that would cause her to be negatively evaluated, thus leading to heightened behavioral avoidance.

Therapy for Lauren was multifaceted and targeted multiple domains of functioning. First, the therapist provided Lauren with psychoeducation about social anxiety and the types of strategies that would be implemented in therapy, including exposure and cognitive restructuring. Next, the therapist worked with Lauren to review social approach strategies and conversation topics for peer interactions and rehearse these skills with her. Lauren worked with the therapist to identify social settings in which she could implement these strategies and success would be likely. Lauren identified one peer who sat near her at lunch who did not engage in bullying and who had originally attempted to befriend Lauren when she first attended the school. Following successful interactions with this peer (including attending the peer's birthday party), Lauren, the therapist, and Lauren's parents agreed to identify group activities in which Lauren could interact in a structured setting with peers with shared interests. Lauren agreed to join the school orchestra to play the violin (no auditions were necessary). During this time, Lauren's mother began to resist supporting Lauren's behavioral avoidance by not excusing her from school and collaborating with the school nurse to limit the amount of time Lauren was allowed to stay in the clinic.

Cognitive monitoring and restructuring was used during each exposure activity. Strategies for cognitive restructuring included using scripted self-statements prior to engaging in peer interactions and directly challenging maladaptive and

irrational beliefs related to fear of negative evaluations, social competence, and self-efficacy. For example, self-statements included "I am a nice person and a good artist and have interesting things to talk about." She also mentally reminded herself that only a minority of students in her class were mean and engaged in bullying behavior and many students had actually been friendly toward her.

Although bullying behavior did decrease per Lauren's report over the course of the school year and as Lauren began to form friendships (though not yet close friendships) with individuals in the orchestra, Lauren and the therapist identified appropriate coping strategies for when she was bullied. These included removing herself from the situation, using self-talk to remind herself of her positive attributes and positive peer interactions, and engaging in enjoyable activities to avoid rumination over the events.

Lauren participated in weekly therapy sessions and completed therapy assignments when not in sessions over the course of approximately 5 months. At discharge, Lauren continued to experience anxiety related to novel social situations and interacting in large groups of peers, though her avoidance of such situations had significantly decreased. Lauren had formed friendships with two peers, with whom she spent time outside of school, and she had regular, positive interactions with students in the orchestra. She reported that the bullying had decreased, as she spent more time with her friends in school, and her depressive symptoms (avoidance of playing the violin, frequent crying, and suicidal ideation) were no longer present.

Conclusions and Future Directions

In conclusion, symptoms of social anxiety disorder can negatively affect the socialization of adolescents by limiting opportunities to engage in positive interactions with peers and placing individuals at risk for being targets of peer victimization and rejection. In turn, negative experiences with peers may exacerbate symptoms of social anxiety by confirming maladaptive cognitions, such as fear of negative evaluation and low social competence, and leading to an increase in social withdrawal and avoidance of social interactions. In this chapter, we proposed a conceptual model of social anxiety disorder, socialization, and social experiences to explain these relationships. Although relationships among individual components have been reported, research has not yet been conducted to empirically support the model in its entirety. Future research should examine the appropriateness of the proposed model for explaining the link between social anxiety disorder and socialization in adolescence. In addition, although cognitive-behavioral treatment approaches that target the various components of the model exist, randomized control trials need to continue to be implemented to assess the efficacy of comprehensive cognitive-behavioral therapy with adolescents with social anxiety.

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PANDAS: Immune-Related OCD

13

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Pediatric Autoimmune Neuropsychiatric Disorder Associated with Streptococcus (PANDAS) is a clinical phenotype gaining more interest and research in the pediatric community. It is a syndrome consisting of new onset of neuropsychiatric symptoms that are linked to a group A streptococcal infection (GAS). This syndrome consists of an abrupt onset of symptoms such as obsessive–compulsive features, tics, behavioral and mood changes, and neurologic abnormalities which are episodic and drastic compared to the child's baseline functioning. PANDAS is not a contagious disease, but the infectious trigger GAS is quite common and contagious in the pediatric population. Treatment is based on the child's presentation of symptoms and often involves treating the underlying infectious process. Clinicians need to be aware of this infection-triggered neuropsychiatric disorder as PANDAS research grows and more evidence-based treatments evolve.

Historical Background/Theory

Many publications dating back to the 1920s have supported the relationship between illness and new-onset OCD and tics. One of the first case reports was by an otolaryngologist in 1929 when he postulated a potential cause of tics due to infectious disease with three cases of new-onset tics and sinusitis (Selling, 1929). Sometime later, Kiessling and colleagues noted an increase in the prevalence of tics when group A streptococcal (GAS) infections were prevalent (Kiessling, Marcotte, & Culpepper, 1993). These observations paralleled the clearly established relationship between GAS and Sydenham's chorea (SC), a movement disorder associated with rheumatic fever. Sydenham's chorea, one of the major criteria for rheumatic fever, is characterized by rapid, irregular, aimless involuntary movements of the arms and legs, trunk, and face. Historically, SC has been described to have many psychiatric manifestations as well, especially compulsive behaviors (Grimshaw, 1964). Further research with SC found as many as 70% of SC patients develop obsessive–compulsive symptoms which are indistinguishable from classic OCD (Swedo, 1994; Swedo et al., 1998). It was from the observations related to this research that this immune subtype of OCD arose.

The phenomenon in which a person's antibodies designed to attack foreign material, such as viruses and bacteria, attack one's own body is termed "molecular mimicry." This is a case of "mistaken identity" in that some proteins on the wall of streptococcal bacteria are similar to those found on human tissues. For example, rheumatic fever (RF) is classified as an autoimmune illness in which antibodies to the GAS attack a person's heart valve, joints, skin, and/or brain. Specifically, the autoimmune process proposed for SC is thought to be due to antibodies to streptococcal

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antigens associated with the M protein of GAS that cross-react with the nervous system (Bronze & Dale, 1993). One additional theory is that these antibodies may bind to neuronal receptors to release excitatory neurotransmitters in the brain and disrupt neuronal cell function (Kirvan, Swedo, Snider, & Cunningham, 2006).

OCD, SC, and Tourette's syndrome all have a common anatomical link thought to be caused by a dysfunction in the basal ganglia of the brain and cortical and thalamic sites which suggests a common genetic and immunologic vulnerability may exist in these patients (Murphy, Kurlan, & Leckman, 2010). Neuroimaging has demonstrated structural changes with SC and PANDAS as volumetric MRI studies have demonstrated enlargement of the basal ganglia in both illnesses (Giedd et al., 1995; Giedd, Rapoport, Leonard, Richter, & Swedo, 1996).

Characteristics of PANDAS

A practitioner should begin to suspect PANDAS when evaluating a child who was functioning well, but suddenly has a new dramatic onset or worsening of obsessions, compulsions, and movements that seem to develop over 1–2 days. In addition to OCD, these children will develop a dramatic onset of other behavioral symptoms such as rages, mood fluctuations, separation anxiety, hyperactivity, and oppositional behaviors. One or all of these symptoms can develop overnight and continue to progress over a few days (Swedo et al., 1998). Symptoms of inattention, new academic difficulties, and worsening handwriting have also been reported. The child may also begin to have frequent urination and/or nocturnal enuresis along with nightmares. In addition, there is a motor component to this illness as many children will also have a new-onset tic disorder or severe worsening of a previous tic disorder. This dramatic change in functioning has a significant impact on the child's social life, academic performance, and family interactions (Tables 13.1 and 13.2).

A recent case series of PANDAS in three sets of identical siblings highlighted the fact that this disorder presents differently in children, even those with identical genetics. Each identical sibling in the family was exposed to very similar environments yet had very different symptoms, which suggests epigenetic factors and small variations in the environment may play a significant role in how the disorder manifests in each child (Lewin et al., 2011).

Table 13.1 Criteria for PANDAS as established by the National Institute on Mental Health

Criteria for PANDAS as established by the National Institute on Mental Health
1. Presence of obsessive–compulsive disorder and/or a tic disorder
2. Pediatric onset of symptoms (age 3 years to puberty)
3. Episodic/dramatic course of symptom severity
4. Association with group A beta-hemolytic streptococcal infection (a positive throat culture for GAS or history of scarlet fever)
5. Association with neurological abnormalities (motoric hyperactivity, or adventitious movements, such as choreiform movements)

The association between a temporal relationship of GAS and OCD remains controversial in the medical community but is gaining support with more evidence-based research. The exact prevalence of those with OCD with PANDAS subtype in the pediatric population is unknown. The course of OCD–PANDAS is different compared to classic OCD. With limited longitudinal research, it is difficult to predict prognosis. Anecdotally, some children will become completely asymptomatic and never have a future episode, while others will have frequent exacerbations with or without full remission between episodes. Over time, some children may develop a course that is indistinguishable from classic OCD.

Immune Triggers

GAS is the cause of 15–36% of pharyngitis among children in the United States, and many children, as high as 20%, are asymptomatic carriers of GAS (Pfoh, Wessels, Goldmann, & Lee, 2008). If the child is infected with GAS, the onset of neuropsychiatric symptoms is usually within a few days. If a longer lag period is noted, it could be that the

Table 13.2 Comparison of OCD, tics, and PANDAS characteristics

	OCD	TS/tic disorders	PANDAS–OCD
Typical age of onset	10 years	7 years	7 years or younger
Gender relatedness	Slightly higher prevalence in boys than girls before age 15; female-to-male ratio increases after puberty	2:1 male-to-female ratio	Not well studied but appears to have higher female-to-male ratio than tics and OCD in prepubertal population
Course	Insidious onset; typically unremitting, though some episodic cases reported	Peak severity at age 10; 50% of cases remit by late teens	Dramatic onset; episodic or sawtooth course; long-term prognosis unknown
GAS trigger	Reported; cause uncertain	Reported in some cases; cause uncertain	Proposed association with infection
Comorbidities	ADHD, other anxiety disorders, hoarding, tics, depression	ADHD, OCD, anxiety disorders, depression, ODD/conduct disorder	Academic decline, worsening handwriting, urinary frequency, new-onset ADHD, affective instability; higher rates of comorbid tics

child has a subclinical infection which makes the diagnosis of a preceding GAS infection even more difficult (Murphy et al., 2012). In addition to a sore throat, common symptoms of streptococcal pharyngitis are fever, swollen lymph glands, and enlarged inflamed tonsils. GAS is highly contagious through respiratory secretions and has an incubation period of 2–5 days. Some children may not have the full clinical presentation of pharyngitis but can lead to enough immune activation to still cause neuropsychiatric symptoms (Murphy et al., 2004). PANDAS presentations have also been associated with other infectious agents such as influenza; Mycoplasma pneumonia (Muller et al., 2004), which is commonly known as walking pneumonia; and Borrelia burgdorferi (Riedel, Straube, Schwarz, Wilske, & Muller, 1998), which causes Lyme disease. As an example, in 1994, Swedo described a 9-year-old female who had a dramatic change in her moods, new-onset anxiety, compulsive hand washing, and ADHD which developed after an upper respiratory infection. She improved when treated with plasmapheresis and penicillin (Swedo, 1994). In 1995, four pediatric cases with new or worsening OCD and/or tics were proposed to have an infectious trigger such as pharyngitis, sinusitis, or flu-like symptoms, and the acronym PITANDS (Pediatric Infection-Triggered Autoimmune Neuropsychiatric Disorders) was proposed (Allen, Leonard, & Swedo, 1995). In this series, not all children had a GAS trigger, and as more cases of other infectious triggers are described, it is likely that PANDAS will be considered a subtype of PITANDS.

Evaluation

A careful history is essential to understanding if new onset of neuropsychiatric symptoms has corresponded to any changes in physical health. There is no test to confirm PANDAS as it is a clinical diagnosis. Clinicians should ask about illnesses and sick contacts as many children will have asymptomatic GAS infections or other illnesses that can trigger such a response. There are some tests that will aid with the diagnosis such as rapid strep test and a throat culture for GAS. A positive culture may occur in children without symptoms of infection, and some of these children are considered carriers of GAS. The role of the carrier state is thought to be benign in the risk for RF, but it is unclear if it may play a role of increasing the risk for neuropsychiatric presentations (Murphy et al., 2007). The NIH does not recommend children with PANDAS be treated with a tonsillectomy (Table 13.3).

Without a positive rapid strep or culture, elevated titers (antibodies to GAS) suggest a role for GAS infection as a trigger, but alone, titers are not definitive proof of an inciting infection. Elevated streptococcal titers are common in the pediatric population and indicate that the body has had previous infection or is fighting an infection. The two streptococcal titers tested are antistreptolysin O (ASO) and anti-deoxyribonuclease B (DNAse B). If one suspects PANDAS in a child with very recent onset OCD, the child should have rapid strep test or throat culture. Titers should be tested at the beginning of new-onset psychiatric symptoms and repeated 4–6 weeks later to see if there has been a rise. It is important to see a fourfold increase in titer levels to help support the PANDAS diagnosis, but it has been reported that titers may remain elevated 6 months to a year after infection (Murphy et al., 2004). In addition, there are many variables that will affect titer levels such as the time since infection when the sample is drawn, the child's immune status, the use of antibiotics, and the age of the patient. Younger patients may not mount a sufficient immune response to reach laboratory threshold values and present with normal titers.

Treatment

There are no prevention strategies for PANDAS, but limiting exposure to sick contacts, scheduled vaccinations, and treatment compliance with prescribed antibiotics are recommended. Children diagnosed with PANDAS should be treated with therapies shown to be beneficial for OCD and tic disorders. The standard treatment for pediatric OCD is cognitive behavioral therapy (CBT) alone or in conjunction with a SSRI (POTS, 2004).

Table 13.3 Reference values: normal range

	ASO titer	Anti-DNase B (Todd units/mL)
Adult	<160 Todd units/mL or <200 IU	<85
Child (5–12 years)	170–330 Todd units/mL	<170
Preschool-aged child	100–160 Todd units	<60

With the use of Selective Serotonin Reuptake Inhibitors (SSRIs) in a pediatric population, and more specifically with a PANDAS population, higher rates of behavior activation have been associated with this class of medication, so lower starting doses are advised (Murphy, Storch, & Strawser, 2006). CBT is the treatment of choice for mild to moderate severity OCD. Children with a PANDAS presentation should also benefit from the skills developed during CBT. In an open trial of seven children, ages 9–14 years old, diagnosed with PANDAS, a 3-week intensive family-based CBT program was helpful for treating the OCD. It should be noted most of these children were also on SSRI medication (Storch et al., 2006). The tic component of PANDAS can be treated with standard pharmacologic interventions and habit reversal therapy. The skills developed in CBT and HRT should prove helpful to empower the patient and family in managing symptoms in future recurrences.

For children presenting with OCD in the context of a documented infection, treatment consists of antibiotic therapy targeted toward the identified infectious agent. For confirmed GAS, the antibiotics typically used consist of penicillins, cephalosporins, and azithromycin and will need to be prescribed by a physician. Some children will have dramatic improvement with antibiotic treatment. The reported recurrence rate for PANDAS has been estimated to be close to 50% requiring children to have repeated treatment with antibiotics (Murphy & Pichichero, 2002). There is conflicting data as to if prophylaxis antibiotic treatment is effective and safe in children with suspected PANDAS, and this is a topic that is being further investigated. A few studies have indicated that a possible benefit may exist for the use of prophylactic antibiotic treatment to decrease neuropsychiatric symptoms in patients with suspected PANDAS (Murphy & Pichichero, 2002; Snider, Lougee, Slattery, Grant, & Swedo, 2005). However, prophylactic treatment is not without risk due to the potential of increasing antibiotic-resistant organisms, allergic reactions, and gastrointestinal side effects. If the child is having recurrent GAS infections, the family should be tested to see if they are carriers and are a source of chronic reexposure.

For severely ill children who have a clear diagnosis of PANDAS and have not had symptoms resolve with appropriate antibiotic treatment, intravenous immunoglobulin (IVIG) or plasmapheresis has been shown in a small study to have beneficial effects on obsessive–compulsive symptoms, depression, anxiety, and global impairment (Perlmutter et al., 1999). IVIG is not a benign treatment and must be administered by a specialized team of health-care professionals, and side effects are common such as nausea, headaches, dizziness, and vomiting. This treatment has not been shown to be helpful in those patients with OCD without an infectious trigger, thus implying that PANDAS has an immune-mediated process (Nicolson et al., 2000). Ideally, this type of treatment should be performed in a research setting until risks and benefits are better clarified (Snider & Swedo, 2003). The NIMH in 2011 began a trial examining IVIG treatment for PANDAS to further explore treatment implications.

Case Study

Jake, a 7-year-old boy, presents to his pediatrician after his parents noticed he has begun to blink his eyes, scrunch his nose, and clear his throat repeated during the day and feel it could be allergies. These new behaviors developed and worsened over a few days along with many other behavioral changes. The parents report that 1 week ago he began to wet the bed again, which had not occurred since age 5.

Jake also showed increased urinary frequency to the point he was going to the bathroom a few times every hour. When he was in the bathroom, he felt compelled to wash his hands multiple times while counting to the number ten. He also developed a new severe separation anxiety from his family, and it was a struggle to drop him off at school which he had previously enjoyed. To enter the school, he required his mother and principal to escort him to his classroom. His parents were confused as they report until the prior week that their child was a very easygoing child who loved school and never had any of these odd fears and behaviors. They reported he became a different child over a few days, and no form of discipline or reasoning with him was effective. The parents reported to the pediatrician that Jake did have a sore throat but was eating and drinking well, had no fever, and no one in the home was ill. In the office, he tested positive via rapid strep test. The pediatrician placed him on a 7-day course of amoxicillin, and within a few days, most of his symptoms remitted, and parents felt that they had their "old Jake" back.

Two months later Jake began to have compulsive hand washing and needed to tap twice each time he walked through a door. His handwriting became very messy and large, and again, he began to have eye blinking and separation anxiety from parents. His parents immediately took him to the pediatrician who again did a rapid strep test and found him to be positive for GAS. At this time, the entire family tested for strep, and the older sister was found to be rapid strep test positive but not symptomatic. Both Jake and his sister were prescribed amoxicillin, this time for a 10-day course, and within 1 week, some of Jake's behavior symptoms remitted. Jake's compulsive tapping and hand washing continued and was still problematic in the morning when getting ready for school. At this time, the pediatrician referred them to a child psychiatrist and psychologist for further treatment. He was evaluated, and it was felt that a trial of CBT would be tried first before starting an antidepressant. Jake went through ten sessions of CBT, and his compulsions remitted. His parents were educated about PANDAS and were told that at the first sign of psychiatric symptoms they have to bring him to the physician to be checked for an infectious cause.

Conclusion

Childhood OCD and tic disorders, along with the multiple other neuropsychiatric symptoms associated with infections, are increasingly recognized by pediatric providers. It is imperative that clinicians become aware of the diagnosis of PANDAS and investigate the infectious processes associated with this disorder. Children with PANDAS often have an acute and severe onset of neuropsychiatric symptoms that are impairing. While standard therapies have shown to be helpful such as psychopharmacological medications, CBT, and habit reversal therapy, they will not address the identified underlying infectious process, and medical treatment is warranted. Further research is needed for helping to clarify the PANDAS diagnosis and the benefit of prophylactic treatment for these children as recurrence is common. The PANDAS diagnosis requires clinicians to take a thorough medical history in order to ensure that this subgroup of children is not missed as they require a different evaluation and close follow-up as recurrences are common. Standard therapies are helpful adjuncts once the infectious process has been medically treated.

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Part III

Complexities in Adult Anxiety Disorders

Treatment of Posttraumatic Stress Disorder and Comorbid Borderline Personality Disorder

14

Melanie S. Harned

Borderline personality disorder (BPD) is a severe and complex psychological disorder characterized by pervasive dysregulation of emotion, behavior, and cognition. Individuals who meet criteria for BPD are the quintessential multiproblem clients, often presenting to treatment with multiple comorbid Axis I and II diagnoses, numerous dysfunctional behaviors, and generally chaotic lives. Of the many complex problems exhibited by individuals with BPD, co-occurring posttraumatic stress disorder (PTSD) is among the most common. However, the clinical challenges encountered in the treatment of individuals with BPD can make it difficult to implement PTSD treatments in this population. Indeed, clients with BPD, particularly those with a severe level of disorder, are generally viewed as inappropriate for PTSD treatments. Conversely, BPD treatments include clients with a range of severity but do not typically target their PTSD. Thus, efforts to develop treatments that can safely and effectively address PTSD in this complex client population are clearly needed. The present chapter will begin by reviewing the prevalence, phenomenology, and clinical complexities of individuals meeting criteria for both BPD and PTSD. Next, an integrated BPD and PTSD treatment that combines Dialectical Behavior Therapy (DBT; Linehan, 1993a, 1993b) with Prolonged Exposure therapy (PE; Foa, Hembree, & Rothbaum, 2007) will be described. Finally, a case example will be presented along with suggestions for future research.

The Nature of the Problem

The comorbidity between BPD and PTSD is well documented and some have even proposed that BPD is better conceptualized as a trauma-related condition known as "complex PTSD" (e.g., Herman, 1992). However, this view has been contested on both theoretical and empirical grounds (e.g., Gunderson & Sabo, 1993), and the current diagnostic system considers BPD and PTSD to be distinct though often co-occurring disorders (American Psychiatric Association [APA], 2000). Epidemiologic research has indicated that 30.2% of individuals with BPD are also diagnosed with PTSD, whereas 24.2% of individuals with PTSD also have BPD (Pagura et al., 2010). Within clinical samples, the rate of comorbidity is even higher with approximately 50% of BPD inpatients and outpatients also meeting criteria for PTSD (e.g., Harned, Rizvi, & Linehan, 2010; Zanarini, Frankenburg, Hennen, Reich, & Silk, 2004). Research

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comparing individuals with BPD and PTSD to those with either disorder alone has shown that those with both disorders report more extensive trauma histories and are more impaired in terms of global psychological distress, Axis I comorbidity, emotion dysregulation, and physical health (e.g., Bolton, Mueser, & Rosenberg, 2006; Connor et al., 2006; Harned, Rizvi, et al., 2010; Pagura et al., 2010; Rusch et al., 2007). Moreover, BPD clients with PTSD engage in more frequent non-suicidal self-injury (NSSI) than those without PTSD (Harned, Rizvi, et al., 2010; Rusch et al., 2007), and PTSD increases the risk of suicide attempts in community samples of individuals with BPD (e.g., Pagura et al., 2010). Given these findings, it is not surprising that the presence of PTSD predicts a lower likelihood of remitting from BPD over 10 years of prospective follow-up (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2006).

Several theories have been proposed to account for the high comorbidity between BPD and PTSD. The biosocial theory of the etiology of BPD (Linehan, 1993a) highlights the role of the invalidating environment, which may include childhood abuse and trauma, in the development of BPD. In addition, individuals with BPD may possess certain vulnerability factors that increase their risk of trauma exposure as adults. For example, childhood sexual abuse and childhood emotional withdrawal by a caretaker have both been found to increase the risk of adult trauma among individuals with BPD (Zanarini et al., 1999). Many individuals with BPD also possess a variety of known risk factors for PTSD, such as low social support, poor psychological adjustment, and childhood abuse (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003) that may make them particularly vulnerable to developing PTSD in response to traumatic events. Finally, trauma and PTSD may maintain or exacerbate BPD by, for example, further intensifying the emotion dysregulation that is central to BPD and increasing the frequency of impulsive, self-destructive behaviors such as NSSI (Harned, Rizvi, et al., 2010).

Factors That Contribute to Complexity

Clients with both BPD and PTSD often present to treatment with multiple severe problems that may create significant obstacles to the successful implementation of PTSD treatments. In this section, several factors that may increase complexity and decrease treatment response among BPD clients seeking PTSD treatment are proposed. Although these complicating factors are likely to interfere with any type of PTSD treatment, emphasis will be placed on PE (Foa et al., 2007), the treatment program that has received the most empirical support and is recommended as a frontline treatment for PTSD (Foa, Keane, Friedman, & Cohen, 2009). PE involves imaginal exposure to the trauma memory followed by processing of the client's experience during the imaginal exposure and in vivo exposure to feared but non-dangerous situations. Both types of exposure are designed to promote extinction of maladaptive emotions by disconfirming erroneous perceptions that maintain PTSD (e.g., the world is extremely dangerous and the self is extremely incompetent). PE is based on Emotional Processing Theory (Foa & Cahill, 2001; Foa & Kozak, 1986) that specifies that effective treatments for anxiety disorders including PTSD involve activating the pathological fear structure underlying the target disorder and presenting information that is incompatible with the pathological elements of the structure. In other words, clients are repeatedly exposed to the situations or memories that elicit anxiety or distress in the absence of their anticipated negative consequences so that they can learn that they do not need to avoid these situations or be distressed by them. Thus, anything that interferes with the ability to experience and tolerate distress so that corrective information can be learned will likely reduce the efficacy of PE and exposure-based treatments more generally.

Intentional Self-Injury

Suicidal behavior and NSSI, together referred to as intentional self-injury, are considered hallmark features of BPD. Among inpatients with BPD, more than 70% report a lifetime history of multiple

episodes and methods of NSSI and 60% report multiple suicide attempts (Zanarini et al., 2008). The rate of death by suicide among individuals with BPD is estimated at 8–10% (Linehan, Rizvi, Shaw-Welch, & Page, 2000; Pompili, Girardi, Ruberto, & Tatarelli, 2005). Among individuals with BPD, intentional self-injury most often functions to provide relief from tension and unpleasant emotions, punish oneself, get away or escape, influence others, and generate feelings (Brown, Comtois, & Linehan, 2002; Kleindienst et al., 2008). The increased risk of intentional self-injury among BPD clients with PTSD may be due to a functional relationship between PTSD symptoms and intentional self-injury. Clients with BPD and PTSD are more likely than those without PTSD to report a variety of trauma-related cues for intentional self-injury, including flashbacks, nightmares, and intrusive thoughts about sexual abuse or rape (Harned, Rizvi, et al., 2010). These findings are consistent with research showing that the relationship between childhood sexual abuse and NSSI is mediated by the PTSD symptom clusters of reexperiencing and avoidance/numbing (Weierich & Nock, 2008). Taken together, these findings suggest that the higher rate of intentional self-injury among individuals with both BPD and PTSD may be due to the use of intentional self-injury as a way to cope with the intense negative affect and cognitions associated with PTSD and trauma.

The high rate of intentional self-injury among BPD clients with PTSD may both add complexity and cause anxiety for therapists implementing PTSD treatments in this population. Given that treatments for PTSD often elicit intense emotions and can cause a temporary increase in PTSD symptoms before they eventually improve (Nishith, Resick, & Griffin, 2002), it is understandable that both therapists and BPD clients may be anxious about the potential risk of intentional self-injury during PTSD treatment. This fear of intentional self-injury may make both therapists and clients wary of allowing trauma-related emotions to be experienced in their full intensity. Alternatively, if clients engage in intentional self-injury as a way to escape from intense emotions elicited by exposure, then the opportunity for corrective learning (e.g., that intense emotions can be tolerated) is decreased.

Other Co-occurring Problems

In addition to the high rate of intentional self-injury, many BPD clients with PTSD exhibit a variety of other co-occurring psychological, social, and functional problems. For example, it is not uncommon for clients with both BPD and PTSD to present to treatment with multiple other Axis I and II disorders, a variety of impulsive behaviors (e.g., shoplifting, gambling), chaotic or nonexistent relationships, no or limited source of income beyond psychiatric disability, unstable housing, and several chronic and disabling medical conditions. Faced with a client who reports a multitude of serious problems, it can be difficult for therapists to decide how and in what order these many problems should be targeted. Further adding to this complexity is the fact that many of these co-occurring problems, such as dissociation, substance use, and ongoing trauma, are likely to interfere with the efficacy of PTSD treatments.

Dissociation. The rate of Axis I dissociative disorders among individuals with BPD is quite high (55–72%; Foote, Smolin, Neft, & Lipschitz, 2008; Sar, Akyuz, Kugu, Ozturk, & Ertem-Vehid, 2006) and 68% of inpatients with BPD report moderate to high levels of dissociative experiences (Zanarini, Ruser, Frankenburg, & Hennen, 2000). Among individuals with BPD, those with high levels of dissociation report more PTSD symptoms, traumatic experiences (particularly childhood trauma), and more severe impairment in a variety of other areas (e.g., Brodsky, Cloitre, & Dulit, 1995; Sar et al., 2006). In addition, the presence of a dissociative disorder has been found to enormously increase the odds of having a history of multiple suicide attempts (odds ratio = 15.09), even after controlling for the effects of BPD, PTSD, and alcohol abuse (Foote et al., 2008). Similarly, dissociation predicts an increased likelihood of NSSI among individuals with BPD over 10 years of prospective follow-up (Zanarini, Laudate, Frankenburg, Reich, & Fitzmaurice, 2011). Dissociation can pose a significant challenge to the successful implementation of exposure therapy for PTSD because it functions to escape intense emotions and is likely to interfere with information processing. Several laboratory-based studies

have shown that, compared to BPD clients low in state dissociation, BPD clients with high state dissociation exhibit reduced emotional reactivity during a startle response task (Ebner-Priemer et al., 2005) and diminished emotional learning during an aversive differential delay conditioning procedure (Ebner-Priemer et al., 2009). In addition, clients with PTSD who are high in trait dissociation are more likely than those who are low in trait dissociation to continue to meet criteria for PTSD following PE (69% vs. 10%; Hagenaars, van Minnen, & Hoogduin, 2010).

Substance use. Approximately 60% of clients with BPD also meet criteria for a substance use disorder (SUD; Trull, Sher, Minks-Brown, Durbin, & Burr, 2000), and individuals with both BPD and PTSD are even more likely to meet criteria for a lifetime SUD than those with BPD alone (Pagura et al., 2010). Like intentional self-injury, substance use often functions as a way to regulate negative emotions and cognitions, including those specifically associated with PTSD. Indeed, individuals with BPD are more likely than those without BPD to report using illicit drugs or misusing prescribed medications to control PTSD symptoms (Leeies, Pagura, Sareen, & Bolton, 2010). Recent research has shown that treatments for co-occurring SUD and PTSD that incorporate exposure procedures significantly reduce PTSD without exacerbating SUD (e.g., Brady, Dansky, Back, Foa, & Carroll, 2001; Mills et al., 2012). However, substance use, particularly when it occurs during or immediately following exposure tasks, is likely to interfere with corrective learning by inhibiting emotional engagement and/or preventing a complete test of problematic expectancies. This is supported by research indicating that daily use of benzodiazepines decreases the efficacy of exposure therapy for PTSD (van Minnen, Arntz, & Keijsers, 2002). Of note, clients with both BPD and PTSD are more likely to be prescribed benzodiazepines than clients with PTSD alone (59.3% vs. 10.8%; Connor et al., 2006).

Ongoing trauma. Ongoing abuse and trauma is present in the lives of many clients with BPD and is commonly used as an exclusion criterion for PTSD treatment. Among inpatients with BPD, 90% report a history of abuse in adulthood and 47–60% report new abusive experiences at each 2-year interval over 6 years of prospective follow-up (Zanarini, Frankenburg, Reich, Hennen, & Silk, 2005). In addition, case descriptions of two clients with BPD and PTSD who received the combined DBT and modified PE treatment indicate that both women experienced new traumas during treatment (Harned & Linehan, 2008). These findings suggest that new or ongoing trauma is likely to complicate PTSD treatment in this population.

Emotion Dysregulation

The biosocial theory of BPD proposes that it is the transaction between an emotionally vulnerable biology and an invalidating environment (including childhood abuse and trauma) that leads to the pervasive disruption of the emotion regulation system that is central to BPD (Linehan, 1993a). In this theory, emotional vulnerability is defined as having a heightened sensitivity to emotional cues, increased emotional reactivity, and a slow return to emotional baseline, and research has generally confirmed the presence of these emotional characteristics among individuals with BPD (see Rosenthal et al., 2008 for a review). The emotion dysregulation exhibited by individuals with BPD is further intensified by the presence of PTSD (Harned, Rizvi, et al., 2010) and can complicate PTSD treatment in several ways.

Over-engagement. In PE, the term "over-engagement" refers to excessive emotional distress that renders clients unable to process and incorporate corrective information that is present during exposure (Foa et al., 2007). Foa et al. (2007) identify two types of over-engaged clients: dissociative (e.g., losing the distinction between a memory that occurred in the past and being in the present moment, having body memories or flashbacks) and emotionally overwhelmed (e.g., sobbing or crying throughout imaginal exposure to the trauma memory across multiple sessions). Foa et al. (2007) report that in their extensive clinical experience using PE they have encountered relatively few clients who manifest severe over-engagement.

However, our clinical experience suggests that both types of over-engagement occur with some regularity during exposure with clients with severe BPD, and this is likely due to the heightened emotion dysregulation and dissociative tendencies found in this client population.

Under-engagement. At the other end of the spectrum, the PE manual defines "under-engagement" as difficulty accessing the emotional components of the trauma memory (Foa et al., 2007). During exposure, under-engaged clients may report feeling numb or detached, and distress or anxiety levels are typically low (Foa et al., 2007). Under-engagement reflects a lack of activation of the emotional structure which, according to Emotional Processing Theory, is a necessary condition for incorporating the corrective information that leads to reduction of maladaptive emotions. Indeed, activation (emotional engagement) is associated with better outcomes during PE (e.g., Jaycox, Foa, & Morral, 1998). Under-engagement is also common in clients with BPD and can reflect a general unwillingness to experience intense emotions. For example, clients with BPD are less willing than clients without a personality disorder to tolerate distress in order to pursue desired goals or to approach a potentially distressing situation (Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006) found, both of which are critical to the success of exposure-based PTSD treatments. Of note, it is not unusual for a client with BPD to vacillate between being over- and under-engaged within and across exposure sessions. For example, under-engagement may follow an experience of over-engagement and reflect an intentional effort to suppress emotions due to fear of becoming over-engaged.

Intense non-fear emotions. Consistent with the conceptualization of PTSD as an anxiety disorder, PTSD is primarily viewed as a disorder associated with maladaptive fear. However, BPD clients often report multiple emotions about their traumatic experiences and fear may or may not be primary. Shame is a particularly common and persistent emotion in BPD (Rizvi, Brown, Bohus, & Linehan, 2011) and is sometimes the primary emotional response to trauma among severe BPD clients. In such cases, shame is often accompanied by intense self-hatred as well as rigidly held beliefs of being inherently bad, disgusting, and unlovable. BPD clients with high levels of shame often exhibit a variety of avoidance behaviors that are likely to reduce the efficacy of exposure, such as avoiding eye contact and leaving out the most shame-eliciting details from the trauma narrative.

Trauma Memory Characteristics

The treatment of PTSD among clients with BPD is often complicated by both the quantity and quality of their trauma memories.

Large quantity of trauma memories. While many individuals with PTSD report multiple traumas, individuals with BPD often report particularly extensive trauma histories. Indeed, repeated abusive experiences, multiple types of abusive experiences, multiple perpetrators of abuse, and early age of onset of abuse in childhood have been found to distinguish BPD from other diagnostic groups (Herman, Perry, & van der Kolk, 1989; Ogata et al., 1990; Zanarini et al., 1997), and the majority of individuals with BPD continue to experience traumatic events as adults (e.g., Zanarini et al., 2005). In addition, nearly all clients with BPD report experiences of neglect, emotional and verbal abuse, emotional withdrawal by a caretaker, and/or chronic invalidation (Zanarini et al., 1997), which, despite not constituting "trauma" according to Criterion A of the PTSD diagnosis (APA, 2000), are often reported by clients with BPD to be among their most distressing experiences. The sheer number of traumatic events experienced by clients with BPD is likely to complicate PTSD treatment.

Poor quality of trauma memories. To complete imaginal exposure for PTSD, individuals must remember at least some details of a traumatic event. While many individuals with PTSD have elaborated memories, some have only short or fragmented memories, particularly those whose PTSD is related to childhood abuse. Many clients with BPD are not able to remember enough details about their trauma(s) to create an elabo-

rated narrative, and some have only fragmented memories or brief images of certain events. The degraded memory quality reported by many clients with BPD is likely explained by a number of factors, such as the early age of trauma onset, peritraumatic dissociation, and autobiographical memory overgenerality (e.g., Crane & Duggan, 2009; Eisen & Lynn, 2001). Importantly, these types of brief memories and images often cause significant distress and are frequently reexperienced as intrusive memories, flashbacks, and nightmares. The treatment of individuals with very fragmented trauma memories is often challenging in part due to concerns about the possibility of reifying "false memories" (see McNally, 2003 for a review of this topic).

Treatment Noncompliance

Clients with BPD are often noncompliant with treatment, frequently missing or arriving late to sessions, failing to complete homework, and dropping out of treatment prematurely (e.g., Gunderson et al., 1989). The risk of treatment dropout among clients with BPD may be particularly high during exposure-based PTSD treatments (McDonagh et al., 2005; Zayfert et al., 2005), although one study did not find a relationship between borderline characteristics and treatment dropout (Clarke, Rizvi, & Resick, 2008). Given research indicating that inconsistent treatment attendance is the best predictor of poor outcome in PTSD treatment (Tarrier, Sommerfield, Pilgrim, & Faragher, 2000), BPD clients with treatment compliance problems are unlikely to benefit from PTSD treatment.

Therapist Factors

Just as clients with BPD may possess characteristics that can interfere with the successful implementation of PTSD treatments, so too can their therapists. Relatively few practicing therapists, including trauma experts, have been trained in or use exposure procedures for PTSD (Becker, Zayfert, & Anderson, 2004; van Minnen, Hendriks, & Olff, 2010). In addition, although not supported by empirical data, many therapists believe that exposure therapy for PTSD is contraindicated for more complex clients and is likely to cause increases in suicidality, self-injury, dissociation, substance abuse, PTSD symptoms, and dropout (Becker et al., 2004; van Minnen et al., 2010). Given these common beliefs, many therapists are likely to be hesitant or even unwilling to implement exposure therapy for PTSD with BPD clients. Thus, providing therapists with the training and support necessary to feel confident in deciding when and how to implement exposure therapy for PTSD with clients with BPD is critical.

Summary

In sum, treating PTSD among clients with BPD, particularly those with a severe level of disorder, is likely to be both complex and challenging. Treatments for this population not only need to be able to address the multiple problems beyond PTSD that are common among clients with BPD but also to provide a clear method for determining when and how to address PTSD in the context of a plethora of potential treatment targets. Some problems may be a higher priority than treating PTSD due to safety concerns (e.g., intentional self-injury, ongoing trauma) or because, if untreated, they are likely to interfere with the successful implementation of PTSD treatment (e.g., severe dissociation, substance use, or treatment noncompliance). Further, several complicating factors are likely to arise during exposure therapy with severe BPD clients (e.g., intense shame, over-engagement), which may interfere with the corrective learning that is necessary for the treatment to work. In addition, strategies for addressing multiple and sometimes fragmented trauma memories, including traumatic non-Criterion A events, are needed in this population. Finally, therapists may not have received training in exposure therapy for PTSD and/or may have concerns about the safety and tolerability of this treatment that make them unable or unwilling to implement it with BPD clients.

A Review of Existing Treatment Approaches

Few approaches exist for treating PTSD among clients with BPD, particularly those with recent intentional self-injury. Indeed, the most common approach is *not* to treat their PTSD by either excluding them from PTSD treatments or including them in BPD treatments that do not typically target PTSD.

PTSD Treatments

Historically, it was not uncommon to exclude clients with BPD from PTSD treatments altogether due to clinical lore suggesting that they would be unlikely to benefit and may even decompensate as a result of these treatments. Indeed, the first formal attempt at defining decision-making guidelines for the use of exposure therapy for PTSD included BPD as a condition thought to contraindicate the use of exposure (Litz, Blake, Gerardi, & Keane, 1990). Although it has become less common to exclude clients with BPD from PTSD treatments, this still occurs in some more recent studies (e.g., Speckens, Ehlers, Hackmann, & Clark, 2006). It remains a common practice, however, to exclude clients that exhibit certain behaviors that frequently co-occur with severe BPD. For example, a meta-analysis found that PTSD treatment outcome studies frequently exclude participants due to suicide risk (46%), substance abuse/dependence (62%), and "serious comorbidity" (62%), resulting in a combination of exclusion criteria that is likely to exclude most clients with BPD from PTSD treatment (Bradley, Greene, Russ, Dutra, & Westen, 2005). Thus, the research data on PTSD treatment among clients with BPD is limited both in the number of studies available and the generalizability of the findings to severe BPD clients.

Single-diagnosis treatments. Eight PTSD treatment studies were located that reported including clients with BPD (10–100% of the total samples), but focused only on treating PTSD (i.e., single-diagnosis treatments). Three of these studies found that clients with BPD or borderline personality characteristics (BPC) show similar rates of improvement in PTSD symptoms (i.e., slopes) during CBT for PTSD as clients without BPD/BPC (Clarke et al., 2008; Feeny, Zoellner, & Foa, 2002; Mueser et al., 2008), and one study found mixed results across two BPD case studies (Hendriks, de Kleine, van Rees, Bult, & van Minnen, 2010). Also, one study found that clients with BPD (11%) were less likely than those without BPD (51%) to achieve good end-state functioning, which was defined as being below clinical cutoffs on measures of PTSD, depression, and anxiety (Feeny et al., 2002). In addition, a feasibility study of narrative exposure therapy for women with BPD and PTSD (n=10) found a significant pre-post reduction in PTSD severity (Pabst et al., 2012). Three studies did not report outcome results specific to the BPD clients in the sample (Ehlers, Clark, Hackmann, McManus, & Fennell, 2005; McDonagh et al., 2005; Sachsse, Vogel, & Leichsenring, 2006). Four of the eight studies specified the exclusion criteria that were used, which included many BPD-relevant behaviors such as recent and/or active suicidality (n=4, 100%), substance abuse/dependence (n=4; 100%), ongoing abuse or trauma (n=3, 75%), and recent and/or active NSSI (n=2, 50%). Thus, the generalizability of these results to more severe BPD clients is not known.

Sequential treatments. Three studies evaluating sequential treatments for childhood abuse-related PTSD have reported including individuals with BPD. These treatments each use modified versions of DBT to increase behavioral skills prior to and/or after exposure therapy for PTSD (Bohus, Kruger, Dyer, Priebe, & Steil, 2011; Cloitre et al., 2010; Steil, Dyer, Priebe, Kleindienst, & Bohus, 2011). Two of these studies (Cloitre et al., 2010; Steil et al., 2011) did not report results specific to clients with BPD (24% of each sample). The third study found that clients with BPD (42% of the sample) showed a comparable rate of improvement in PTSD symptoms as those without BPD (Bohus et al., 2011). All three studies excluded clients with recent acute suicidality thereby limiting their generalizability.

BPD Treatments

In contrast to PTSD treatments, BPD treatments have often included the most severe BPD clients but have not specifically targeted their PTSD. DBT (Linehan, 1993a, 1993b) is the most empirically supported treatment available for BPD, and a recent meta-analysis of 16 DBT studies found a low dropout rate (27.3%) and moderate effect sizes for DBT in terms of global improvement and reductions in intentional self-injury (Kliem, Kröger, & Kosfelder, 2010). DBT is a comprehensive, principle-based treatment that allows for simultaneous targeting of multiple disorders and includes a number of protocols specifying how to target common problems in BPD (e.g., suicide crisis protocol, hospitalization protocol). Although the DBT manual recommends the use of exposure to treat PTSD, it does not include a protocol specifying when or how to do this. In addition, the DBT manual warns therapists to be particularly cautious about treating PTSD and suggests that PTSD treatment is likely to increase suicide risk and wreak havoc in the lives of individuals with BPD. Thus, although DBT has been shown to be effective in reducing behavioral dyscontrol among clients with both BPD and PTSD (Harned, Jackson, Comtois, & Linehan, 2010), it has not routinely targeted PTSD itself. In a study of DBT for suicidal and self-injuring BPD women in which PTSD was not routinely targeted, few clients (13%) remitted from PTSD during 1 year of treatment (Harned et al., 2008). Similarly, an effectiveness trial of DBT for individuals with Cluster B personality disorders found that DBT resulted in only a small reduction in PTSD symptom severity compared to treatment as usual (Feigenbaum et al., 2011). Other BPD treatments that have been evaluated in randomized controlled trials (RCTs) have not included PTSD as an outcome and their impact on PTSD is therefore unknown (Bateman & Fonagy, 1999; Blum et al., 2008; Clarkin, Levy, Lenzenweger, & Kernberg, 2007; Giesen-Bloo et al., 2006).

Summary

In sum, the few studies that have examined PTSD treatments among clients with BPD uniformly exclude clients with suicidal behaviors and some use a variety of additional exclusion criteria that limit the generalizability of the findings. The PTSD treatments that have utilized the broadest inclusion criteria (e.g., allowing actively self-injuring clients to receive treatment) have also used more restrictive treatment settings, including inpatient (Sachsse et al., 2006), residential (Bohus et al., 2011; Steil et al., 2011), or intensive outpatient programs (Hendriks et al., 2010). Thus, no standard outpatient treatments exist that specifically target PTSD among severe BPD clients, particularly those with recent intentional self-injury. In addition, although the current state of the art for treating comorbid disorders is integrated treatment that allows for simultaneous and flexible targeting of both disorders by one provider, the existing PTSD treatments that have been examined with BPD clients are either single diagnosis or sequential treatments. Finally, the effects of BPD treatments on PTSD are either minimal or unknown.

An Integrated BPD and PTSD Treatment: DBT with the DBT PE Protocol

The DBT PE protocol provides a structured method for targeting PTSD within the larger context of standard DBT and differs from existing treatments by (1) providing integrated, concurrent treatment for both BPD and PTSD; (2) focusing specifically on clients with severe BPD, particularly those with recent intentional self-injury; (3) administering treatment in an outpatient (i.e., least restrictive) setting; and (4) implementing standard DBT (instead of modified DBT treatments) in combination with PE for PTSD. In addition, the DBT PE protocol incorporates DBT strategies and procedures into PE to address the complexities that are likely to arise when treating PTSD in a severe BPD

client population. Initial case studies (Harned & Linehan, 2008) and an open trial (Harned, Korslund, Linehan, & Foa, 2012) have been published, and a pilot RCT is currently underway. Results from the open trial ($n = 13$) indicate that this treatment is feasible to administer, highly acceptable to clients, can be implemented safely (e.g., no clients exhibited worsening of intentional self-injury), and shows considerable promise as an effective intervention for PTSD. At posttreatment, the majority of clients no longer met criteria for PTSD (71.4% of DBT PE protocol completers, 60.0% of the intent-to-treat [ITT] sample), and these remission rates are comparable to those found in a meta-analysis of exposure treatments for PTSD (68% of treatment completers, 53% of the ITT sample; Bradley et al., 2005). However, it is important to note that the DBT PE protocol is still actively being developed, and some of the procedures described below may change as they continue to be evaluated.

Standard DBT

Standard DBT (Linehan, 1993a, 1993b) forms the foundation of the treatment and is implemented without modification across four treatment modes: (1) weekly individual DBT psychotherapy (1 h/week), (2) group DBT skills training (2.5 h/week), (3) telephone consultation (as needed), and (4) therapist consultation team (1 h/week). Individual DBT session agendas are determined by a target hierarchy, with life-threatening behavior (e.g., suicidal behavior and NSSI) as the top priority, followed by therapy-interfering behaviors (e.g., noncompliance, non-collaboration), and serious quality-of-life-interfering behaviors (e.g., severe Axis I disorders, homelessness, unemployment, relationship problems). Group DBT skills training is didactically focused and includes four skill modules: (1) mindfulness, (2) interpersonal effectiveness, (3) emotion regulation, and (4) distress tolerance. Brief telephone contact between sessions is used for problem-solving and coaching in generalization of skills, crisis intervention, and relationship repair. Finally, therapists attend a structured weekly consultation meeting to assist each other in the implementation of the treatment.

Within the DBT target hierarchy, PTSD is considered a quality-of-life-interfering behavior and is not targeted until life-threatening and therapy-interfering behaviors are sufficiently controlled. During the pretreatment phase of DBT, clients' treatment goals are assessed, and those clients who express interest in treating their PTSD are explicitly told that PTSD will not be targeted until all forms of life-threatening behavior are stopped. Clarifying this contingency early in treatment has proven to be an effective strategy for increasing commitment to stop intentional self-injury, particularly for clients who are motivated to receive PTSD treatment. Treatment then begins with standard DBT that focuses on helping clients gain control over higher-priority behaviors and acquire the skills necessary to begin the DBT PE protocol. The specific criteria that are used to determine readiness to begin the DBT PE protocol are the following: (1) not at imminent risk of suicide, (2) no recent (past 2 months) life-threatening behavior (i.e., suicide attempts or NSSI), (3) ability to control life-threatening behaviors when in the presence of cues for those behaviors, (4) no serious therapy-interfering behaviors, (5) PTSD is the highest priority target (for the client), and (6) ability and willingness to experience intense emotions without escaping. Once these criteria are met, the DBT PE protocol is begun and occurs concurrently with ongoing individual DBT therapy, group DBT skills training, and telephone consultation. In addition, the DBT therapist consultation team functions to provide support and training to therapists and to address any therapist factors that may interfere with the implementation of the DBT PE protocol. After the DBT PE protocol is complete (and assuming time remains in the agreed-upon treatment period), standard DBT continues with the focus of treatment determined by the client's remaining treatment goals, which often include working to improve

existing and develop new relationships. This general treatment structure is compatible with theories proposing that trauma recovery occurs in three stages, including establishing safety and stability, remembering and mourning past trauma, and reconnecting with the world (Herman, 1992).

The DBT PE Protocol

The DBT PE protocol is based on PE for PTSD (Foa et al., 2007) and incorporates DBT strategies and procedures into PE to address the particular complexities of severe BPD clients. During the implementation of the DBT PE protocol, clients receive either one combined individual therapy session per week (90 min of the DBT PE protocol and 30 min of DBT) or two separate individual therapy sessions per week delivered by the same therapist (one DBT PE protocol session (90 min) and one DBT session (1 h)). The choice of one or two individual sessions is at the discretion of the client and the therapist and is typically determined by the number of additional (non-PTSD) treatment targets as well as logistical considerations. Identical to PE, the DBT PE protocol includes three treatment phases: pre-exposure, exposure, and termination/consolidation.

Pre-exposure sessions. As in PE, Session 1 begins with an overview of the treatment, an orientation to the rationale for exposure, and an assessment of the client's trauma history. Consistent with PE, the DBT PE protocol can be used to treat one or more traumatic events, and the norm in this client population is to target multiple traumatic events. During the trauma assessment, clients are therefore asked to identify the three traumas that are most distressing and/or most related to current problems, and these can include non-Criterion A events as well as fragmented memories and images. One strategy for narrowing down the large number of potential events is to group trauma memories into different categories by trauma type and perpetrator (e.g., childhood sexual abuse by father, childhood sexual abuse by brother, intimate partner violence by ex-husband) and identify the most distressing memory within each category. The potential advantages and disadvantages of starting with each of the three identified traumas are discussed, and consistent with PE, clients are encouraged to start with the most distressing trauma unless there are clinical reasons to believe that this would not be appropriate. The decision about which trauma to target first is ultimately left to the client. Once this decision is made, additional information specific to the selected target trauma is collected. Next, DBT strategies for obtaining, strengthening, and troubleshooting commitments are used, and clients are asked to commit to (1) not engaging in intentional self-injury during the DBT PE protocol, (2) actively participating in the treatment (including completing homework), and (3) controlling any other problem behaviors (e.g., dissociation, substance use) that are likely to interfere with exposure. In addition, a Post-Exposure Skills Plan is created that includes DBT skills that can be used to manage increased urges to engage in intentional self-injury or other distress that may be present after exposure tasks. In addition to the regular PE Session 1 homework, clients are asked to finalize this Post-Exposure Skills Plan, share it with primary support people (if any), and practice skills from the plan at least once per day.

Session 2 begins with reviewing homework and providing clients with didactic information on common reactions to trauma, including reactions that are more common in severe BPD clients (e.g., dissociation, self-injury, increased sexual behavior). As in standard PE, the therapist then orients clients to the rationale for in vivo exposure, introduces the Subjective Units of Distress (SUDs) scale, and works with the client to construct the in vivo exposure hierarchy. Consistent with the DBT skill of opposite action for shame (Rizvi & Linehan, 2005), in vivo exposure is also used to confront situations that elicit unjustified shame (e.g., saying "no" to sex with a partner, sharing aspects of their trauma history with supportive family and friends). Standard PE homework tasks are assigned, including instructing clients to complete their first in vivo exposures and scheduling an optional phone check-in following completion of the first in vivo exposure

task. In addition, clients are asked to continue daily practice of skills from the Post-Exposure Skills Plan.

An optional third pre-exposure session may be conducted with the client and one or more support people (e.g., a partner, friend, or parent). The goals of this session are to orient relevant family members and friends to the plan to begin the DBT PE protocol, prepare them for the likelihood that the treatment will be challenging, and enlist their help and support. This session is conducted in accord with standard DBT strategies for joint or family sessions. For example, the DBT strategy of consultation to the client is used such that therapists generally do not speak for clients and instead encourage clients to speak for themselves. Clients are also coached to use specific DBT skills while interacting with the support person (e.g., using the "DEARMAN" skill to ask their partner to provide support in specific ways). The timing of this session is flexible, but typically takes place prior to Session 1 or between Sessions 1 and 2. If clients do not have people in their lives that are likely to be effective supports, or they would prefer not to involve such people in the treatment process, this session can be skipped.

Exposure sessions. In PE, exposure sessions are structured to include time to review homework, present the session agenda, conduct imaginal exposure, process the imaginal exposure, and assign in vivo and imaginal exposure homework. This general session structure is followed with two modifications. First, the session begins with a brief review of the DBT diary card to ensure that no behaviors (e.g., intentional self-injury) have occurred that would require the DBT PE protocol to be stopped. Second, if one 2-h individual therapy session is being held per week, the standard 90-min PE session is augmented by an additional 30 min of DBT. The DBT portion of the session can occur at the beginning (e.g., for clients who tend to be too exhausted at the end of exposure to engage in additional treatment tasks) or after the exposure (e.g., as an additional strategy to help clients regulate emotions before leaving the therapy office).

Imaginal exposure adheres to the procedures outlined in the PE manual with the addition of methods for monitoring problematic urges or emotions that may arise as a result of exposure. This is accomplished via a modified version of the Exposure Recording Form that clients complete before and after all exposure tasks. In addition to recording the standard SUDs ratings, clients also report pre-, peak, and post-exposure urges to commit suicide, self-injure, quit therapy, and use substances as well as levels of state dissociation. Clients also provide pre-/post-ratings for seven specific emotions, which are intended to increase clients' ability to identify and label emotions (a DBT skill) and to allow therapists to monitor whether intense non-fear emotions are present (and perhaps interfering). Clients also rate the degree to which they radically accept that the trauma occurred. Radical acceptance is a DBT skill that focuses on letting go of fighting reality and is particularly relevant to practice in regard to past trauma. Finally, to aid in monitoring whether corrective learning is occurring, clients provide pre-/post-estimates of the likelihood and severity of feared outcomes of exposure.

Imaginal exposure begins with the trauma memory the client selected. Consistent with standard PE, non-Criterion A events are targeted for treatment when they constitute highly distressing events for the client, and this happens routinely with BPD clients. For example, imaginal exposure is often used to address specific episodes of severe invalidation or verbal abuse by a parent as well as relationship breakups that were experienced as traumatic. Similarly, fragmented trauma memories are routinely targeted during imaginal exposure with BPD clients given that many of these clients have only partial memories or images of some traumatic events. As in standard PE, the following precautions are taken to minimize the risk of suggestibility when targeting these types of fragmented memories. First, clients are clearly told that the goal of imaginal exposure is to make whatever memories and images they do have less distressing—not to try to remember more details. Although many clients do naturally remember more trauma details as

they stop avoiding the memories, this is not the goal of imaginal exposure, and therapists do not make any effort to "uncover" new memories. Second, clients are asked to describe anything they can remember in as much detail as possible while also being sure not to fill in the memory gaps with things they do not actually remember. Importantly, traumatic events for which the client has no clear image or memory are not targeted (e.g., when clients report only a vague sense that "something happened").

During imaginal exposure, standard PE strategies for managing over-engagement are used as needed to decrease emotional intensity (e.g., recounting the trauma narrative with eyes open and in the past tense, writing out the trauma memory instead of verbally recounting it). In addition, clients are coached to use specific DBT skills to downregulate emotions, such as skills to reduce emotional intensity (e.g., opposite action), decrease physiological arousal (e.g., progressive muscle relaxation), and tolerate distress (e.g., self-soothe, distraction). Specific DBT strategies for managing dissociation are also used, including using skills designed to provide intense sensory input to ground clients in the present moment (e.g., holding ice packs, eating sour candy, standing on a balance board, doing wall squats) and implementing contingency management strategies to reinforce non-dissociative behavior (e.g., praise, increase warmth) and punish dissociative behavior (e.g., withdraw warmth, express irritation). Under-engagement is addressed via standard PE strategies such as prompting clients to include additional details, validating clients' concerns about experiencing emotions, and reorienting clients to the rationale for exposure. In addition, clients are coached to use specific DBT skills to upregulate emotions, such as mindfulness observe and describe skills, mindfulness of current emotions and thoughts, willingness, turning the mind, and radical acceptance.

Standard DBT therapist strategies (e.g., dialectical, communication, problem-solving, and validation strategies) are used throughout the DBT PE protocol to increase compatibility with the larger DBT treatment framework and address the particular emotional, behavioral, and cognitive characteristics of severe BPD clients. The use of DBT therapist strategies is particularly prominent during the processing portion of exposure sessions. For example, in standard PE therapists are encouraged not to tell the client how she should view the trauma or how the therapist views it and to instead rely on Socratic questioning to help the client develop these new beliefs for herself. In DBT, cognitive modification strategies include directly challenging maladaptive styles of thinking and suggesting more adaptive cognitions. This more directive approach reflects the fact that many clients with BPD have experienced such pervasive invalidation that they are not able to generate more adaptive beliefs on their own. For example, many clients with BPD simply cannot conceive of the possibility that they may not have been to blame for their abuse. Thus, during the DBT PE protocol, therapists may initially suggest or model more validating ways of conceptualizing their traumatic experiences. As these adaptive cognitions become more believable, clients are asked to generate these self-validating beliefs on their own. Other examples of DBT therapist strategies that are commonly utilized during processing include the communication strategy of irreverence (e.g., saying something unorthodox to get clients "unstuck" from a rigidly held belief) as well as dialectical strategies (e.g., highlighting polarized thinking styles and searching for a synthesis).

Throughout DBT PE protocol sessions, DBT procedures are used when needed to address problems that arise from or interfere with exposure. As described above, clients are coached to use specific DBT skills to address problems with emotional engagement during imaginal exposure. In addition, standard DBT protocols are used to target problems that occur during DBT PE protocol sessions. For example, if a client reports that she did not complete her homework, the therapist would implement the DBT therapy-interfering behavior protocol to assess and solve the problem and get a commitment to complete homework in the future. Similarly, if a client reports high urges to commit suicide or self-injure after completing an imaginal exposure task, the therapist would utilize the DBT life-threatening behavior protocol to assess risk, generate solutions, obtain a commitment to a behavioral plan, and troubleshoot the plan. Other DBT protocols (e.g., DBT suicide

crisis and quality-of-life-interfering behavior protocols) are used as needed. The overall goal is to utilize DBT to increase the likelihood that exposure will be successful with severe BPD clients and to decrease the need to stop or postpone PTSD treatment once it has been started.

Termination and consolidation. The duration of the DBT PE protocol, including the number of trauma memories that are targeted, is not predetermined and is instead based on continuous assessment of the client's PTSD symptoms and treatment goals. Once one memory has been sufficiently processed (e.g., the memory elicits mild to moderate distress), other trauma memories are reassessed to determine which, if any, continue to elicit high levels of distress. Of note, targeting the most distressing memory from a larger category of recurrent trauma is often sufficient to relieve distress associated with the entire trauma category. However, if more work is needed on memories from the same or different trauma categories, therapists may gather information about the next memory and then proceed with imaginal exposure. Ultimately, it is up to the client to decide when she has made sufficient progress and is ready to end targeted PTSD treatment. To date, the DBT PE protocol has been conducted in an average of 13 sessions and clients have targeted approximately 3 trauma memories during this time (Harned et al., 2012). Once the decision to end the DBT PE protocol is made, the final session follows the same general procedures outlined in standard PE, including conducting a brief imaginal exposure, reviewing progress, and discussing relapse prevention strategies. In addition, the DBT PE protocol includes a set of structured worksheets on relapse prevention strategies. These strategies include creating specific plans for continued self-directed exposure practice, learning skills to promote an "exposure lifestyle," planning and rehearsing DBT skills to manage high-risk situations, and identifying DBT skills to use in the event of a future increase in PTSD. The DBT PE protocol emphasizes relapse prevention given that many severe BPD clients are likely to experience additional trauma as well as periods of high stress and crisis that may increase their risk of future relapse.

Procedures for treating higher-priority behaviors. The DBT PE protocol also includes specified procedures for addressing any higher-priority behaviors that may occur. Consistent with the DBT target hierarchy, these behaviors would include life-threatening behaviors, serious therapy-interfering behaviors, or other quality-of-life targets that require priority treatment over PTSD. There is a "zero tolerance" policy for all forms of life-threatening behavior (e.g., suicide attempts, NSSI, suicide threats, suicide preparation behaviors), and the DBT PE protocol is immediately stopped if these behaviors occur or if there is reason to believe the client is at imminent risk of engaging in these behaviors. This rule aims to decrease safety concerns and also functions as a contingency management strategy to decrease the likelihood that these behaviors will occur (given that prematurely stopping the DBT PE protocol is experienced as aversive for nearly all clients). As in standard PE, the decision to stop the DBT PE protocol due to therapy-interfering or quality-of-life behaviors is at the discretion of the therapist (in consultation with the DBT treatment team). This decision is based on whether (1) stopping the DBT PE protocol would effectively punish the behavior, (2) the DBT PE protocol is unlikely to be effective in the presence of the behavior (e.g., severe dissociation during exposure, significant homework noncompliance), and (3) the behavior must be treated now and cannot be effectively or sufficiently treated while continuing the DBT PE protocol (e.g., active psychosis, threats of violence to others). While the DBT PE protocol is stopped, standard DBT strategies and protocols are used to target the higher-priority behavior(s) with the goal of resuming PTSD treatment as soon as possible. The DBT PE protocol is not resumed until the following conditions have been met: (1) the behavior that triggered the stopping is no longer present (if life-threatening) or is sufficiently controlled so as not to interfere with PTSD treatment (if therapy-interfering or quality-of-life), (2) the circumstances that contributed to the behavior have been altered or addressed, (3) the therapist and client believe that the client can prevent further occurrences of the behavior, and (4) when appropriate, the client has made sufficient repairs to those individuals (including

possibly the therapist) who were negatively impacted by the behavior. As a general rule, the length of time that the DBT PE protocol is stopped should match the severity of the behavior that triggered the stopping. Of note, in the open trial such higher-priority behaviors occurred infrequently during the DBT PE protocol and these procedures were therefore rarely implemented.

Case Example of DBT with the DBT PE Protocol

Identifying Information and Relevant History

"Jody" was a 33-year-old, married, Caucasian woman who lived with her husband and three young children. Her parents divorced shortly after she was born, and she grew up with her mother and stepfather, both of whom she described as being very strict and emotionally distant. After graduating from high school, Jody joined the Army where she had her first "episode" in which she suddenly fell to the floor screaming while covering her genitals. She saw a military counselor after that for 6 months, but no further episodes occurred. She was honorably discharged from the Army after 7 years of service and met and married her husband shortly thereafter. She worked full time in a variety of jobs for the next 6 years and reported always excelling at her work. Two years prior to her intake, however, she reported having an "emotional breakdown" after she began to have intrusive, vivid images of severe childhood sexual abuse for the first time in her life. She became unable to work at that time and was placed on psychiatric disability. In the past 2 years she had been psychiatrically hospitalized three times, attended a partial hospital program for 1 month, and had been in treatment with a supportive counselor. During her first hospitalization, she was diagnosed with Dissociative Identity Disorder (DID) and her dissociation had gotten progressively worse since that time. At intake, she reported having four alters that she switched to about four times per week as well as conversion episodes 2–3 times per month during which she would suddenly fall to the floor and become catatonic for up to 30 min. Both the "switching" and the conversion episodes were triggered by exposure to trauma-related cues. She also engaged in NSSI (hitting her hand against objects and cutting) about once per month, and these episodes always occurred while she was dissociated. She met criteria for BPD, PTSD, DID, major depression, panic disorder with agoraphobia, marijuana abuse, ADHD, and obsessive-compulsive personality disorder. At intake, she was taking Xanax, Klonopin, Effexor, Abilify, and Ritalin.

Treatment Process and Complexities

Standard DBT. Jody reported that her primary treatment goals were to treat her PTSD and become "one integrated person" (i.e., no longer have DID). Given these treatment goals, the early stage of DBT focused on helping her to gain control over life-threatening behaviors and other behaviors that would likely interfere with PTSD treatment. This was primarily achieved through contingency management and skills training strategies. Namely, Jody was told that the DBT PE protocol would not be implemented until she stopped self-injuring for a period of at least 2 months and demonstrated the ability to control her dissociation, particularly during therapy sessions. In addition, she was taught DBT skills to help prevent and manage urges to self-injure and dissociate. These treatment strategies were effective in helping Jody to immediately stop self-injuring and to increasingly prevent dissociation (including switching) during therapy sessions. She and her therapist also discussed the possibility that her use of benzodiazepines and marijuana may interfere with the effectiveness of PTSD treatment. As a result, Jody decided to taper off her benzodiazepines under the supervision of her prescriber and to rely instead on DBT skills to manage anxiety. However, she was unwilling to decrease her marijuana use (4–5 days per week), as she did not view it as problematic. Because there was no evidence that her marijuana use was causing significant impairment or interfering with

treatment, her therapist agreed that the DBT PE protocol would not need to be delayed because of it. Given her success at quickly gaining control over higher-priority behaviors, Jody and her therapist decided that she was ready to start the DBT PE protocol after 8 weeks of DBT. It was also decided that the treatment would be implemented in one 2-h session per week due to the limited availability of childcare for Jody's children.

The DBT PE protocol. In preparation for beginning the DBT PE protocol, a session was conducted with Jody's husband to orient him to the plan to begin the PTSD treatment and to identify ways in which he could provide support (e.g., being available after exposure sessions as needed). During Session 1, it became clear that Jody's memories of her sexual abuse were very fragmented and that many were only flashbulb images. She became very distressed while describing these trauma memories during the trauma assessment and nearly switched to an alter during this portion of the session. With coaching from her therapist, she succeeded in using a number of anti-dissociation skills to prevent this (e.g., holding an ice pack, standing on a balance board). She ultimately decided to begin imaginal exposure with her most distressing memory—a violent childhood sexual abuse episode during which she was threatened with a knife. Commitments to no suicide, self-injury, switching to alters, or dissociating during the PTSD treatment were re-obtained and strengthened. In addition, she committed to coming to all sessions and completing all exposure homework while not under the influence of marijuana and to not using marijuana for at least 2 h after completing any exposure task. Session 2 progressed smoothly, after which she successfully completed her first in vivo exposure task involving sitting in a position that was associated with her abuse.

Sessions 3–8 focused on imaginal exposure to her most distressing memory, and she experienced several difficulties during these sessions. During her first imaginal exposure in Session 3, she experienced three flashbacks and was moderately dissociated. To address the dissociation, she completed the imaginal exposure with her eyes open while using anti-dissociation skills as needed. After Session 3, she became quite depressed and had difficulty functioning (e.g., stayed in bed most of the day and was unable to care for her children). This lasted for 3 days, after which she returned to her regular level of functioning. As a result of this experience, in Session 4 she intentionally suppressed her emotions due to fear that she would become depressed and unable to function again. This under-engagement was addressed by validating her urges to suppress emotions while also reorienting her to the importance of allowing herself to feel emotions in their full intensity. Although she then became more willing to experience her emotions, she remained under-engaged in Session 5. Further assessment indicated that this under-engagement was due to the fact that, in an effort to make her trauma narrative more coherent, she had included details in the imaginal exposure that were things she did not actually remember. This had the effect of decreasing the intensity of the memory by making it less "real." To address this, Jody was instructed to only describe details that she could actually remember and was assured that it was fine if the narrative remained fragmented. She was then effectively emotionally engaged in Sessions 6 and 7, including feeling intense anger and disgust toward the perpetrator for the first time. By Session 8, her peak SUDs during imaginal exposure was a 30 and it was decided that she was ready to move to another trauma memory.

Sessions 9–14 then focused on addressing her second and third most distressing memories (different incidents of sexual abuse by the same perpetrator). Because these two memories were flashbulb images and only took several minutes to recount, she completed imaginal exposure by describing them one after the other and then repeating. In Session 10, Jody switched to an alter for the first time during a session, and the exposure had to be stopped for 20 min to get her reoriented. She then resumed the exposure and effectively completed two more repetitions of the narrative in that session. Although she had previously been very compliant with homework, between Sessions 11 and 12, she did not complete any of her imaginal or in vivo exposure homework.

This increased avoidance was due to her desire to be "normal," which included not having to deal with difficult trauma memories. She reengaged in treatment with validation from her therapist and review of her goals and by Session 15 she no longer met criteria for PTSD and was satisfied with the progress she had made. In this final session, she reported that she had "found peace" with her past abuse. She had also radically accepted that she may never remember all the details about her abuse and she no longer felt it was important to do so. She had stopped avoiding all trauma-related cues, rarely dissociated or switched to alters, had not had a conversion episode for 3 months, and had decreased her marijuana use to approximately once per week. She also reported that her relationships with her children had greatly improved and that she felt much more skillful and able to cope with stressors.

Treatment after the DBT PE protocol. The remaining 6 months of treatment consisted of standard DBT focused on Jody's remaining treatment goals, including (1) continuing to experience and discuss sadness and anger related to her abuse, (2) addressing shame and guilt related to having initiated sexual behavior with a cousin on several occasions as a child, and (3) improving her relationship with her husband. She succeeded at reaching all of these additional goals, while also maintaining the gains she had already made in terms of her PTSD, DID, and self-injury.

Conclusions and Future Directions

Over the past several decades, a number of empirically supported PTSD treatments have been developed and evaluated among increasingly representative samples of clients with PTSD. However, these highly effective treatments remain largely inaccessible to clients with severe BPD who are typically viewed as unsuitable candidates for PTSD treatment. As a result, these clients often suffer tremendously under the burden of chronic PTSD, a co-occurring condition that frequently underlies or exacerbates BPD-related problems. As one of our BPD clients, a 25-year utilizer of mental health services said, "I haven't had a chance to tell my story and am derailed by it. I feel like a hamster running in circles because I haven't been able to talk about it." Thus, treatments that can safely and effectively treat PTSD among severe BPD clients are critically needed, and the combined DBT and DBT PE protocol treatment described here has been developed specifically to address the needs of this complex client population. Although initial results of this integrated BPD and PTSD treatment are promising, additional research is clearly needed to evaluate its efficacy and inform ongoing treatment development.

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Treatment of Anxiety and Comorbid Cluster A Personality Disorders

15

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Overview

Having comorbid conditions represents the norm rather than the exception among individuals with anxiety disorders (Brown & Barlow, 1995; Brown, Campbell, Lehman, Grisham, & Mancill, 2001). There is convincing evidence that personality disorders (PDs) frequently co-occur with anxiety disorders. For example, in an early study based on a large outpatient sample with a primary diagnosis of anxiety disorder, 35% of the patients presented with at least one diagnosable PD (Sanderson, Wetzler, Beck, & Betz, 1994). One of the highest comorbidity rates was reported by Skodol, Oldham, Hyler, and Stein (1995); 62% of patients with anxiety disorders were diagnosed with a comorbid PD. It should be noted that this line of research has consistently shown that anxiety disorders are strongly associated with Cluster C PDs (i.e., avoidant, dependent, and obsessive-compulsive PDs). This is understandable, because the fearful and anxious PDs in Cluster C share numerous similarities with anxiety disorders in their core clinical manifestations such as fearful emotional reactions, marked avoidance, and passivity, as well as in their diagnostic criteria (American Psychiatric Association (APA), 2000). For instance, generalized social phobia and avoidant PD are considered to lie on a continuum (Holt, Heimberg, & Hope, 1992; Turner, Beidel, & Townsley, 1992).

In contrast to Cluster C PDs, there is a paucity of empirical research on Cluster A PDs in the context of anxiety disorders, although there is some evidence that suggests important phenomenological and theoretical linkage between anxiety disorders and these odd and eccentric PDs. Further, this rarely reported but clinically significant co-occurrence of Cluster A PDs is suspected to pose numerous challenges to the treatment for anxiety disorders. In order to discuss the complexity of such comorbidity, this chapter presents the following: (a) a brief review of Cluster A PDs in DSM-IV-TR, (b) research evidence that supports the phenomenological linkage between Cluster A PDs and anxiety disorders, (c) clinical complications that may arise from Cluster A PDs comorbid with anxiety disorders, (d) therapeutic strategies to address complications and challenges of such comorbidity cases, and (e) a clinical case that illustrates how to understand and treat an individual presenting with complex anxiety problems mixed with comorbid Cluster A PD.

Cluster A Personality Disorders

The DSM-IV-TR defines a PD as "an enduring pattern of inner experience that deviates markedly from the expectations of the individual's culture, is pervasive and inflexible, and had an

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onset in adolescence or early adulthood, is stable over time and leads to distress or impairment" (APA, 2000). The DSM-IV-TR subdivides PDs into three clusters. For the purposes of this chapter, we will be focusing on Cluster A, the odd and eccentric pattern of personality, which is comprised of paranoid, schizotypal, and schizoid PDs. A hallmark feature of Cluster A PDs is severe distortion in interpreting other people's behavior and resulting social isolation.

Paranoid PD is characterized by a pattern of distrust and suspiciousness of others and affects 2.4% of the general population (Torgersen, Kringlen, & Cramer, 2001) and an even greater proportion in inpatient psychiatric populations, in the range of 10–30% (APA, 2000). People with paranoid PD are constantly on guard and ready to detect threats in their environment. This preoccupation with monitoring ones surroundings leads paranoid individuals to appear on edge, unable to relax, and hypersensitive. Once they perceive a threat, whether the threat is real or imagined, the individual is likely to respond in an aggressive manner that further elicits a hostile response in return. Individuals with paranoid PD are wary of entering into close relationships because they are mistrustful and believe others are capable and motivated to use any information obtained in a manipulative or deceitful manner.

Individuals with *schizotypal PD* experience discomfort in social relationships in addition to clear disturbances in cognition and perception. Prevalence of schizotypal PD in the general population has been estimated to be approximately 3%; more males than females are affected (APA, 2000). Some individuals with schizotypal PD believe they have the ability to read others' minds and control others' behavior. Additionally, many individuals with schizotypal PD exhibit superstitious or magical thinking and display inappropriate or constricted affect. They often lack proper hygiene or have a disheveled appearance.

A person with *schizoid PD* exhibits social and emotional detachment as indicated by a lack of desire for friendships as well as romantic and familial relationships. They have a preference for engaging in solitary activities and are likely to experience a lack of pleasure in daily activities. The schizoid individual displays a flattened emotional response to what others might respond with joy, anger, or sorrow. Due to the lack of adaptive social skills, an individual with schizoid PD communicates disinterest and further withdraws from the social world, resulting in increased social isolation. Prevalence estimates for schizoid PD are believed to be around 1.7% (Torgersen et al., 2001).

Phenomenological Linkage Between Cluster A PDs and Anxiety Disorders

The DSM-IV criteria indicate that fear and anxiety are likely to contribute to clinical manifestations of Cluster A PDs. Similar to social phobia, schizotypal PD is characterized by excessive social anxiety, although its core threat is centered on paranoid suspiciousness rather than negative evaluation about the self. Likewise, paranoid PD's diagnostic criteria describe unwarranted fear about the malicious use of one's own information and concerns (doubts) about the trustworthiness of friends or associates, which may be similar to the qualities often evidenced by pathological worrying or obsessional rumination.

Evidence from well-controlled longitudinal studies also provides support for the close linkage between Cluster A PDs and anxiety disorders. Adolescent anxiety disorders significantly predicted *schizotypal*, *schizoid*, borderline, avoidant, and dependent personality traits measured in early adulthood, even after controlling for the Axis I diagnostic status in adolescence (Lewinsohn, Rohde, Seeley, & Klein, 1997). Johnson et al. (1999) also found that adolescent PDs were significantly associated with elevated risk for developing anxiety disorders during early adulthood. In this analysis, Cluster A PDs revealed a higher odd ratio (OR = 3.83), relative to Cluster B (OR = 2.64) and Cluster C (OR = 3.32) PDs. Moreover, the association between Cluster A PDs in adolescence and anxiety disorders in early adulthood remained significant even after controlling for Axis I conditions and co-occurring PDs in adolescence (Johnson et al., 1999). Overall, the causal pattern of interplay between Cluster A PDs and anxiety disorders on developmental tra-

jectory is not clear, but these findings certainly provide important empirical data supporting their close phenomenological linkage.

As reviewed here, there is reason to suspect that coexistence of anxiety disorders and Cluster A PDs is not as uncommon as usually thought. However, there is a remarkable lack of research concerning the impact of comorbid Cluster A PDs on treatment of anxiety disorders. The most suspected culprit for the profound lack of available data on this important topic is clinical characteristics of Cluster A PDs themselves. Individuals with paranoid and schizotypal PDs are characterized by enormous difficulties in confiding in others and hypervigilance and suspiciousness in interpersonal contexts. Schizoid PD is characterized by indifference and aloofness. Thus, it is highly unlikely for individuals with Cluster A PDs to spontaneously seek relevant treatment resources or participate in clinical research activities. Not surprisingly, Cluster A PDs have not been studied as thoroughly as Cluster B and C PDs, which may again be attributed to the difficulty in recruiting participants with Cluster A PDs. To date, there seems to be no randomized clinical trial research conducted for Cluster A PDs.

Likewise, very little is known about how each of the Cluster A PDs is linked to anxiety disorders in terms of its impact on clinical manifestations and therapy processes. Despite deficient empirical data, we have identified a few relevant topics that have repeatedly appeared in the literature, which may assist in understanding the nature of this comorbidity: (a) schizotypal PD as a negative prognostic factor in treatment for obsessive-compulsive disorder (OCD), (b) schizotypy (schizotypal personality features) and its close linkage to OCD, and (c) paranoid PD frequently co-occurring with panic disorder.

Comorbid Schizotypal PD and Its Negative Impact on Treatment Outcome for OCD

Empirically supported pharmacological and behavioral interventions exist for OCD, but almost half of patients with OCD seem to refuse, drop out, or fail to respond to existing treatments (Moritz et al., 2004). Thus, many researchers have made efforts to examine putative treatment moderators for OCD in order to improve the overall treatment response rates for this debilitating anxiety disorder. Among several candidates, schizotypal PD has received much attention as a negative prognostic factor in treatment for OCD. Jenike, Baer, Minichiello, Schwartz, and Carey (1986) examined 43 patients with treatment-resistant OCD and showed that 26 out of 29 OCD patients without schizotypal PD (90%) improved at least moderately, whereas 13 of 14 OCD patients with schizotypal PD (93%) failed to show improvement. Minichiello, Baer, and Jenike (1987), using a sample of 29 patients with OCD, found that of 19 patients without schizotypal PD, 16 (84%) showed at least a moderate level of improvement in response to exposure and response prevention (ERP) alone or a combined ERP and pharmacotherapy. In contrast, of 10 OCD patients with comorbid schizotypal PD, only 1 (10%) improved in response to the same treatments. Similarly, Baer et al. (1992) found that in patients with OCD who were treated by clomipramine over a 10-week period, schizotypal, borderline, and avoidant PDs were significantly associated with poorer treatment outcome.

These findings were further elaborated by some evidence showing that only a certain aspect of schizotypal features is responsible for treatment failure in OCD (Moritz et al., 2004). This study showed that positive schizotypal symptoms such as perceptual aberrations, magical thinking, and sensory irritation were associated with increased risk for poorer treatment outcomes, whereas negative schizotypal symptoms were not. However, it should be mentioned that some authors failed to find a significant association between poor treatment outcome in OCD and PDs (e.g., Dreessen, Hoekstra, & Arntz, 1997; Fricke et al., 2006). Other studies found alternate types of PDs to be negative treatment predictors for OCD such as borderline, avoidant, and passive-aggressive PDs (Baer et al., 1992; Hermesh, Shahar, & Munitz, 1987; Steketee, 1990). Nevertheless, there is considerable evidence that demonstrates the negative impact of schizotypal

PD on treatment outcome in OCD. Further research is warranted on this topic due to its direct relevance for enhancing overall treatment response in OCD.

Elevated Schizotypy in Individuals with OCD

A closely related line of research has investigated the association between OCD and schizotypy. Although OCD and schizophrenia are easily distinguished, there is a growing line of research that has demonstrated a significant linkage between OCD and schizotypy. Schizotypy is defined as personality traits that are similar to symptoms of schizophrenia but are manifested in an attenuated form (Meehl, 1962). From this point of view, schizotypy is regarded as nonspecific psychosis-proneness (Claridge et al., 1996), or a liability to schizophrenia (Lenzenweger & Korfine, 1995). In the literature, schizotypy and schizotypal personality traits are treated as interchangeable terms that reflect dimensional characteristics varying on a continuous spectrum. Currently, there are two diverging views on the nature of the schizotypy continuum (Asai, Sugimori, Bando, & Tanno, 2011): (a) a fully dimensional view suggesting that schizotypy is a general personality trait evidenced by *all* people to a varying degree, and (b) a quasi-dimensional view conceptualizing that schizotypy is a predisposition to schizophrenia that is shown *only by those with schizophrenic genes*. Regardless of where the lower-end limit of the schizotypy spectrum exists, the majority of authors (e.g., Calkins, Curtis, Grove, & Iacono, 2004; Kerns, 2006) suggest schizotypy is a multidimensional construct that encompasses (a) positive schizotypy (cognitive dyscontrol such as magical thinking and unusual perceptual experiences), (b) negative schizotypy (social anhedonia and interpersonal suspiciousness), and (c) disorganized schizotypy (disorganized speech and behavior tendencies).

There is compelling evidence for elevated schizotypy scores (schizotypal personality traits) in OCD. Patients with OCD were found to show significantly greater schizotypal features, relative to a mixed group of patients with other anxiety disorders (Enright, Claridge, Beech, & Kemp-Wheeler, 1993). Sobin et al. (2000) proposed that there is a schizotypy subtype in OCD based on the findings that mild to severe levels of positive schizotypy signs such as magical ideation and ideas of reference (IOR) were displayed in approximately half the study sample consisting of OCD patients. Moreover, OCD patients were found to show as high self-report schizotypy scores as schizophrenic or bipolar patients, whereas all three groups showed higher schizotypy scores than unipolar depressive patients (Rossi & Daneluzzo, 2002). In addition, Lee and Telch (2005) showed that mental intrusions characterized by sexual, aggressive, and religious obsessions are significantly associated with positive schizotypy such as magical thinking and unusual perceptual experiences among nonclinical students. Poyurovsky and colleagues (2008) study found that OCD patients showed poorer insight, more negative symptoms, overall lower functioning, greater need for antipsychotic augmentation, as well as more schizophrenia-spectrum conditions among first-degree relatives, when they had comorbid schizotypal PD. Some authors have suggested that at least a subgroup of OCD patients may be linked to the schizophrenic spectrum along a multidimensionality of schizotypy (Pallanti, 2000).

However, these data are mostly cross-sectional, and no conclusions can be drawn about the pathogenetic mechanisms underlying the coexistence of OCD and schizotypy. This line of research also takes a dimensional view of schizotypy, and thus elevated schizotypy scores do not necessarily indicate the presence of DSM-IV schizotypal PD. Nevertheless, these findings contribute to revealing the linkage between OCD and schizotypy and are also consistent with the well-known fact that OCD is commonly associated with magical beliefs (Einstein & Menzies, 2004; Tibbo, Kroetsch, Chue, & Warneke, 2000). One way for OCD to link to Cluster A PD appears to be through the shared schizotypy features.

Panic Disorder and Paranoid Personality Features

Another line of empirical data that reveals the linkage between anxiety disorders and Cluster A PDs concerns the elevated paranoid personality features in panic disorder. Overall, panic disorder, along with social phobia and GAD, tends to show highest rates of comorbid PDs among anxiety disorders (Grant et al., 2005; Sanderson et al., 1994). However, in light of clinical features of panic disorder, it is somewhat puzzling to see elevated paranoid PD in individuals with panic disorder.

Reich and Braginsky (1994) reported that among 28 patients with panic disorder who were presented to a community mental health center, 54% of them showed paranoid PD, as assessed by the Personality Diagnostic Questionnaire-Revised (PDQ-R; Hyler et al., 1988). The patients displaying elevated paranoid personality features also revealed an earlier age of onset, longer duration of illness, and overall greater psychopathological symptoms. In a recent study involving 122 adult patients with panic disorder (Ozkan & Altindag, 2005), 33.9% who showed at least one comorbid PD were found to show earlier ages of onset, more severe anxiety, depression, and agoraphobic symptoms and overall lower levels of functioning. Particularly, comorbid paranoid PD was found to be a significant predictor of suicide attempts, along with borderline PD. A more recent study examined the prevalence and associations between DSM-IV mood and anxiety disorders and PDs using the National Institute on Alcohol Abuse and Alcoholism's 2001–2002 national epidemiologic survey data (NESARC; Grant et al., 2005). The wave 1 data assessed 7 PDs (i.e., avoidant, dependent, obsessive-compulsive, paranoid, schizoid, histrionic, and antisocial). Overall, avoidant and dependent PDs were more strongly associated with current anxiety disorders than any other PDs assessed in the study. The odd ratios (ORs) of having avoidant and dependent PDs were 21.0 and 37.2 times greater among those with panic disorder with agoraphobia, relative to the odds of those who did not have current anxiety disorders. Paranoid and schizoid PDs were also found to be strongly associated with the prevalence of panic disorder with agoraphobia (OR = 12.4, 13.1), social phobia (OR = 10.0, 10.4), and GAD (OR = 10.9, 8.2). Taken together, this study revealed that Cluster A PDs were significantly associated with current anxiety disorders, particularly panic disorder.

Marchesi, Cantoni, Fontò, Giannelli, and Maggini (2005) conducted a longitudinal study, in which patients with panic disorder were treated with pharmacotherapy and PDs were assessed using the structured diagnostic interview for DSM-IV personality disorders (SIDP) before and after the treatment. At baseline, 60% of the patients with panic disorder showed comorbid PDs, whereas only 8% of normal matched controls showed PDs. The most frequent PDs included obsessive-compulsive (18.3%), dependent (13.3%), narcissistic (13.3%), avoidant (11.6%), and paranoid PDs (11.6%). After treatment, the overall comorbidity rate was diminished to 43%, and the reduction of panic symptoms was found to be associated with the reduction in paranoid, avoidant, and dependent traits.

Taken together, although paranoia is not considered an obvious clinical feature of panic disorder, several studies have reported significantly elevated paranoid PD from the Cluster A family among individuals with panic disorder. The reason for this unexpected but frequently observed association is not clear, but one possible explanation is that some aspects of the way individuals experience panic disorder seem to have some parallels with experiences of individuals suffering from paranoid PD (Noyes, Reich, Suelzer, & Christiansen, 1991). Individuals with panic disorder tend to be hypervigilant and have difficulty relaxing. They also have trouble having others accept their illness and show heightened interpersonal sensitivity. Moreover, those with panic disorder with agoraphobia generally perceive people around them to be unhelpful in the event of a panic attack, which may suggest the possibility that they have a low level of interpersonal trust.

Additionally, given findings that show a significant reduction of paranoid personality features in panic disorder after pharmacotherapy (Marchesi et al., 2005; Noyes et al., 1991),

comorbid paranoia may be a state-like alterable personality feature. Further research is warranted to explore the nature of the association between panic disorder and paranoid PD.

Factors of Cluster A PDs That Contribute to the Complexity of Anxiety Disorders

A comprehensive review by Dreessen and Arntz (1998) including only high-quality clinical trials (which assessed PDs using a structured diagnostic interview in a prospective research design) concluded that the overall evidence for negative impact of comorbid PDs on treatments of anxiety disorders is weak. Nevertheless, there are several studies that suggest the negative effects of schizotypal and paranoid PDs on treatment of anxiety disorders. Long-standing clinical observations also speak to the increased difficulty in treating individuals with anxiety disorders when comorbid Cluster A PDs are present. Considering the characteristics of the odd and eccentric PDs, irrespective of the resulting efficacy of existing clinical interventions when treating anxiety disorders combined with Cluster A PDs, therapeutic processes would become more challenging when such personality features are present in addition to anxiety problems. There are at least five important clinical features of Cluster A PDs that would render the treatment of anxiety disorders highly complicated (see Fig. 15.1).

Comorbid Cluster A PDs May Dampen the Motivation for Treatment

There is a notable shortage of empirical data on treatment of Cluster A PDs. Clinical observations suggest that one hardly encounters patients who present Cluster A PDs as their chief complaints in clinical settings. We believe that this lack of empirical data at least in part reflects the serious motivational issue linked to Cluster A PDs that impedes spontaneous treatment seeking. Overall, more than 90% of all treatment outcome studies are focused on borderline PD, and this is the only group for which sufficient information exists to formulate treatment guidelines (APA, 2001). There is some research evidence that clearly indicates the low treatment-seeking tendency associated with Cluster A PDs. Tyrer, Mitchard, Methuen, and Ranger (2003) proposed a classification scheme for PDs based on their willingness to seek treatment: treatment seekers (type S) and treatment rejectors (type R). Type R is characterized by their unwillingness and reluctance to present with personality issues as part of treatment problems, engage in psychological assessment and treatment, take drug treatments, accept diagnosis of PD, and change to at least some degree. In their study using 68 patients with PDs, patients with Cluster C PDs were significantly more likely to be type S, whereas patients with paranoid and schizoid PDs were significantly more likely to be type R. Only 3 out of 25 patients with paranoid PD (11%) and 1 out of 17 patients

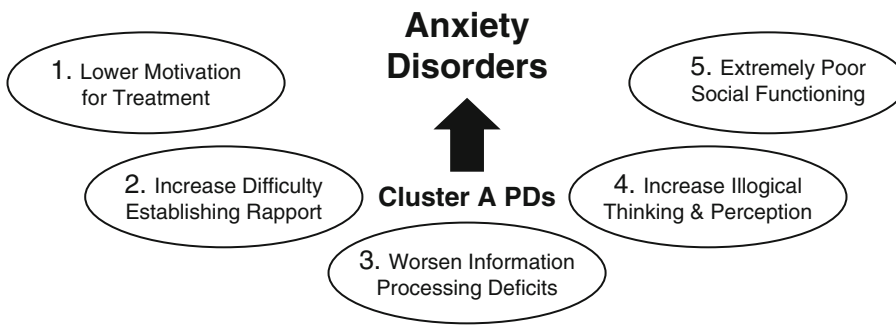

Fig. 15.1 Complexity in treating anxiety disorders with comorbid Cluster A PDs

with schizoid PD (6%) were classified as type S (Tyrer et al., 2003).

Given these findings and common clinical observations (i.e., a remarkable lack of patients presenting with Cluster A PDs), it is conceivable that comorbid Cluster A PDs may adversely affect the individual's willingness to seek treatment for anxiety disorders, as compared with the cases of pure anxiety disorders with no Axis II comorbidity. Likewise, comorbid Cluster A PD may hinder the patient's overall efforts during treatment, reduce overall compliance with treatment procedures such as homework, and increase the chance of early termination of therapy, resulting in overall suboptimal treatment outcomes for anxiety disorders. Considering that receptivity to treatment rationale and procedures (e.g., homework) significantly predicts CBT outcomes (Addis & Jacobson, 2000; Burns & Spangler, 2000), comorbid Cluster A PDs may likely serve as an obstacle for favorable treatment outcome by dampening overall willingness, adherence, and commitment to the treatment for anxiety disorders.

Deficient motivation and willingness for change associated with Cluster A PDs may manifest differently among individuals whose presenting problems are anxiety disorders. Alternatively, the negative influence of Cluster A PDs on patients' motivation may be overridden by willingness to change the acute distress caused by primary anxiety disorders. Hence, efforts to collect empirical data should be made to elucidate this important issue.

Cluster A PDs Likely Cause Significant Difficulty in Establishing Rapport

Another complicating factor of comorbid Cluster A PDs is the individual's difficulty in forming trustworthy and intimate relationships, which would contribute to the difficulty in establishing good rapport (Bender, 2005). Interpersonally, Cluster A PDs are associated with remarkable difficulty in trusting people, lack of warmth, guardedness and defensiveness, and indifference to or avoidance of intimate relationships. The pervasive sense of mistrust and suspiciousness are core interpersonal features of paranoid and schizotypal PDs. Similarly, schizoid PD is also characterized by extremely level of interpersonal aloofness and lack of desire and need for close relationship (APA, 2000). Even if an individual with comorbid Cluster A PD manages to come into therapy, the formation and maintenance of a trustworthy and confiding relationship with the therapist would be a challenging task.

As a general therapeutic factor, sound therapeutic alliance is instrumental in promoting positive treatment outcomes in psychological treatments (Martin, Garske, & Davis, 2000). Particularly, in the treatment of anxiety disorder, many therapeutic techniques require the patients to be courageous and fully committed to highly intense and distressing procedures (e.g., in vivo or imaginal exposure to intense fear-provoking objects, events, or situations) and directly confront the aversive stimuli that they have striven to avoid for months or years. Oftentimes, self-guided exposure or behavioral experiments are an essential part of homework that is critical in generalizing in-session therapeutic gains to day-to-day life and also in collecting further data to disconfirm erroneous beliefs about the feared objects or situations. Thus, from theoretical and clinical standpoints, it is believed that good therapeutic alliance is needed as a prerequisite for behavioral treatment of anxiety disorders (Langhoff, Baer, Zubraegel, & Linden, 2008). Collaboration is a key philosophy in any psychological interventions for anxiety disorders, whereas a fragile and mistrustful relationship with the therapist is highly likely to impede effective implementation of therapeutic procedures and restrict the patient's ability to gain adequate benefits from treatment. In this regard, comorbid Cluster A PD is suspected to present a significant challenge in treating individuals with anxiety disorders, although their mistrustful, hypervigilant, or distant interpersonal qualities may not be highlighted as the primary agenda in session unless they become a roadblock in making progress. Considering the critical importance of initial therapy sessions for therapeutic alliance (Horvath & Luborsky, 1993; Horvath & Symonds, 1991), this is one of the most important areas that deserve special attention in treatment.

Information-Processing Deficits

Cognitive theories of anxiety disorders (e.g., Beck, Emery, & Greenberg, 1985) propose that pathological anxiety problems are caused and maintained by distorted information processing, by which the meaning and consequences of the event are misperceived or exaggerated. Thus, treatments based on this perspective highlight the importance of identifying and altering distorted information processing. For example, individuals with social phobia have a markedly biased pattern of perceiving and interpreting social situations in a way that boost their pathological fears about being negatively evaluated by others or humiliating themselves in front of others (Rapee & Heimberg, 1997). Over the past few decades, there has been an explosive growth in experimental psychopathology research that has illuminated the nature of information-processing biases underlying anxiety problems.

From these considerations, another potential noteworthy complication in treating such comorbid anxiety cases is information-processing deficits that may be worsened by the presence of Cluster A PDs. Particularly, individuals with heightened schizotypal personality traits show information-processing deficits across multiple domains, which are often shown to be as impaired as those demonstrated by patients with schizophrenia: impaired working memory (e.g., Mitropoulou et al., 2005); impaired executive functioning, including cognitive inhibition deficits (e.g., Laurent et al., 2000; Moritz & Mass, 1997); and impaired selective attention and sustained attention (e.g., Bergida & Lenzenweger, 2006).

Despite the lack of empirical data for paranoid PD, paranoid symptoms in general (mostly in schizophrenia) have been associated with numerous cognitive deficits and biases: (a) cognitive rigidity as shown by perseverative errors on cognitive tasks such as the Wisconsin Card Sorting Task (Spaulding, Fleming, Reed, Sullivan, & Storzbach, 1999) and the tendency to jump to conclusions (Mujica-Parodi, Malaspina, & Sackeim, 2000); (b) extreme self-serving bias, in which positive events are attributed to self while negative events to other people (Chadwick & Trower, 1997); and (c) misperception of emotional cues as anger or disgust (Peer, Rothmann, Penrod, Penn, & Spaulding, 2004; Smari, Stefansson, & Thorgilsson, 1994). Moreover, individuals with paranoid PD are overly confident in selectively filtered evidence in support of their biased beliefs and suspicions, while effectively disregarding disconfirming evidence.

Thus, comorbid Cluster A PDs might adversely affect patients' abilities to benefit from standard cognitive interventions for anxiety disorders by diminishing their overall cognitive capabilities to effectively and objectively select, interpret, and integrate data in modifying cognitive biases associated with their emotional distress. Deficits in selective attention are also problematic in treatment for anxiety, considering there is some evidence that anxiety reduction can be best achieved when attention is consistently sustained to the feared objects (Rodriguez & Craske, 1993). Paranoid suspicion often takes the form of unwarranted convictions about the malevolent motives of others, which would be a very tough complication in the context of treating social phobia. Given that every interpersonal situation contains ambiguity to a degree, such information-processing deficits would make the threat disconfirmation and habituation of anxiety more difficult.

Additionally, such information-processing deficits, which heavily tap memory processes, are likely to make it a challenge for the therapist to glean reliable data about the history related to emotional distress when the patient is the only informant. In this manner, the accuracy of information regarding the patient's social interactions may be easily lowered when comorbid Cluster A PD discolors his social information processing.

Cluster A PD May Induce Overvalued Ideas, Magical Thinking, and Ideas of Reference

It is not uncommon for individuals with anxiety disorders to report illogical or unrealistic experiences such as overvalued ideas, magical beliefs, superstitious behaviors, and dissociation, as the core threat of their anxiety symptoms. Patients

with OCD in particular often display magical ideation and superstitious behaviors as their core symptom presentations (Einstein & Menzies, 2004; Tolin, Abramowitz, Kozak, & Foa, 2001). Overvalued ideas have been conceptualized and found to predict poor treatment outcomes in OCD (e.g., Basoglu, Lax, Kasvikis, & Marks, 1988; Neziroglu, Pinto, Yaryura-Tobias, & McKay, 2004).

Considering the biased cognitive processing and elevated schizotypy in Cluster A PDs, this comorbidity may increase the likelihood that individuals with anxiety disorders develop illogical and idiosyncratic accounts about their own experiences (particularly, by making connections between irrelevant events/situations). Relatedly, Lee, Cougle, and Telch (2005) showed that a type of magical thinking such as likelihood thought-action fusion (i.e., merely having this thought will increase the likelihood of the event), which is often elevated in OCD, is significantly associated with schizotypal personality traits, after controlling for the influence of general emotional distress.

Disconfirming exaggerated fears in cognitive-behavioral treatments essentially entails accurate perception of the threat objects/situations and realistic evidence-based reevaluation of their value, meaning, probability, and consequence. Thus, the presence of schizotypal thinking, particularly cognitive disorganization symptoms such as magical ideation and aberrational perception, may render the individuals more resistant to cognitive changes, which would result in increased difficulty in reality monitoring and threat disconfirmation. This may also explain why patients with OCD show poorer treatment outcome when comorbid schizotypal PD is present.

Comorbid Cluster A PDs May Exacerbate Social Skills Deficits and Hypervigilance

Individuals with Cluster A PDs show very poor social functioning characterized by social skills deficits, extreme sensitivity in interpersonal contexts, tendency to perceive malevolent motives, and chronic and pervasive social withdrawal and avoidance (Ahmed, Green, Buckley, & McFarland, 2012; Calkins et al., 2004; Martens, 2010; Peer et al., 2004; Schmidt, Lerew, & Trakowski, 1997; Waldeck & Miller, 2000). These interpersonal features present numerous challenges in implementing exposure-based therapy procedures for anxiety disorders, particularly when the core target of exposure encompasses public or social situations (e.g., various social phobic symptoms, agoraphobia, worries focused on interpersonal relationships, OCD symptoms that involve social interactions). First, the extreme level of vigilance in social situations may discourage them from making efforts to carry out behavioral treatment regimens. Given that many of the anxiety symptoms need to be reexperienced and processed in a therapeutic context (Foa & Kozak, 1986), this reluctance to be exposed to public situations could be a significant obstacle in treatment for various anxiety problems. Second, social skills deficits (along with other odd and eccentric features of Cluster A PDs) may increase the risk for the individuals to indeed evoke unfavorable reactions from others, which would eventually corroborate their extremely negative views of others and interaction with them. Thus, the therapist should be cautious in designing self-administered exposure work for these patients, taking into account the patient's current ability to adequately perform the required social activities. Third, due to their hypervigilance coupled with social information-processing deficits, patients with comorbid Cluster A PDs may be quick to perceive and magnify any negative aspects of such social interactions that occur during the exposure work. Distorted social-cognitive information processing would also confirm their aversive view of social interactions, thereby strengthening their oversensitivity and hypervigilance.

Severe deficits in social skills may reflect the consequence of chronic social withdrawal and avoidance but may also directly contribute to maintaining or strengthening distorted social cognitions and hypervigilance in a self-perpetuating way. The relationship with the therapist is an example of a social context, in which individuals

with Cluster A PD would reveal their maladaptive pattern of relating to others. The difficulty in establishing rapport may be largely attributed to the challenges caused by these interpersonal deficits and biases characterized by extreme levels of vigilance, guardedness, distorted social perception, and social withdrawal.

Treatment Approaches to Address Complexity of the Comorbidity

Overall, cognitive-behavioral treatments with a strong emphasis on exposure procedures and selective serotonin reuptake inhibitor (SSRI)-based antidepressant medications are most empirically supported and widely used clinical interventions for anxiety disorders (see Baldwin et al., 2005; Butler, Chapman, Forman, & Beck, 2006; Hofmann & Smits, 2008; Olatunji, Cisler, & Tolin, 2010, for a review). However, there are no known specific guidelines developed for treating anxiety disorders concomitant with Cluster A PDs. On a positive note, a recent meta-analytic review on the efficacy of existing psychological and pharmacological treatments for anxiety disorders showed that comorbidity was generally not related to the effect sizes at posttreatment and follow-up (Olatunji et al. 2010). Likewise, another review (Dreessen & Arntz, 1998) concluded that there is no clear evidence that comorbid PDs negatively affect treatment outcome for anxiety disorders. Given these findings, it may be that we are currently equipped with therapeutic interventions that can specifically address the target anxiety problems regardless of their complicated comorbidity picture. However, apart from the relationship between comorbid schizotypal PD and poor treatment outcome in OCD, very little is known as to whether the demonstrated efficacy of existing treatments for anxiety disorders would still hold when the comorbid conditions are Cluster A PDs. Much research is needed in this area.

In this section, we suggest some therapeutic approaches that would be useful in addressing complex clinical issues arising from the presence of comorbid Cluster A PDs in anxiety disorders, mostly from the perspective of cognitive-behavioral treatment. However, we expect our suggestions to be relevant for other types of psychological interventions or pharmacological treatments since our emphasis is on overcoming various hurdles in establishing and maintaining therapeutic relationships.

Enhancing Motivation for Change

As discussed earlier, one of the most important tasks in treating anxiety problems comorbid with Cluster A PDs would be to enhance the patient's motivation and willingness to adhere to treatment regimens given its direct relevance for treatment outcome. One promising approach to address this challenge is motivational interviewing (MI; Miller & Rollnick, 2002), which has started to be applied to anxiety disorders in conjunction with CBT (Westra & Dozois, 2008). In addition to potentially distressing procedures of exposure, Cluster A PDs are also expected to generate strong ambivalence about initiating, maintaining, and committing oneself to treatment. MI conceptualizes what is typically considered resistance or noncompliance as a reflection of ambivalence for change and offers effective methods to identify, clarify, and resolve the patient's ambivalence. Core components of MI include (a) expressing empathic understanding of the patient from his own point of view, (b) reflecting and amplifying the discrepancies between desired goals/values and one's current behavior, (c) respecting patient's autonomy and rolling with resistance to diffuse it rather than directly confronting it, and (d) supporting self-efficacy and guiding the patient to generate "change talk," make his own decision, and develop a change plan.

MI has shown promising therapeutic outcomes as an adjunct to CBT for anxiety disorders through controlled case studies with CBT nonresponders (e.g., Arkowitz & Westra, 2004). The spirit of MI is also consistent with Beck, Freeman, Davis, and Associates' cognitive therapy (2004) for paranoid PD that primarily aims to enhance patient's sense of self-efficacy rather than directly addressing maladaptive interpersonal functioning. Moreover, given that ambivalence is considered

a strong emotional component of schizotypy (Mann, Vaughn, Barrantes-Vidal, Raulin, & Kwapil, 2008), MI is expected to significantly enhance adherence to treatment and commitment for change.

Enhancing the Therapeutic Relationship

Collaborative empiricism is the ideal context for conducting effective CBT, and this is particularly important in establishing rapport with individuals presenting with comorbid Cluster A PDs given their characteristic mistrust, hostility, and guardedness. In this regard, therapeutic interventions developed for Cluster A PDs and schizo-spectrum conditions provide useful guidance to design effective communication strategies to prevent or address potential relationship problems (Beck et al., 2004; Beck, Rector, Stolar, & Grant, 2009). Incorporating the existing work, we suggest the following therapeutic approaches:

- Maintain consistently warm, respectful, and nonjudgmental stance throughout treatment.
- Monitor a shift in mood and explore any potential relationship issues in session that may prevent the successful implementation of potentially aversive and effortful procedures such as exposure.
- Avoid jargon and use very plain and straightforward language; frequently provide summary to help patient clearly understand the procedures and rationale of the in-session activities and homework; a written summary of the previous session could help.
- Offer a dimensional, as opposed to diagnostic, framework along with normalization in explaining anxiety and relevant personality problems (i.e., "anxiety and mistrust in interpersonal contexts could be adaptive and protective to some extent, but too much could be counterproductive") in order to improve self-esteem, promote understanding of psychological problems, and reduce stigma.
- Grant as much control to the patient as possible within the allowable range of the specific treatment strategies employed for the main anxiety problem—collaborate with the patient in setting session agenda, goals, and homework.
- Reduce unpredictability while increasing the transparency of the sessions by providing proper overviews in advance to help the patient gain a sense of control over the course of treatment.

Additionally, although paranoid ideation or suspiciousness is not the main focus of the treatment in addressing the main anxiety symptoms, the therapist is recommended to address this relationship issue as soon as it starts to impede therapy progress or in-session relationship. In this regard, we have found it very useful to teach paranoid patients how subtle and powerful self-fulfilling prophecies could be in molding social interaction in a certain anticipated way. Self-fulfilling prophecy (Merton, 1948) explains how a perceiver's (false) beliefs contribute to shaping the target's future behavior. It is helpful for the patient to learn that his own unfavorable belief can indeed contribute to the occurrence of perceived malevolence in people, thereby creating a self-perpetuating cycle of suspiciousness and vigilance. Further, the discussion on self-fulfilling process can be used to improve the patient's sense of control over interpersonal contexts by emphasizing that he can indeed influence what seems to be an uncontrollable social interaction through the power of balanced evidence-based thinking, in addition to commanding a better understanding of interpersonal dynamics. The therapy relationship usually provides easily accessible examples for illustrating the operation of self-fulfilling process, and this effort can also provide a useful channel for communicating about relationship problems taking place in therapy session.

Make New Information as Manageable as Possible

Considering the numerous information-processing deficits associated with Cluster A PDs (particularly impairment in working memory and selective/sustained attention), procedural aspects of the standard CBT protocols for anxiety disorders

may need to be modified to help patients incorporate materials more efficiently. First, use visual aids to explain complex concepts or procedures. For example, in order to explain the maladaptive self-sustaining role of rituals in OCD, the patient may be presented with a diagram depicting a vicious cycle linking obsessional triggers, mental intrusions, resulting distress, rituals, and temporary fear reduction that eventually reinforces the OCD symptoms. Second, provide written review sheets to assist the patient in consolidating materials learned from the sessions. For example, patients with marked deficits in working memory and attentional processing may benefit from reviewing printouts of educational materials about anxiety disorders, instructions for homework assignments, and procedures of therapeutic techniques that patients should continue to practice at home (e.g., relaxation training, diaphragmatic breathing exercise). Relatedly, it would also be useful for the patient to review the session by listening to an audio recording of the session. Third, the fear hierarchy should be designed to include more exposure steps with a shorter duration than usual, as well as allowing a very concrete prediction to be tested, because individuals with significant deficits in fundamental information-processing abilities (e.g., working memory, sustained attention, and integrative/abstractive reasoning) are likely to find it difficult to properly conduct lengthy therapy procedures with full attention or adequately understand and integrate the implications of the results. Fourth, considering cognitive rigidity associated with Cluster A PDs, it would be particularly important to guide the patient to practice generating numerous alternative interpretations in response to ambiguous social situations. Considering that most interpersonal contexts contain some level of ambiguity that is open to multiple interpretations, improved cognitive flexibility may help weaken the tendency to develop paranoid ideation and suspiciousness.

Additionally, in the event of marked cognitive deficits associated with schizotypy, the efficacy of exposure-based treatment work may be enhanced by providing cognitive rehabilitation designed to teach specific memory strategies and organization strategies in order to improve general executive processing capabilities (see McKay & McKiernan, 2005).

Addressing Illogical Thinking and Unusual Perceptual Experiences

Comorbid Cluster A PDs (particularly schizotypy) may add odd and eccentric features to clinical manifestations of anxiety disorders, mostly through magical thinking and aberrational perception. For example, the patient with social phobia in our case study complained of IOR added on to his excessive social fears such that he believed that even total strangers in the street somehow knew about him and spoke ill of him. In such cases, cognitive restructuring work in the form of empirical hypothesis testing conducted through exposure-based behavioral experiment is expected to be more effective in modifying paranoid beliefs rather than relying solely on cognitive reappraisal (Chadwick & Lowe, 1990).

In general, to the extent that these cognitive anomalies are part of the main anxiety problem, existing cognitive interventions developed for paranoid and schizotypal ideation (Beck et al., 2004) may be integrated into the main CBT protocols for anxiety problems. Incorporating the existing work, we offer the following suggestions:

- Communicate empathic understanding of the distress associated with such cognitive anomalies, but avoid validating the beliefs.
- Collaborate with the patient to generate the *evidence for* and *against* such beliefs.
- Generate *alternative interpretations* of such beliefs.
- Discuss how to distinguish vague feelings/suspicions from observed facts relying on empirical evidence-based examination.
- Discuss the impact of maintaining such beliefs on the current anxiety symptoms.
- Discuss the pros and cons of holding onto the beliefs vs. alternative beliefs in terms of promoting positive changes in the main anxiety symptoms.

- Discuss the pros and cons of abandoning the belief (or ignoring the unusual experiences).
- Discuss how such beliefs contribute to maintaining the current anxiety symptoms.
- If needed, in order to reduce emotional distress and potential stigma, provide information to help normalize the experience, including the commonness of paranoid and hallucinatory experiences in the general population and potential utility of certain unusual experiences (Kingdon & Turkington, 1994; McCreery & Claridge, 2002).
- Bring patient's attention to common cognitive distortions (Beck et al., 2004) that are often observed in PDs. Particularly, paranoid ideation, magical thinking, and IOR may be promoted by filtering (i.e., exclusive focus on negative details), jumping to conclusions (i.e., unfounded and hasty conclusion), overgeneralizing (i.e., broadly apply negative outcomes limited to a certain situation), and magnifying/minimizing (i.e., amplify negative details while minimizing positive details).
- Self-serving bias and fundamental attribution error (Ross, 1977) are useful educational materials to help reduce paranoid ideation.

Additionally, with respect to SSRI-based pharmacotherapy for anxiety disorders, the presence of strong Cluster A personality features (particularly schizotypal personality features) may be a useful indicator for augmentative antipsychotic medications in the presence of partial or nonresponse (e.g., Bogetto, Bellino, Vaschetto, & Ziero, 2000; Keuneman, Pokos, Weerasundera, & Castle, 2005; McDougle et al., 1990).

Social Skills Training

In the event that patients with comorbid Cluster A PDs (particularly schizoid and schizotypal PDs) show difficulty following through with treatment procedures that involve interpersonal contexts, social skills training may be added to provide necessary guidance for their successful implementation. Exposure work involving social interactions (e.g., conversation with a stranger, public speaking, speaking with a boss) would be no simple task when social phobia is complicated by severe and chronic social skills deficits or excessive vigilance. Despite a wide variation, most social skills training includes common strategies such as role modeling, rehearsal, positive reinforcement, corrective feedback, and homework (Kurtz & Mueser, 2008). In assisting patients with comorbid anxiety disorders and Cluster A PDs to get prepared for exposure work that involves social interactions, in-session modeling and rehearsal are expected to provide quite practical guidance.

Therapy sessions can serve as a relatively safe interpersonal context for the patient to learn and rehearse basic social skills that are necessary for conducting therapeutic procedures for anxiety problems. For these individuals, unprepared social interactions may produce negative consequences, which would reinforce their excessive social fear, suspiciousness, and vigilance. Relatedly, patients with Cluster A PDs may benefit from learning skills to accurately perceive and label emotions in the self, as well as others, to facilitate social interactions, given their deficits in processing emotional cues accurately.

Case Study

Background Information

Jeffrey, a 27-year old male, had been seen by a psychiatrist and was prescribed antidepressant medication for 3 years until he was referred to our clinic. Our intake phone-interviewer indicated his primary problems as "some complex and unusual obsessive-compulsive symptoms and interpersonal difficulties" on the record sheet. When he visited our clinic for the first time, he was rather poorly dressed and groomed. He looked quite tense and nervous while sitting in the waiting room. He maintained a rigidly upright posture with his back and neck straightened and often looked around vigilantly. In the initial assessment session, he reported his primary problem as "OCD." Our comprehensive assessment and ongoing clinical observation revealed that he met criteria for multiple conditions:

OCD and social phobia in Axis I and schizotypal PD in Axis II.

Clinical Presentations

OCD. Jeffrey presented a peculiar constellation of OCD symptoms that seemed difficult to be expressed in everyday language. His primary obsession occurred in the form of strong urges to clean his body by releasing "bad mental energy" that he perceived as being frequently accumulated inside his body. He reported intense distress due to these frequent urges, although he was not able to clarify what negative consequences he anticipated in the event of holding the mental energy inside. He had developed several bizarre rituals to emit the mental energy, including overstretching his body to straighten his back and limb, standing frozen in a very rigid posture for about 30–60 s, yawning in a very unnatural and exaggerated way, and repeating behaviors he was engaging in when the urge occurred until he felt right about it. These rituals granted him a temporary relief from intense distress, but over the past few years, he continued to expand his repertoire of bizarre rituals by adding new items. Moreover, he suffered severe back pains because of the rigid and tense body posture he assumed repeatedly throughout the day. He reported not being clear as to what specific situations usually triggered his mental intrusions about the mental energy but said he could physically feel the flow of the annoying energy.

Social Anxiety. Jeffrey appeared very tense, introverted, inhibited, shy, timid, and oversensitive early in session. He displayed severe anxiety about being negatively judged by other people and reported having scrambled to avoid social interactions. His social anxiety turned out to be complicated by IOR. He believed that people around him, including total strangers in the street, often knew him well and spoke about him. Although at some level he thought this may not be true, his emotional reactions appeared to accept that as a quite realistic situation. Moreover, he was worried about intentionally or unwittingly making judgment about other people's appearance or behavior because he thought they would easily find out about it (by reading his mind) and be upset with him. Due to his excessive social fears that were complicated by these magical ideas, he had become socially withdrawn, maintaining only minimal social relationships with his family members.

Schizotypal Personality Features. His speech often became vague and circumstantial, and his attention often needed to be redirected to the topic at hand. He also showed quite eccentric and bizarre fantasies. When the topic in session was focused on his hobbies as part of the effort to establish rapport and explore his social resources, he volunteered to bring in his sketchbook to show his drawings. His drawings were full of unrealistic fantasies that mingled nudity, evil spirits, monsters, human, and grotesque patterns. However, despite numerous peculiarities and eccentricities in his clinical presentations, none of them seemed delusional or actively psychotic. He was clearly distressed by OCD and social anxiety symptoms that were complicated and intensified by schizotypal features but maintained an adequate level of insight.

Case Conceptualization

In many aspects, the clinical presentations of Jeffrey closely fit the anxiety-Cluster A PD comorbidity case discussed in this chapter. Evidently, the comorbid schizotypal PD rendered his overall clinical manifestations highly odd and eccentric. Nevertheless, his primary OCD and social phobia symptoms appeared to be maintained in a way that is consistent with current cognitive-behavioral formulations. That is, he engaged in numerous rituals, safety behaviors, and avoidant strategies to minimize his emotional distress, which indeed strengthened and maintained his maladaptive fears and behaviors. His OCD and social phobia were interconnected in the sense that his urges for relieving mental energy usually occurred when he became keenly conscious of other people in either reality or fantasy. This case was further complicated because by-products of his schizotypal thinking (e.g., IOR

and magical thinking) seemed to directly contribute to triggering the intrusions of obsessional urges, which are related to almost hallucinatory perception of the mental energy and odd beliefs of its harmfulness, and amplifying the intensity of socially threatening cues (i.e., IOR and paranoid suspiciousness contributed to increasing his fears of negative evaluation and idea that other people may be able to read his mind).

Treatment Approaches

Jeffrey was treated by weekly CBT sessions over the course of a full year. There were three main components of the treatment. First, using the ERP paradigm, he was exposed to various situations that triggered his perception of mental energy and subsequent urges to release it (e.g., being at a restaurant, walking down a crowded street, hearing unclear human voice from the next room, imagining people speaking about him, tolerating certain physical sensations that increase the urge for rituals) while being inhibited from performing his odd rituals. As he started to make progress in tolerance of the urges related to mental energy, his odd belief about the existence of mental energy was examined. The potential benefits and risks of disregarding the vague physical sensation of mental energy were also discussed. Over time, he showed improved abilities to tolerate the distress related to mental energy, and the overall frequency of rituals dropped.

Second, exposure-based behavioral experiments were repeatedly conducted to improve his social anxiety. Because his social anxiety was interwoven with schizotypal thinking (e.g., IOR and magical beliefs about mind reading), each exposure task was conducted in the form of behavioral experiments by incorporating cognitive intervention focused on collaborative empirical hypothesis testing. However, he was easily distracted and showed difficulty sustaining attention on the topic in question. Thus, for each behavioral experiment, the rationale and purpose of the experiment needed to be explained to him repeatedly using slide presentation on a computer monitor. Collaboratively generating hypotheses, as well as anticipated outcomes, was an essential part of behavioral experiments. For example, one task was to intentionally judge someone's appearance while sitting close to her in a lounge in order to test if she showed any signs of having read his mind and being upset about his negative judgment.

IOR was revisited throughout the course of treatment when it was indicated that Jeffrey's anxiety symptoms were negatively influenced by his fluctuating level of such illogical ideation. To encourage him to openly bring up his trouble with IOR rather than concealing such symptoms, his IOR was labeled as "mental noise" as part of normalizing efforts. Cognitive restructuring was aimed at helping him learn (a) anyone could experience such mental noise particularly under stress, and having it in mind does not mean anything; (b) everyone has the inherent ability to get used to such noise and even forget about its presence; (c) struggling with the noise (e.g., trying to suppress, prove, or dispute) is counterproductive; (d) its content is meaningless because it is merely noise with no evidence; and (e) even in the extremely unlikely event that they indeed speak about you, it does not affect you in any practical way if you simply ignore it. This approach was very helpful for him to openly acknowledge the emergence/presence of IOR and engage in constructive discussion on how (not) to respond to such "mental noise" in session.

Overall, the treatment was successful in reducing OCD and social anxiety symptoms below the clinical cutoff level (Y-BOCS total score = 9, LSAS total score = 35). His schizotypal thinking and unusual perceptual experience persisted in a diminished form and sometimes worsened as a function of his stress level. However, he seemed to have learned how to prevent his schizotypal factors from deteriorating his anxiety problems.

Summary

We have discussed how Cluster A PDs can complicate the clinical features of anxiety disorders and their therapeutic approaches. This issue is crucial because comorbid Cluster A PDs may interfere with the implementation and outcome

of existing treatments for anxiety disorders. As discussed in this chapter, a variety of factors challenge the treatment process for anxiety disorders when working with individuals who present with comorbid Cluster A PD: motivational issues, barriers to establishing a strong therapeutic alliance, information-processing deficits, and impaired social functioning. Finally, we presented a case study illustrating the highly complicated clinical manifestation of such a comorbidity case, several challenging issues that emerged during treatment, and our therapeutic approaches to address such challenges. By addressing the key obstacles arising from comorbid Cluster A PDs that challenge otherwise efficacious therapeutic approaches, we believe that anxiety problems can still be effectively treated within the basic framework of existing treatments for anxiety disorder with proper procedural modifications. It is imperative to continue research in this area to advance our understanding on the relationship between anxiety disorders and comorbid Cluster A PD.

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Treatment of Comorbid Depression 16

Jonathan S. Abramowitz and Lauren Landy

Anxiety disorders are not only among the most common complaints seen by mental health clinicians; they are also very often associated with comorbidity in the form of depression (American Psychiatric Association [APA], 2000). This should not be surprising to readers who are familiar with anxiety disorders as these syndromes are, simply put, *depressing*. The anxious rumination, personal distress, and functional interference resulting from fear and avoidance can be devastating. Consider a man with social phobia who, fearful of most social interactions, spends most of the time alone or a woman with obsessive-compulsive disorder (OCD) whose days are dominated by senseless distressing obsessive thoughts and repeating compulsive rituals that never seem to be done to perfection. Posttraumatic stress disorder and generalized anxiety disorder (GAD) include depressive symptoms in their very diagnostic criteria, and individuals with panic often end up rearranging their lives to accommodate their fears of unexpected anxiety attacks. *Nondepressed* anxious individuals end up being the exception, and yet relatively little attention has been paid to the development and evaluation of treatments for anxiety disorders with comorbid depression. In this chapter, we discuss the nature of the relationship between anxiety disorders and depression, review evidence suggesting that depression attenuates the effects of psychological treatment, offer some hypotheses as to why this is so, and outline and illustrate a promising psychological treatment approach that addresses this complicated clinical picture.

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Nature of the Problem

Overview of Depressive Symptoms

Depression is a psychological state characterized by a chronically sad mood (e.g., feeling empty or hopeless) that is often associated with a diminished interest or pleasure in activities that were once enjoyed. The following other signs and symptoms are also often present: reduced appetite or weight loss, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue, feelings of guilt and worthlessness, diminished ability to concentrate, and recurrent thoughts of death or suicide. Although depression is observed within the context of many psychological syndromes, as well as in nonclinical individuals, a person meets the criteria for a major depressive episode if the aforementioned symptoms persist for at least a 2-week period and interfere with daily functioning (APA, 2000). Major depressive disorder (MDD) is defined by the occurrence of one or more major depressive episodes (APA). Dysthymia, a less severe form of depression, involves a chronically

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depressed mood and reduced interest but does not grossly disable the person's daily functioning (APA).

Co-occurrence of Depression and Anxiety

Depressive symptoms are prevalent across psychological disorders, yet appear to be most closely related with anxiety disorders. Research indicates that anxiety is the single best predictor of the development of clinically severe depressive symptoms (Hirschfeld, 2001; Hranov, 2007). Depression also ranks as the single most commonly co-occurring problem among anxiety diagnoses, affecting up to 90% of people with anxiety disorders (Gorman, 1996).

Comorbidity Rates. The rate of comorbid depression varies across the anxiety disorders, with panic disorder (with or without agoraphobia) being among the most likely to be accompanied by depression—comorbidity rates between 32 and 70% have been reported (Bystritsky et al., 2010; Roy-Byrne, 2000; Weissman, Bland, & Canino, 1997). Among individuals with PTSD, rates of MDD similarly range from 21 to 94% (e.g., Frayne et al., 2005; Ginzburg, 2007; Mollica et al., 1999; Salcioglu, Basoglu, & Livanou, 2003; Sundquist, Johansson, DeMarinis, & Johansson, 2005). Between a quarter and a third of people with OCD meet criteria for MDD (e.g., Antony, Downie, & Swinson, 1998; Nestadt et al., 2001; Yaryura-Tobias et al., 1996), and rates of MDD among individuals with social phobia are less consistent, ranging from 19.5 to 45% (Moitra, Herbert, & Forman, 2008; Ohayon & Schatzberg, 2010). Among patients with specific phobias, 25.4% met criteria for MDD in one study (Marom, Gilboa-Schechtman, Aderka, Weizman, & Hermesh, 2009), and a large epidemiological study in Hong Kong found a somewhat higher rate of 38.5% (Chou, 2009). Approximately 39% of individuals with GAD also meet criteria for MDD (Bruce, Machan, Dyck, & Keller, 2001).

Research and clinical observations also suggest four clinical presentations of comorbid anxiety and depressive disorders: (a) a principal anxiety disorder diagnosis with subthreshold depressive symptoms, (b) a principal MDD diagnosis with subthreshold anxiety symptoms, (c) coprincipal diagnoses of MDD and an anxiety disorder, and (d) subthreshold presentations of both disorders (Hirschfeld, 2001). There is, however, disagreement over the nature and mechanisms underlying the relationship between anxiety and depression. Some theories attribute comorbidity to an overlap in diagnostic criteria, while others highlight common putative underlying genetic and neurobiological substrates (Bremner & Charney, 2010). Still others propose that the distress and disruption in functioning associated with having an anxiety disorder leads to the development of depression (Mineka, Watson, & Clark, 1998; Newman, Przeworski, Fisher, & Borkovec, 2010).

Is Depression the "Chicken" or the "Egg"? As in the folk riddle which asks *which came first, the chicken or the egg*? There are reasons to consider that anxiety could lead to depression and depression to anxiety. Anxiety disorders, for example, typically impair functioning and are personally distressing, which can lead to social isolation, hopelessness, and depressed mood. Depression, on the other hand, is associated with ruminative thinking and negative intrusive thoughts that are reminiscent of anxiety symptoms such as obsessions. It is important to note, however, that despite the clear overlaps in the signs and symptoms of anxiety and depression (Davis, Barlow, & Smith, 2010), clear distinctions can be found. For example, there are cognitive differences: the tendency toward helplessness in anxiety and hopelessness in depression. Differences in neurological and psychophysiological reactivity have also been observed: hyperarousal dominates in anxiety versus anhedonia in depression (American Psychiatric Association, 2000).

Probably the best way to determine whether anxiety precedes depression, or vice versa, is to examine the temporal nature of these two symptom constellations. Accordingly, consistent findings demonstrate that among patients with this comorbidity pattern, the onset of anxiety disorders is more likely to temporally precede that of mood disorders (e.g., Alloy, Kelly, Mineka, & Clements, 1990; Lepine, Wittchen, &

Essau, 1993). In a large study, for example, 59% of individuals with comorbid mood and anxiety disorders experienced their first anxiety disorder at least a year before the onset of their mood disorder, while only 15% had experienced a mood disorder first, and 26% experienced the onset of both a mood and anxiety disorder within the same year (Lepine et al.). These data vary across the different anxiety disorders, yet the general trend holds. For instance, in two studies, the majority of individuals with OCD and comorbid depression experienced the onset of their obsessive-compulsive symptoms before their depressive symptoms began (Bellodi, Scioto, Diaferia, Ronchi, & Smiraldi, 1992; Demal, Lenz, Mayrhofer, Zapotoczky, & Zitterl, 1993). Temporal examination of the onset of comorbid social phobia and MDD also indicate that in the majority of cases, social phobia develops first (e.g., Schneier, Johnson, Hornig, & Liebowitz, 1992). Indeed, the avoidance, isolation, and social anxiety that characterize social phobia often leave sufferers without opportunities for interpersonal interactions, which are at the foundation of many enjoyable activities, thereby perhaps creating vulnerability to depression. Although these studies are primarily based on retrospective self-report data, the general pattern of results suggests that the direction of the causal arrow involves anxiety disorder symptoms leading to the secondary development of depression.

Predictors of Depressive Symptoms in Anxiety Patients. Why do some anxious patients, but not others, develop comorbid depression? A number of researchers have sought to elucidate variables that predict the presence of depression among patients with anxiety disorders. de Graaf, Bijl, ten Have, Beekman, and Vollebergh (2004), for example, found that physical disability and stressful life circumstances (past and present) were the strongest predictors of MDD among individuals with anxiety problems. Eison (1990) found data consistent with the view that the prolonged central nervous system arousal involved in anxiety disorders depletes forebrain neurotransmitters, leading to depression.

In an investigation of comorbid social phobia and MDD, Moitra et al. (2008) found that behavioral avoidance mediated the relationship between these two disorders. Research with OCD patients found that depression is associated with more severe obsessions, but not compulsive rituals (Ricciardi & McNally, 1995), and particularly with the presence of obsessional intrusions concerning sexual and religious themes (Hassler, et al., 2005). Moreover, relative to nondepressed OCD patients, those with MDD evince more severe cognitive distortions (i.e., the tendency to misinterpret the significance of obsessional thoughts) and poorer insight into the senselessness of obsessions and rituals. Thus, the presence of depression is not only associated with greater overall OCD symptom severity, but also with certain presentations of this highly heterogeneous condition. In similar fashion, patients with GAD with comorbid MDD report more severe anxiety symptoms than do GAD patients without MDD (Newman et al., 2010).

Factors That Contribute to Complexity

Clinical Picture

Clinical observations and research findings indicate that the presence of comorbid depressive symptoms and MDD complicates the clinical picture and treatment of anxiety disorders (Abramowitz & Foa, 2000; Angst & Dobler-Mikola, 1985; Stavrakaki & Vargo, 1986). Anxiety disorder patients with depression might become demoralized, giving in to their fear and avoidance patterns and subsequently becoming more impaired than less depressed anxiety patients. Their risk of suicide might also be greater than nondepressed patients. Finally, the physical signs which accompany depression (sleep disturbance, weight loss or gain, and psychomotor retardation) may directly exacerbate the symptoms of various anxiety disorders. Indeed, one study found that patients with GAD and with panic disorder with agoraphobia, who also had comorbid MDD, were half as likely to recover from these anxiety disorders as compared to individuals with GAD and panic

patients without MDD (Bruce et al., 2005). In this study, comorbid MDD was also associated with a twofold increase in the risk of long-term recurrence of the anxiety disorder. Other investigations confirm the clinical observations that individuals suffering from anxiety disorders complicated by depressive disorders (e.g., MDD) experience more severe and lasting psychological symptoms, a greater risk for suicide, higher relapse rates, and greater functional impairment when compared to nondepressed anxious individuals (Belzer & Schneier, 2004; Davis et al., 2010; Goldenberg et al., 1996; Hecht, Von Zerssen, & Wittchen, 1990; Huppert, Simpson, Nissenson, Liebowitz, & Foa, 2009).

Treatment Response

Effective psychological treatment for anxiety disorders involves cognitive-behavioral interventions such as exposure therapy and cognitive restructuring (e.g., Abramowitz, Deacon, & Whiteside, 2011). Yet, these techniques require the patient to work hard to learn and practice skills, some of which involve facing their fears and deliberately provoking anxiety and distress. Depressed patients often lack the motivation and willpower to do this difficult work and may even fall prey to dysfunctional beliefs that they don't *deserve* to get better. Whereas cognitive-behavioral therapy (CBT) can be highly effective in reducing the symptoms of anxiety disorders, for the reasons mentioned above, the presence of unmanaged depression often hinders the effects of this treatment. For example, in separate studies, Abramowitz and Foa (2000) and Steketee, Chambless, and Tran (2001) found that OCD patients with comorbid MDD fared worse with exposure-based CBT relative to nondepressed OCD patients. Similar results were found with depressed and nondepressed panic disorder patients (Steketee et al.).

There are several potential explanations for why depression negatively impacts the outcome of CBT for anxiety. For example, decreased compliance with treatment demands among depressed individuals. As mentioned previously, successful exposure therapy requires practicing prolonged and repeated confrontation with feared stimuli, and depressed individuals might comply with these demanding instructions. Indeed, noncompliance is related to attenuated treatment response. It is also possible that depressed anxious patients perceive themselves as more helpless (Seligman, 1975) or less efficacious (Bandura, 1977) than nondepressed ones, resulting in lower expectations of improvement, thus interfering with treatment gains. Perhaps depressed patients attribute any limited gains in treatment to external sources and therefore evidence less improvement and more relapses than nondepressed patients.

In the remainder of this chapter, we discuss approaches to the psychological treatment of anxiety disorders with comorbid depression.

Treatment Approaches to Address Comorbid Depression

When the negative impact of depression on the effects of CBT for anxiety is considered along with high prevalence rates of comorbid depression among people with anxiety disorders, one recognizes the importance of developing treatments for patients with this pattern of comorbidity. For the most part, research on the treatment of anxiety has focused on more or less "straightforward" or "clean" presentations of anxiety disorders (e.g., Foa et al., 2005). Less attention has been paid to complex cases such as those involving comorbid disorders. Yet ironically, the majority of individuals with anxiety present with complexities of one sort or another—comorbid depression being among the most common presentations.

As mentioned, exposure—repeated and prolonged confrontation with feared stimuli—along with help refraining from subtle and overt avoidance and safety-seeking behaviors (i.e., response prevention) is the centerpiece of CBT for most anxiety disorders (Abramowitz et al., 2011). Someone with social phobia, for example, is helped to confront situations in which he or she might become the center of attention (e.g., speaking in a group, dropping a handful of coins on the floor of a crowded

mall) while simultaneously refraining from any avoidance or anxiety-reducing behavior (e.g., excessive rehearsal, going shopping at off-peak hours). Exposure-based CBT is a highly effective therapy for anxiety disorders, producing an average of 60–70% reduction in fear, avoidance, and the use of safety behaviors (Abramowitz et al., 2011). A drawback of this approach, however, is that patients must confront their fear-evoking stimuli and resist urges to immediately reduce anxiety via escape or avoidance. Because exposure therapy requires compliance with these somewhat demanding procedures, approximately 25% of patients either refuse this form of therapy or terminate prematurely. Moreover, exposure therapy is highly focused on alleviating anxiety and fear and does not directly address comorbid problems such as depression.

Cognitive conceptualizations of anxiety disorders have led to the inclusion of cognitive therapy (CT) strategies along with exposure in many treatment protocols (e.g., Beck & Emery, 1985; Wells, 1997). In CT, a number of verbal and skill development techniques are used to (a) educate patients about the nature of anxiety and how pathological anxiety is maintained and (b) help patients correct dysfunctional beliefs and automatic thoughts that lead directly to anxiety and fear (e.g., exaggerated estimates of probability and severity of catastrophes). For example, someone who experiences recurrent panic attacks would be helped to recognize that the symptoms of panic are nothing more than the harmless sensations associated with anxious arousal (fight-or-flight), and as such, panic attacks will not lead to physical or mental harm. In addition to verbally challenging dysfunctional thinking patterns, patients test out the validity of these (and corrected) beliefs using real-life "experiments" (that are similar to exposure exercises), such as trying to "bring on" a panic attack. The efficacy of CT is suggested by numerous outcome studies, yet CT does not appear to be quite as effective as exposure-based therapy for anxiety (Abramowitz et al., 2011).

Treatment protocols developed for anxiety disorders have not routinely addressed the common comorbid depressive symptoms that are known to present challenges. There are, however, a few possible ways in which CBT could be implemented to address comorbid depression. These are described below, along with the theoretical and practical considerations relevant to each. We then present a case study illustrating what we believe is the most useful approach.

Adding Antidepressant Medication to CBT

Antidepressant medications, such as the serotonin reuptake inhibitors (SRIs), are the most widely used treatments for both depression and anxiety disorders. Thus, intuitively, the use of these agents should improve outcome for anxiety patients with comorbid depression. Very few studies, however, have addressed whether antidepressants offer an advantage over exposure-based CBT, specifically for comorbid samples, and the existing studies have numerous methodological difficulties which limit the conclusions that can be drawn. The OCD literature provides the best examples of such studies. In one investigation with OCD patients, Marks et al. (1980) found that clomipramine (CMI) helped severe depression and OCD symptoms more than did placebo. However, the comparison included only five patients on CMI and five on placebo, and the statistical analysis was conducted at the 4-week point in treatment, which may not have been enough time for CMI to yield full benefit in all patients.

In another study, Foa, Kozak, Steketee, and McCarthy (1992) examined whether using imipramine (IMI) prior to CBT would facilitate improvement in OCD symptoms once CBT began. In their prospective study, mildly and severely depressed OCD patients received either pill placebo or IMI for 6 weeks prior to CBT. Results indicated that although IMI improved the symptoms of depression, it did not potentiate the effects of CBT on OCD symptoms. Abramowitz et al. (2000) also included a comparison between severely depressed OCD patients who either were or were not using SRI medications during CBT. No difference between groups were reported, although the small size of the severely depressed group in that study ($n = 11$) limits the generaliz-

ability of this finding. To date, there is little compelling evidence that medication potentiates the effects of CBT with severely depressed anxiety patients.

One explanation for the above conclusion is that because SRI medications are the most widely used therapy for anxiety, patients with anxiety disorders have often already tried these agents before presenting for psychological treatment. Thus, many depressed anxiety disorder patients in treatment studies might have been "medication resistant," thus putting a ceiling on the effects of medications. Nevertheless, since the average improvement with SRI medication is somewhat modest (about 20–40% on average), there is a need to consider non-medication strategies for augmenting psychological treatment for depressed anxiety disorder patients.

Adding Cognitive Therapy for Depression

Cognitive therapy is a useful intervention for anxiety disorders and can also be applied in the treatment of depression. Indeed, CT yields high responder rates, few adverse effects, and good durability of gains in depressed patients (e.g., Elkin et al., 1989). Cognitive therapy for depression involves identifying and challenging overly negative beliefs about oneself, world, and the future that lead to overly negative and biased interpretations of events, giving rise to feelings of extreme hopelessness, helplessness, and personal failure. It also includes the use of behavioral activation in which the patient increases his or her engagement in activities he or she finds enjoyable. This helps positively reinforce behavior that is the opposite of depressive behavior (e.g., sleeping, social isolation). Numerous studies report significant and lasting improvement in dysphoric mood and other MDD symptoms following CT (Dobson, 1989). Typically, 50–70% of MDD patients who complete CT no longer meet criteria for MDD at posttreatment, and only 20–30% show significant relapse at follow-up (Craighead, Evans, & Robins, 1992).

Another reason CT is a good choice to use in the treatment of anxiety disorder patients with comorbid depression is efficiency: that is, the conceptual approach and implementation of CT as used for depression (e.g., identifying and challenging beliefs) are largely similar to those used in CT for anxiety disorders—although the content of the dysfunctional beliefs that are targeted is different. For example, cognitive restructuring can be used to modify dysfunctional cognitions relevant to panic attacks (e.g., "too much panic will lead to a heart attack") as well as those relevant to depression (e.g., "I am a total failure as a human being and can't do anything right"). Thus, patients could learn to make use of the same skills to reduce both anxiety and depressive symptoms.

Engaging in CT to reduce depressive symptoms prior to beginning exposure techniques might alleviate some depressive symptoms and help the patient increase motivation and compliance with difficult exposure therapy assignments, thereby enhancing reductions in anxiety symptoms. Unfortunately, however, no systematic evaluations of such treatment programs have been conducted, although we are currently conducting a small study involving a series of patients with OCD and comorbid depression. The following case report describes the details of how we have implemented this treatment approach with one such patient.

Case Study

Patient Background and Assessment

"Elaine" was a 26-year-old woman from the southeastern United States who came to our outpatient clinic seeking treatment for "depression and obsessive thoughts." She stated that her obsessive thoughts about her new baby were "ruining her life." Elaine and her husband of 3 years, Joe, had recently given birth to their first child, a son named Ryan. But Elaine was avoiding interacting with Ryan, especially if Joe was not around to "supervise." This was because Elaine was having thoughts that she might sexually

molest the baby when no one was looking. She was unable to bathe Ryan, change his diaper, or breastfeed him.

Assessment using the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) and Symptom Checklist (Goodman et al., 1989a, 1989b) indicated prominent sexual obsessions, mental rituals (e.g., praying), and rituals involving asking for reassurances from her mother and husband that she would "never do such a thing." Specific obsessional thoughts included unwanted images of the baby's penis and impulses to touch his genitals. Elaine was very religious and spent hours praying that she wouldn't act on her unwanted thoughts (i.e., mental rituals). She also repeatedly asked others questions such as "Do you think I will molest the baby?" and "What does it mean that I think about doing such evil things?" Elaine's pretreatment score on the Y-BOCS severity scale was 27, indicating fairly severe OCD symptoms.

A diagnostic interview confirmed both a diagnosis of OCD and of major depression. Elaine had experienced some minor OCD symptoms as a teenager, but her anxiety got noticeably worse during her pregnancy, and her symptoms spiked after Ryan was born. For the last few months, Elaine reported feeling down, having decreased energy, decreased interest in activities or hobbies, and feelings of worthlessness, hopelessness, and passive suicidal thinking. Her Beck Depression Inventory (BDI) score was 29, and her Hamilton Depression Rating Scale score was 20, suggesting clinical depression of moderate severity.

Elaine had never received treatment for OCD or depression except to speak with the pastor at her church. After several sessions with the pastor, she saw the advertisement for our clinic and decided to contact us. After an assessment and discussion of treatment options, Elaine was quite ambivalent about beginning therapy, primarily because she feared engaging in exposure exercises. Her therapist explained how treatment would indeed be a challenge but would progress at a level Elaine was comfortable with and that she would never be forced into doing exposure practices. Instead, it would be the therapist's job to help Elaine to see how trying CBT (exposure and response prevention) would help her achieve relief from her symptoms even if it meant "investing anxiety up front in a calmer future." After some discussion with her family, Elaine opted to enter our program.

Conceptualization and Treatment

Treatment involved 16 90-min twice-weekly sessions over the course of about 2 months (8 weeks). During the first two treatment sessions, the therapist continued to collect information about Elaine's depressive symptoms, and she was introduced to the cognitive model of emotional disorders wherein negative emotions are considered to be evoked by dysfunctional interpretations of situations. It became clear that Elaine's depression was secondary to her OCD symptoms; she described feeling guilty, worthless, and like a "bad mother" as a result of her unwanted sexual obsessions. Like many individuals with OCD, Elaine overinterpreted the occurrence and significance of her senseless obsessional thoughts. She believed that deep down, she was becoming a sexual predator and that it was only a matter of time before she eventually gave in and ended up sexually assaulting her own child. Elaine attributed her problems to demonic possession and often berated herself for not being a good enough servant of God. Cognitive therapy for depression was begun, and the therapist taught Elaine to recognize cognitive errors including "overgeneralizing," "catastrophizing," and "discounting the positive" (Greenberger & Padesky, 1995). Elaine was helped to generate more realistic appraisals of herself and her future. For example, "I am a terrible mother" was modified to "I want what's best for my baby, but am having problems with OCD that make me have thoughts about strange things."

Elaine was instructed in how to use daily thought diaries to practice identifying and modifying dysfunctional thoughts on her own. She also worked with her therapist to develop a routine of activities that she enjoyed (behavioral activation), such as watching the Comedy Central TV network, renting movies she liked, and ice

skating. It became clear that Elaine felt that how others perceived her as a parent was very important. Thus, she was encouraged to get involved in playgroups and "Mommy and Me" classes where she and Ryan would interact with other mother-child dyads. Numerous cognitive therapy worksheets were dedicated to thoughts regarding the importance of what others thought of her and her ability to be a good parent. Thus, Elaine was helped to reduce the emphasis she placed on what she *thought* others might be thinking of her.

Sessions 3 and 4 involved learning to apply the cognitive model (and cognitive therapy) to OCD symptoms. In particular, Elaine was taught that distressing intrusive thoughts—even those about unwanted or taboo subjects—are normal experiences for most people, and that such thoughts do not mean anything significant or threatening about the thinker. A model of OCD in which normal obsessional thoughts get misinterpreted as overly significant, leading to anxiety, was outlined. Anxiety then leads to urges to avoid Ryan, engage in compulsive prayer, and ask for excessive reassurance from her family. These avoidance behaviors and rituals, which reduce anxiety and provide reassurance in the short term, paradoxically reinforce obsessional anxiety in the long run because they lead to greater preoccupation with the unwanted thoughts and the sense that the thoughts are "out of control." Elaine understood the conceptual model, and it came as a relief to learn that others also experience strange intrusive thoughts from time to time (her therapist self-disclosed many of his own). She understood that once she realized her sexual thoughts about Ryan were not dangerous, her urges to engage in avoidance, excessive prayer, and reassurance seeking would be diminished and that her anxiety preoccupation with the unwanted thoughts would similarly decline.

In the fourth session, an exposure hierarchy was developed collaboratively. After a thorough discussion of the rationale for therapeutic exposure and response prevention, Elaine agreed to confront a number of situations that she had been avoiding over the remaining 11 sessions while also attempting to gradually drop her compulsive behaviors. Elaine also continued to practice cognitive restructuring and behavioral activation for her depressive symptoms during and between these treatment sessions. During her fifth visit, however, she reported that her mood was improved, that she felt a good deal of confidence in her therapist, and that she was hopeful of improving with continued therapy.

Exposure began with confronting objects such as diapers and pictures of babies from magazines. Elaine was instructed to allow unwanted sexual thoughts to enter her mind and just "hang out" there. She was also told to allow herself to worry about molesting Ryan; she needed to confront, rather than avoid, these thoughts and ideas. Although Elaine had some difficulty refraining from compulsive praying rituals at first, by the seventh treatment session, she had cut her prayer to acceptable levels, such as before bedtime, and was not asking Joe for reassurance about her unwanted thoughts. Joe had attended an early exposure session and had been instructed by the therapist in how to offer supportive reinforcement for successful exposure practice, rather than giving reassurance that "everything would be OK." At the eighth session, a mid-treatment evaluation revealed a Y-BOCS score of 20, BDI score of 13, and a Hamilton Depression Rating Scale score of 10.

Sessions 9 through 16 included reviewing exposure and cognitive therapy homework assignments as well as conducting in-session exposure practice with gradually more difficult situations. With some reluctance, Elaine was able to confront most items on her exposure hierarchy including changing Ryan, playing with him while he was naked, giving him a bath, and putting lotion on and around his penis when he developed a rash. She also was able to allow her unwanted intrusive thoughts to enter her mind without needing to resist or suppress them. Although urges to say prayers about these thoughts sometimes occurred, Elaine understood the importance of resisting these urges and practicing exposure to her fear cues. She reported being able to spend more and more time with Ryan and being alone with him. She also began to feel more worthwhile as a parent, and her feelings of being a bad mother had disappeared. An

important aspect of Elaine's reduction in depression was the genuine recognition and reinforcement she received from her family, who had observed her hard work and improvement over the course of therapy.

At the end of treatment, Elaine's Y-BOCS score was 11, indicating a near 60% reduction in OCD symptoms. Her BDI score was 7 and her Hamilton score was 4, both within normal range. She felt much more in control of her obsessional and depressive symptoms. Elaine also felt able to continue her trajectory of improvement after the end of therapy. Three months following the end of treatment, Elaine's Y-BOCS score was 12. She arranged to see her therapist for four additional sessions to practice exposure to a few situations that continued to give her trouble, including bathing and changing Ryan. She was only infrequently asking for assurances and was no longer praying about her intrusive thoughts.

Clinical Issues and Summary

Elaine's case indicates that CBT using CT methods to augment exposure-based CBT procedures holds potential for treating OCD patients with comorbid major depression. At least for this particular individual, the 16-session, twice-weekly treatment regimen appeared to improve the tolerability of anxiety-evoking exposure assignments so that she was able to engage in (and benefit from) them. Given Elaine's negative disposition toward exposure during her initial assessment, it is likely that she would have had difficulty with compliance (if not discontinued therapy altogether) if exposure had been begun immediately. Instead, by introducing CT first, Elaine had the opportunity to (a) establish rapport with her therapist, (b) see that the therapist understood her OCD symptoms, (c) come to better understand her own obsessional thoughts in a less threatening way, and (d) develop cognitive coping strategies to reduce her depressive symptoms and prepare her for exposure sessions. It is interesting to speculate whether these factors contributed to Elaine's engagement in the more difficult aspects of the therapy. Indeed, some have advocated that CT strategies be used routinely to help patients confront feared situations during exposure.

Elaine's depression was clearly secondary to her OCD. That is, she was primarily depressed about having intrusive obsessional thoughts. Indeed, she believed these thoughts indicated that she was a terrible person—perhaps unfit to raise a child. Such a belief system is the sort that routinely leads to depressive symptoms. Very likely, reduction in her OCD symptoms toward the middle stages of treatment resulted in further improvements in her depression. In some instances, as mentioned earlier in this chapter, patients' depressive symptoms represent primary complaints in their own right, over and above the distress associated with having OCD. For example, one patient we evaluated had experienced depression for several years before the onset of her OCD. An important question concerns whether patients whose depressive symptoms are related to the distress or functional impairment associated with OCD would fare better in CBT for OCD as compared to patients for whom OCD and depression represent truly unrelated diagnoses.

Conclusions and Future Directions

To date, the following can be said about the influence of comorbid depression among psychological treatment for anxiety disorders: (a) depression and anxiety go hand in hand, and many patients with anxiety disorders also suffer from depression; (b) it appears that in most instances, depressive symptoms emerge following the onset of anxiety disorder symptoms, and perhaps in response to the distress and functional impairment associated with severe anxiety; and (c) the presence of comorbid depression hinders outcome of CBT, which is the most effective treatment for anxiety disorders, although the precise mechanisms for this are not fully understood.

Although the anecdotal case notes we present above provide reason for cautious optimism, much work is required before more firm conclusions

regarding the effectiveness of adding CT for depression to exposure-based CBT for anxiety disorders can be made. Additionally, important questions need to be answered in order to determine the clinical and cost-effectiveness of this treatment approach. For example, it will be necessary to determine whether or not this comprehensive treatment package is more effective than exposure therapy, CT, or SRI medication alone or that it is superior to the combination of psychotherapy and medication in this population.

In the future, clinicians and researchers would do well to continue to develop and evaluate treatment programs for so-called "complex" cases of anxiety disorders. Especially in the case of depression, where comorbidity with anxiety is as much the rule as it is the exception, strategies to increase motivation for improvement, beliefs about and response to exposure therapy, and overall behavioral activation seem critical to implement in CBT protocols. We look forward to this next phase of treatment development in the anxiety disorders.

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17 Limited Motivation, Patient-Therapist Mismatch, and the Therapeutic Alliance

Alessandro S. De Nadai and Marc S. Karver

As described in Chap. 1 of this volume, anxiety disorders present a series of disabling conditions, which affect millions worldwide and create a staggering cost to society, which is estimated to be $42.3 billion annually in the United States (Greenberg et al., 1999). Furthermore, they have a profound impact for those who suffer from them at work, school, and in the home (Langley, Bergman, McCracken, & Piacentini, 2004; Rubin et al., 2000). Fortunately, through progress in behavioral science research in the past century, a variety of empirically supported treatments (ESTs; American Psychological Association Presidential Task Force on Evidence Based Practice, 2006) have been developed and established as efficacious interventions for anxiety disorders, with the vast majority that are applicable to anxiety disorders consisting of forms of cognitive behavioral therapy (CBT) that include the principles of exposure and response prevention (ERP; Barlow, 2008; Ollendick & King, 2010). In comparison to other efficacious treatments such as pharmacotherapy, CBT has the benefits of no medicinal side effects, provides the requisite skills for retaining treatment benefits and preventing relapse, and is often more cost-effective than pharmacotherapy for anxiety disorders in the long term (McHugh et al., 2007).

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However, while CBT presents many outstanding benefits in the treatment of anxiety disorders, a substantial proportion of children and adults do not experience full remission of symptoms at the end of treatment. This could be for several reasons. One explanation is that CBT requires active patient participation, which involves coming to treatment sessions on a regular basis, actively participating during treatment, and participating outside of treatment sessions in the form of homework. Given dropout rates of approximately 23% observed in randomized controlled trials (RCTs) for anxiety disorders for both adults and youth (Hofmann & Smits, 2008; Kendall & Sugarman, 1997; Pina, Silverman, Weems, Kurtines, & Goldman, 2003), along with the observation that such rates may be even higher in the real world (Westen & Morrison, 2001), it is clear that many patients do not even complete treatment.

For those who remain in treatment, within-session participation may be suboptimal, thus negating the benefits provided by CBT. While coming to treatment out of one's own volition indicates a desire for symptom relief, patients display differing levels of willingness to change behavior, which can affect willingness to participate in the procedures that achieve such change. In CBT, such procedures can sometimes be difficult. Given that patients have often gone years with the same behavior patterns of anxiety, which often are seen as a protective mechanisms against harm (Barlow, 2002), it is not surprising that in-session participation would at times be suboptimal.

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Moreover, factors outside of the treatment session itself can further attenuate the effects of CBT. Given that patients often spend only 1 h per week in treatment, they have 167 other hours in the week that can serve to either improve or reinforce their symptoms. Patients can spend this time working to generalize the components of therapy, or they may revert back to the same behaviors that maintain their anxiety. CBT often addresses this situation through the assigning of homework (Huppert, Ledley, & Foa, 2006). Indeed, patients who spent more time doing homework between sessions have been observed to have better outcomes for agoraphobia (Edelman & Chambless, 1993), and early compliance with homework has also been associated with more positive outcomes for social phobia (Leung & Heimberg, 1996) and obsessive-compulsive disorder (OCD; Araujo, Ito, & Marks, 1996). However, homework completion has been observed to be suboptimal (Kazantzis, Deane, & Ronan, 2004). Helbig and Fehm (2004) surveyed 77 CBT therapists, who identified that 74.5% of their patients displayed difficulties in completing homework and indicated that only 38.9% were fully compliant. This is particularly unfortunate given the substantial influence homework has on CBT treatment; in a recent meta-analysis (where 57% of the studies focused on anxiety; Kazantzis, Whittington, & Dattilio, 2010), homework was observed to have a large effect size in treated groups ($d = 1.08$) and presented an incremental improvement over no-homework conditions ($d = 0.48$).

In considering these external factors, it becomes readily apparent that treatment for anxiety disorders goes far beyond applying CBT to patients. While the principles of CBT have been established with rigorous scientific control in the laboratory, other factors are involved in transitioning them to the field. Thus, the core elements of CBT are empirically justified and necessary but also are not sufficient in order to achieve symptom relief. A further examination of issues that can moderate and mediate the effects of CBT on symptomology is warranted.

Factors That Contribute to Complexity

A number of factors contribute to treatment complexity for treatment of anxiety disorders for a variety of populations. At present, practitioners have access to treatments that work well but require patients to be engaged, and traditional CBT manuals provide little guidance regarding how to achieve such engagement. Given the aforementioned difficulties of engaging patients in session and outside of sessions (via completing homework), many patients receive an inadequate dose of this efficacious treatment. Several hypothesized barriers that contribute to the complexity in treating anxiety disorders are ambivalence in motivation for behavior change, mismatch of clinician and patient, and limited therapeutic alliance. Indeed, clinicians have reported that two substantial barriers to treatment for panic disorder are forming an alliance with patients and having patients present for treatment with limited motivation (Goldfried, 2011). If a patient has less than full conviction to leave his or her avoidant habits behind, feels mismatched with the clinician, or does not feel connected with the therapist (i.e., a poor therapeutic alliance), this likely will lead to difficulties in engaging in the treatment. In the sections that follow, we review the evidence supporting how these factors contribute to the complexity of treating patients struggling with anxiety disorders.

Motivation

Motivation for behavior change is a pivotal factor in a variety of psychological treatments, and anxiety disorders are no exception. Given that human beings typically seek homeostasis and stability (Monroe, 2008), it should not be surprising that patients have some reluctance to make changes in any behavior, including their ingrained habits which have provided comfort but also have maintained anxiety symptoms. Motivation is often construed in the context of patient readiness for behavior change where such readiness is presented in a framework of multiple stages, where

patients may have not considered change (precontemplation), may be considering change (contemplation), may be actively planning change (preparation), or are taking action and/or working to maintain such change (action and maintenance, respectively; Prochaska & DiClemente, 2005). This conceptualization of motivation eschews the notion of the construct being an inherent trait and rather construes it as one that is modifiable over time. Motivation for behavior change first received major attention in psychological treatments when addressing problematic alcohol use (Miller, 1983) and has been identified as presenting barriers to treatment for conditions such as diabetes, asthma, and AIDS (Rubak, Sandbæk, Lauritzen, & Christensen, 2005). Oftentimes, patients seek relief for conditions and have initial motivation to work toward symptom management, but difficulties in engaging in the tasks necessary for such relief can present impediments to symptom reduction. Thus, the degree of commitment to change can attenuate the effect of the prescribed intervention on the target outcome.

Similar parallels can be drawn in the role of motivation for behavior change in CBT for anxiety disorders. Many patients come in for treatment where they commit a substantial amount of money and time for services, but even then, substantial patient effort both within and outside the session is required in the context of competing life priorities. In psychological treatments as a whole, those identified to be at the precontemplation stage of readiness for change have been indicated to not fare as well in treatment as those in the contemplation or action stages (Krebs, Prochaska, & Norcross, 2011), and there is no reason to indicate that these findings should differ in anxiety disorders. In a recent survey of therapists who treat panic disorder, 67.1% of participating therapists reported initial motivation from patients as a common barrier to treatment (Goldfried, 2011). Patients low in motivation have been observed to have poor treatment outcomes for manualized CBT for panic disorder, no matter how well the therapist follows the outlined procedures (Huppert, Barlow, Gorman, Shear, & Woods, 2006). Similarly, low motivation for treatment participation has predicted poorer treatment outcomes in generalized anxiety disorder (GAD; Dugas et al., 2003). Ambiguity in commitment to change has been indicated to be as a particularly troublesome barrier in the treatment for compulsive hoarding, and suboptimal outcomes in a recent open trial for compulsive hoarding were attributed to problems with completing homework in the context of such motivation (Tolin, Frost, & Steketee, 2007). While many patients may have sufficient initial motivation to achieve benefit from CBT for anxiety disorders, a large number of patients appear to experience ambivalence and outright resistance to treatment, which are complications that are typically not addressed by traditional CBT.

In considering the issue of patient motivation for patients with anxiety disorders, it is important to understand that it is not necessarily that patients low in motivation have no motivation to change at all but rather that they often experience ambivalence related to conflicting feelings about enacting change. Anxiety disorders differ from alcohol and substance use regarding the sources of ambivalence, as many patients with anxiety disorders do not want to participate in behaviors that are characteristic of their psychopathology (unlike substance use) but may have ambivalence about the specific tasks required to achieve relief (e.g., exposure; Slagle & Gray, 2007). While the major part of the motivational balance in alcohol use hinges on the adverse consequences of alcohol use in comparison to the enjoyment of drinking, in anxiety disorders the primary decisional balance is that changing via CBT offers a more distal goal of eventual anxiety reduction in comparison to the immediate salience of fear, which is immediately reduced by safety behaviors (that are the opposite of CBT-prescribed behaviors). Paradoxically, exposure increases distress in the short term compared to safety behaviors which escape stressors and give an initial sense of comfort but then lead to adverse distal consequences via negative reinforcement of safety behaviors (which maintain stimuli as anxiety provoking). Indeed, it is logical that patients with anxiety disorders would have ambivalence with regard to enacting procedures for changes in thinking and behavior. Problems that bring people to seek

treatment do not solely cause discomfort but also involve these protective but ineffective safety behaviors (both cognitive and behavioral) that temporarily alleviate anxiety-related distress with an associated long-term cost. For example, individuals with GAD report worry to be problematic but also simultaneously to serve as a protective mechanism (Borkovec & Roemer, 1995). For such patients, worry helps to prevent from forgetting and acts as a mechanism to exert perceived control over circumstances. Similarly, patients with agoraphobia who have concern over having public panic attacks and patients with OCD who have concern with becoming ill engage in unproductive avoidance behaviors in an attempt to protect from such undesired outcomes. Thus, patients with anxiety disorders may have ambivalence in simply abandoning such behaviors that have provided a great deal of comfort for them despite their adverse consequences in the long run. Such ambivalence in behavior change is one of the reasons that patients with panic disorder, social phobia, and OCD have been found to enter CBT with some hesitancy (Buckner & Schmidt, 2009; Dozois, Westra, Collins, Fung, & Garry, 2004; Maltby & Tolin, 2005). A further threat to motivation is the high comorbidity rate of anxiety disorders with depression (Kessler, Chiu, Demler, Merikangas, & Walters, 2005) and the associated anhedonia and reduced motivation to participate in a variety of activities that come with depressed mood. Motivation for these comorbid patients may be further impaired with regard to engaging in treatment for anxiety.

Therapist-Patient Mismatch

> What treatment, by whom, is most effective for this individual with that specific problem, and under which set of circumstances? (Paul, 1967, p. 111)

Gordon Paul's seminal quotation still resonates today in a variety of fields, with treatment for anxiety disorders being particularly relevant. A mismatch between therapist and patient is of specific interest, as currently there are a variety of efficacious treatments for anxiety disorders (i.e., "what treatment for which specific problem"), but less research has focused on them being delivered "by whom" and "for this individual," along with the interaction between these two variables. Patients and therapists can mismatch on a variety of issues, which may lead to attenuation in motivation for change, therapeutic alliance, and overall treatment outcome. For example, if a therapist focuses on psychoeducation before a patient is ready while the patient is still internally wary of the consequences of giving up safety behaviors (a mismatch on readiness to change), this can increase negative perceptions about treatment and thus tip the decisional balance away from change (reducing motivation) and result in a disagreement/mismatch on treatment goals (reducing the therapist-patient alliance). Reduced motivation to change and a poor alliance could then work to attenuate overall treatment outcome. It is important to note that therapist-patient mismatches do not occur in isolation but rather in the context of a variety of issues when applying CBT with a particular patient.

Research in psychotherapy has traditionally divided variables for matching in terms of therapist characteristics (Beutler et al., 2004), patient characteristics (Clarkin & Levy, 2004), content and tailoring of specific interventions (Griner & Smith, 2006), and therapist behaviors in the context of therapist-patient interaction (Beutler et al., 2004). Patient and therapist factors can be further classified in terms of basic demographics (e.g., SES, gender, age, ethnicity), psychological variables (e.g., patient presenting problem, patient personality traits, level of impairment, patient insight into symptoms), and the values, beliefs, attitudes, and cultural history of all parties in treatment. Given these characteristics, therapist behaviors and the use and adaptation of interventions may differ depending on patient and therapist characteristics in order to achieve an optimal match.

Therapist-patient mismatches can be divided into two major categories: structural mismatches, which refer to mismatch on preexisting characteristics over which the therapist has little control (e.g., ethnicity, gender), and behavioral

mismatches, which refer to mismatch of specific behaviors over which the therapist exerts direct control (e.g., interpersonal style, therapeutic expectations). Hypotheses regarding the effects of structural mismatch often trace their empirical origins to Festinger (1954), who found that people often prefer and identify with others who are similar to themselves, relative to those who are more dissimilar (and thus are more mismatched structurally). The notion of behavioral mismatch has been highlighted by Beutler et al. (2004), who have emphasized specific therapeutic approaches for specific patient characteristics under the umbrella of "prescriptive psychotherapy." Tailoring treatments for idiographic patient care has also become of increasing importance in the strategic aims of the National Institute of Mental Health (Insel, 2009) in recent years, as psychotherapy moves beyond a "one-size-fits-all" approach. As patients with anxiety disorders are a heterogeneous group (Kessler et al., 2005), such efforts to identify and ameliorate mismatches are also of increasing relevance for these patients as well. In this section, we aim to identify a variety of potential structural and behavioral mismatches for patients with anxiety disorders.

The two most readily identified structural mismatches involve race/ethnicity and gender. Current indications are that racial and ethnic mismatch may not predict distal outcomes in psychotherapy (e.g., symptom reduction) but may predict proximal outcomes such as treatment retention and comfort in participating in therapy (e.g., Zane, Hall, Sue, Young, & Nunez, 2004). Research into racial/ethnic matching effects in CBT for anxiety is extremely sparse, with one study directly evaluating its impact. Rosenheck, Fontana, and Cottrol (1995) identified that when African-American patients with posttraumatic stress disorder were matched with Caucasian therapists, increased termination rates were observed both after the first session as well as before the completion of the 12-week treatment protocol relative to those patients who were paired with African-American therapists. Additionally, these Caucasian therapists rated the same patients as having reduced clinician-rated commitment to treatment relative to Caucasian patients who matched their clinician on ethnicity. However, these effects were not seen when pairing Caucasian patients with African-American therapists. Precisely disentangling these effects would require further prospective work given the confounding concept of pretreatment commitment to participate, as once this variable was included in the model along with treatment type, the ethnic matching effects were no longer observed. Nonetheless, these results do coincide with other findings that African-Americans are more likely than other racial groups to prematurely terminate treatment (Diala et al., 2000; Sue, Ivey, & Pedersen, 1996) and, in multiple other instances, ethnicity has been identified as a predictor of treatment retention in both adult and child therapy (Miller, Southam-Gerow, & Allin, 2008; Sue & Lam, 2002). A variety of ethnic and racial groups have cited treatment stigma and apprehension in working with a treatment provider as the main reason for not seeking psychological treatments and have also reported these attributes at elevated levels relative to Caucasians (U.S. Public Health Service, 1999).

Despite the dearth of evidence for ethnic mismatch and its effect on treatment, its consequences on the treatment process can be significant. For example, consider the situation where a therapist treats a native Irish patient with compulsive hoarding, who comes from a cultural subgroup that values minimal waste and reuse of all items (possibly in response to limited resources) and has some disdain toward regular discarding of items. In this case, simply proceeding with treatment to reduce such behavior would be inadvertently invalidating to cultural values. Additionally, some ethnic minority groups may emphasize a more somatic presentation of anxiety disorders (U.S. Public Health Service, 1999), and failure to recognize these symptoms as consistent with a traditional anxiety disorder presentation could lead to clinician-patient disagreement/mismatch on what to treat and the best way to approach treatment.

With regard to gender match, early studies evaluating this construct suggested that patients stayed in therapy longer or had better outcomes when patients and therapists matched on gender.

However, closer examination of these studies showed numerous methodological flaws. In more recent, methodologically stronger studies, gender match has not been indicated to be an overall predictor of treatment dropout or outcome over a variety of conditions (Beutler et al., 2004), and the one study of this concept in anxiety disorders also did not find gender match as a predictor of outcome in CBT for panic disorder (Huppert et al., 2001). Nevertheless, given the limited research base with anxiety disorders, potential for mismatch on gender issues does exist. For example, a clinician treating a postpartum female with OCD could easily forget that mothers of children who have not yet received vaccinations are often instructed to keep a relatively clean environment for their children. Thus, a new mother with OCD could view a male therapist who challenges notions of appropriate cleanliness as insensitive and lacking proper understanding of the issues being dealt with. Such a process could lead to a rupture of the therapeutic alliance which the mother attributes to the therapist's gender mismatch.

While structural mismatches exist regardless of any behavior of the individual clinician, behavioral mismatches depend on the behavior of the clinician once he/she is in the presence of a patient. One common mismatch that can occur in CBT treatment for anxiety disorders is rooted in the decision of when to implement specific CBT procedures during treatment (Kendall & Chu, 2000). Adhering too closely to a treatment manual with a patient who wishes to discuss emerging personal issues can result in mismatch on session and overall treatment goals. The therapist following a manualized treatment too closely could force an agenda of exposure to anxiety-provoking stimuli while neglecting a patient goal to address recent matters aside from anxiety that have occurred in the patient's life. Adhering too little to the manual, however, can also lead to mismatch on session and treatment goals. A therapist feeling the need to be responsive to every issue raised by a patient can end up neglecting standardized procedures that address the main goal of symptom remission, thus delaying the associated benefits and leaving the patient to wonder when the therapist will adequately address their primary presenting issues. Consistent with the principal that match is based on a flexible balance between patient and therapist agendas, Hogue et al. (2008) found that moderate levels of adherence to a treatment protocol predicted the best treatment outcomes for adolescents with externalizing problems, especially in comparison to those therapists who displayed very high or low adherence levels. Moreover, in treatment for panic disorder, Huppert et al. (2006) found that high protocol adherence was associated with poorer outcomes among those who had low motivation for behavior change. Kendall and Chu (2000) have found that therapists vary in how flexibly they deliver a manualized treatment, which is not necessarily a nuisance confound if some patients require differing levels of protocol deviation. A therapist must have grounding for the specific treatment that he/she implements but also must be careful regarding how and when to implement it with a specific patient. Clinicians are thus presented with a balancing act, where they are tasked with implementing CBT to reduce symptoms of anxiety while simultaneously addressing other patient goals.

A related issue where a therapist can mismatch with a patient is on the method of presentation of treatment roles to a patient. On one end of the spectrum, therapists can work as collaborative partners, whereas on the other end it is more an authoritative style. Here, matching is in context of the notion of patient reactance, which refers to a style of responding to an authority figure (where high reactance corresponds to defiance in the face of authority and low reactance corresponds to submissive behavior; Brehm, 1966). Thus, a patient who is high on reactance would match poorly with a therapist who takes an authoritative stance, while a patient low in reactance may match poorly with a therapist who is well meaning in a collaborative style but does not provide sufficient direction. Over a variety of treatments, it has been observed that patients high in reactance benefit from less structured treatment, while those in low reactance benefit from more therapist guidance and structured therapy (Beutler, Harwood, Michelson, Song, & Holman,

2011). The implications of these findings quickly become pertinent in CBT for anxiety, given that patients sometimes require urging by clinicians to engage in exposures. Patients high in reactance may bristle at being strongly encouraged into conducting exposures and may refuse to participate, or they may acquiesce to participating half-heartedly in session and not practice at home. Conversely, patients low on reactance may work better with clinicians who use authoritative expectations of success to progress the patient through an exposure hierarchy.

Another issue to consider in behavioral mismatches is the patient's perception of what treatment ought to be and how quickly it will work and whether therapist behaviors match these expectations. Patient expectations of treatment may play a powerful role, as they have been indicated to predict better CBT outcome for several anxiety disorders (through a pathway of increased homework compliance and early symptom improvement; Westra, Dozois, & Marcus, 2007) and have been indicated to mediate SES-dropout relationships (Pekarik, 1991). Indeed, establishing positive expectancies for treatment outcome has been related to stronger rates of symptom reduction in fear of flying (Price, Anderson, Henrich, & Rothbaum, 2008). With regard to such outcome expectancies, patients who expect no relief may feel a mismatch if their therapist does not validate their view (e.g., portraying relief by exposing oneself to feared situations as straightforward and easy to those who have struggled for years with various fears). On the other hand, those who expect immediate relief would experience mismatch if symptom reduction was not immediate but experienced over a period of weeks. With regard to matching on expectations on treatment procedures, patients may have differing views from their therapists for their expectancies of what treatment consists. While increasing in popularity, many patients may still not be well aware of the principles of exposure treatments and CBT in general (Westra, Aviram, Barnes, & Angus, 2010) and may instead expect long hours on a couch for a period of years. When the therapist starts explaining about CBT skills and procedures, the patient may experience a mismatch of treatment expectations. Patients may be unaware of how much of the focus of CBT is on their participation, expecting the therapist to do a preponderance of the work. Thus, such mismatches between what a therapist and patient may expect in therapy could be troublesome.

Patients and therapists can also mismatch on patient-specific preferences for treatment procedures, and conversely achieving such a match can serve to assist in treatment implementation. With regard to overall treatment choices for a broad array of psychological conditions, a small but consistent effect has been observed for matching treatment techniques to patient preferences (in contrast to not matching at all). Some ethnic and racial minority patients have displayed a propensity for alternative treatments as opposed to traditional psychological treatments, such as self-help methods or consultations with religious or other group leaders (Thomason, 2000; Thompson, Bazile, & Akbar, 2004), and this may well be true for some patients from majority groups as well. These findings create a challenge for clinicians when treating patients with anxiety disorders, where CBT prevails and other effective behavioral treatment options are not readily available. This presents a continued challenge to the clinician who may feel compelled to implement a CBT framework while negotiating a variety of potential patient preferences.

In a similar vein to the consideration of outcome, role, and procedural expectancies is the potential mismatch with the patient in the content and pace of exposures, with particular regard to pairing exposures carefully to a patient's hierarchy of fears. It is possible that the clinician may want to move up the hierarchy too rapidly, thus risking mismatch with the pace and content that the patient is ready or willing to address. With this mismatch, the patient may resist participation in the exposure or may attempt an exposure that is perceived as too difficult, which can lead to patient withdrawal during the exposure. This can lead to additional consequences such as reduction of patient self-efficacy, adversely affecting any future willingness to participate in exposures. On the other hand, if the clinician chooses an exposure pace that is slower than what

the patient desires, this mismatch can lead to patient frustration with rate of progress, which can also lead to treatment dropout. Also, exposures that are not matched to specific patient concerns/fears may not be perceived as particularly relevant and thus not of optimal benefit to a patient. For example, a patient with a fear of snakes may have little difficulty encountering a harmless snake in the clinic, but only in his backyard does he experience the necessary arousal to begin emotional processing, which is believed to be necessary to achieve extinction of anxious symptomology (Foa & Kozak, 1986). However, a patient may not verbalize this unless a clinician is attentive and ensures that the content of exposures is pertinent to the patient.

Also related to appropriate matching on expectancy for treatment procedures is agreement on the rationale behind treatment, as some patients may not be fully understanding of or agree with the cognitive behavioral model of anxiety. The importance of conveying a rationale to patients has been emphasized for a variety of treatments (Addis & Carpenter, 2000; Wampold, 2001). With respect to anxiety disorders, patient agreement with treatment rationale has been indicated to predict adherence to within-session exposure procedures and better treatment outcomes for OCD (Abramowitz, Franklin, Zoellner, & DiBernardo, 2002) and improved outcome in treatment for GAD (Borkovec, Newman, Pincus, & Lytle, 2002). In finding agreement on the treatment rationale for CBT, careful consideration may be necessary in the context of the CBT methods of cognitive restructuring and challenging maladaptive thoughts, which involve challenging thinking that has often served a function for the patient in the past. Telling patients who do not match therapists in their belief in the treatment rationale that their thinking is maladaptive can be a potential source of invalidation (Addis & Carpenter, 2000), which can interfere with the therapeutic alliance.

In psychological treatments for pediatric anxiety disorders, mismatch with parents also needs to be addressed, as they are essential participants in therapy. If a child is in the therapy office for 1 h per week, he/she has 167 other hours during the week without the therapist. Parents can either accommodate the avoidant behavior often seen in anxiety disorders (and thus maintain symptoms; Storch et al., 2007), or conversely they can augment treatment by becoming "co-therapists" and being strong advocates to reduce avoidance and achieve full symptom remission (Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008). For example, if parents are not on the same page with therapists as far as treatment rationale and agreement with the tasks to be performed during treatment, this can undermine the in-treatment procedures. It can be difficult for parents to see their children endure marked distress from anxiety, and a natural instinct can be to provide short-term comfort and accommodation. Without proper understanding of the rationale of treatment, this tendency to accommodate can be difficult to resist, leading to poorer longer-term outcome. Furthermore, parents matching with clinicians regarding their role in treatment is also essential, as some parents may wish to drop off their child at therapy and remain relatively uninvolved, while on the other end of the spectrum, some may wish to be active participants in every aspect of treatment. Mismatch on such parental role expectations can preclude implementation and generalization of treatment and perhaps even lead to parental withdrawal of their child from therapy. Incorporating parents and adapting treatment to child needs in CBT for pediatric anxiety disorders raise additional factors to consider in comparison to conventional treatment for adult anxiety disorders.

Several other issues with regard to patient-therapist match include mismatch on perception of the problem (as therapists, parents, and patients often have different perceptions of the priority problem; Grills & Ollendick, 2003), coping orientation (e.g., active in solving problems or avoidant in addressing challenges), and interpersonal style (e.g., a high-energy therapist being a poor match for a low-energy patient). As can be seen, a series of pitfalls await therapists implementing treatment for anxiety disorders for a heterogeneous patient population. Navigation of these potential mismatches has direct implications for engaging

patients in CBT and in developing a working treatment relationship.

Therapeutic Alliance

The sections on patient motivation and therapist-patient mismatch reveal that treating youth and adult anxiety disorders is more complex than just having a clinician select and implement an EST. An unmotivated patient will not be suddenly motivated by the clinician starting to implement an EST. Similarly, a patient who is mismatched with the therapist on matters such as role and outcome expectations also will not just disregard the mismatch because the clinician has evidence-based treatments at his/her disposal. These points demonstrate that an important additional element of treatment, the therapeutic alliance, must be taken into account when treating patients with anxiety disorders.

The therapeutic alliance has been defined as a working relationship between a therapist and a patient in which the patient feels an affective bond with the therapist and agrees with the therapist on the goals and therapeutic tasks of treatment (Bordin, 1979). It should be noted that present research supports these three factors with adult patients but has not supported these components as making up elements of the alliance with youth patients (Zack, Castonguay, & Boswell, 2007). Karver, Handelsman, Fields, and Bickman (2005) suggested that the therapeutic alliance in youth therapy consists of an emotional/affective connection (e.g., bond, trust), a cognitive connection (e.g., agreement on goals, hopefulness), and a behavioral connection (e.g., collaboration on tasks and other forms of patient participation). More recently it has been suggested that youth treatment participation may be a separate construct that follows from or is facilitated by the affective and cognitive elements of the alliance (Shirk & Karver, 2011). An additional element that needs to be attended to uniquely in youth therapy is the therapist-parent alliance (Karver et al., 2005).

Studies of the therapeutic alliance in adult and youth treatment have revealed that the therapeutic alliance is a robust predictor of treatment outcome (Martin, Garske, & Davis, 2000; Shirk & Karver, 2011). Consistent with the general treatment literature, the alliance has been found, predominantly in CBT studies, to predict proximal (e.g., change in cognitions) and distal (e.g., change in anxiety symptoms) outcomes in the treatment of multiple anxiety disorders (e.g., Casey, Oei, & Newcombe, 2005; Hayes, Hope, VanDyke, & Heimberg, 2007; Langhoff, Baer, Zubraegel, & Linden, 2008; Newman & Stiles, 2006; VanDyke, 2002; Vogel, Hansen, Stiles, & Gotestam, 2006) and youth anxiety disorders (e.g., Chiu, McLeod, Har, & Wood, 2009; Creed & Kendall, 2005; Hughes & Kendall, 2007; McLeod & Weisz, 2005).

Although most studies have found a positive therapeutic alliance related to therapeutic gain in treatment of anxiety disorders, a handful of studies have not found this relationship (Kendall, 1994; Kendall et al., 1997; Southam-Gerow, Kendall, & Weersing, 2001; Woody & Adessky, 2002). It has been suggested that this may be because the anxious patients in these studies reported very high and/or increasingly high therapeutic alliance ratings, thus resulting in limited variability (Kendall, 1994; Ramnero & Öst, 2007). It may be that anxious patients are quite vulnerable to a social desirability bias, where they worry about the consequences of not reporting a positive alliance. Another possibility for why alliances may be highly rated could be that the anxious patients may be very eager to be approved by their therapist and thus readily form a relationship with the therapist.

A patient-therapist alliance is likely an integral element that needs to be addressed in successful CBT for anxiety disorders. While the primary component of CBT for anxiety disorders involves exposure, this process requires getting patients who are often shy, avoidant, and inhibited to approach rather than escape from that which is directly related to their disorder. Without a healthy alliance, the therapist may be unable to convince the anxious patient (or the patient's parents in a youth treatment case) to trust enough to cooperate and expose him/herself to a feared stimulus, especially the first time when the patient

has not yet experienced proof that doing something that seems illogical to them due to their cognitive biases (and may seem cruel to parents) is actually helpful (Hayes et al., 2007; Langhoff et al., 2008). It may be that an initial emotional connection with the therapist, one that establishes the therapist as safe and trustworthy, needs to be made in order to facilitate a patient's willingness to listen to the therapist explain the treatment rationale. This is not to be taken for granted given that anxious patients likely question whether or not the therapist likes or respects them (VanDyke, 2002). A well-explained treatment rationale with proper orientation and clarifications as to what to expect can establish therapist trust, credibility, and hopefulness that will be vital when a patient starts an emotionally demanding exposure where the therapist supports and encourages the patient to endure discomfort while preventing escape from the threatening stimulus (Caron & Robin, 2010). In fact, the therapeutic alliance has been found related to patient participation in treatment for anxiety disorders (Liber et al., 2010) and, more specifically, patients' willingness to expose themselves to higher anxiety-provoking situations and to stay in the situations long enough to habituate (Hayes et al., 2007). It logically follows that the emotional bond elements of the alliance would decrease during an exposure due to the unpleasant feelings experienced, leading to the patient feeling upset at the therapist for making them experience the uncomfortable feelings. However, the more cognitive aspects of the alliance, focusing on negotiating agreement with the patient on goals and tasks to achieve those goals and encouraging the patient to remain in an exposure, even if they are voicing concerns that they cannot do it or are failing, may contribute to why a reluctant patient would continue to participate in the exposure and not drop out of the exposure and/or the treatment (Kendall et al., 2009). This probably explains why both youth and adult patients receiving an exposure treatment for anxiety disorders would attribute treatment success to features of the therapeutic relationship (Kendall & Southam-Gerow, 1996) and why the therapeutic alliance has been found related to anxious patients' ratings of session helpfulness (Hayes et al., 2007). Further, the alliance likely also facilitates patient willingness to learn and practice additional CBT therapeutic skills both in session and at home (Chu & Kendall, 2004). This is additionally important given that patient willingness to learn new coping skills and participation in treatment (including homework) have been found to be related to treatment outcome (Chu & Kendall; Edelman & Chambless, 1995; Huppert et al., 2006; Karver, Handelsman, Fields, & Bickman, 2006; Kazantzis et al., 2010).

In addition, it should be noted that the parent-therapist alliance has also been found related to treatment dropout (Hawley & Weisz, 2005) and improvement in youth anxiety symptoms (McLeod & Weisz, 2005). This is not surprising given that anxious youths have frequently been found to have anxious parents (Bienvenu, Hettema, Neale, Prescott, & Kendler, 2007). By forming an alliance with the parents, the therapist may be able to overcome parental anxieties (such as embarrassment that their child has a problem or anxiety about being evaluated negatively by the therapist) about bringing their youth to treatment and about seeing their child be required to face anxiety-provoking situations. The clinician may even be able to involve the parents in the treatment, which has been found to result in better youth treatment outcomes (e.g., Mendlowitz et al., 1999).

Approaches to Address Issues with Alliance, Motivation, and Therapist-Patient Mismatch Building Alliance

Although it is beneficial that much of the treatment literature and many treatment manuals now mention the importance of forming a therapeutic alliance with an anxious patient, they neglect to address how this should actually be done. In fact, the therapeutic alliance is often confusingly addressed as if it were a treatment approach rather than the product or result of treatment techniques. Indeed, the common terse recommendation for a clinician to "form an alliance" with a patient is not particularly informative. Fortunately, research has attempted to identify therapist behaviors that

predict the formation of a positive therapist-patient alliance.

In particular, therapist interpersonal skills such as empathy, warmth, and genuineness have repeatedly been found to predict the therapist-patient alliance and patient engagement in both youth and adult treatment (Horvath & Bedi, 2002; Karver et al., 2006). Therefore, not surprisingly, these same interpersonal skills have been recommended as playing an important role in a therapist getting an anxious patient to participate in cognitive behavioral treatments (Langhoff et al., 2008). Surprisingly little research has examined and demonstrated the relationship between therapist empathy and the therapeutic alliance in the treatment of anxiety disorders (e.g., DeGeorge, 2008). However, indirect evidence in the form of the relationship between interpersonal skills relating to symptom improvement in anxiety disorders suggests that these skills help build the alliance with these patients (e.g., Keijsers, Schaap, Hoogduin, & Lammers, 1995; Newman & Stiles, 2006). When using these skills, it is important that therapist provides an interest in the patient that is genuine, as clinician overemphasis on commonality with the patient can come across as inauthentic and has been found to negatively predict the therapeutic alliance (Creed & Kendall, 2005). On the other end of the spectrum, clinicians who are overly formal, didactic, directive, and who grill their patients with questions about anxiety and anxiety-provoking situations, especially early in treatment, have been found to form poor alliances and/or have worse outcomes with anxious patients (Creed & Kendall; Keijsers et al., 1995). Thus, it is likely that at treatment onset, therapist rapport-building behaviors that focus on building an affective bond/emotional connection, such as empathy, respect, validation, acceptance, and genuineness, will be needed for an anxious/avoidant patient to initially engage and open up with a therapist. This would be the clinician listening to the anxious patient and showing understanding by validating the patient's experience such as acknowledging how difficult the patient's feelings are and how challenging and painful anxiety-provoking situations seem. The clinician would make statements that it makes sense how the patient feels and acts given past or current circumstances, automatic thoughts that are generated, and situations that have been experienced. Instead of challenging patient reluctance, the clinician would validate reluctance and avoidance and acknowledge that the patient's reluctance makes sense given how certain situations make the patient feel.

However, the therapist will need to switch to other alliance-building behaviors as therapy progresses. Keijsers et al. (1995) found that empathic listening in the third vs. the first session was related to worse therapeutic outcomes, while directiveness/guidance later in treatment predicted better outcomes. This makes sense in that while initially a patient needs to feel understood and accepted, eventually the patient needs the focus of therapy to shift toward change. Without more direction from the clinician, therapy will seem purposeless and avoidant of change (i.e., it will appear that nothing will be done to actually deal with the patient's anxious symptoms). The therapist of course should still be supportive and caring, as this has been found to be related to patient willingness to enter anxiety-provoking situations (Williams & Chambless, 1990).

An additional related therapeutic approach to engaging anxious patients that has been suggested as very important is for the therapist to take a teamwork-oriented collaborative stance with the patient (Caron & Robin, 2010; Chu, Suveg, Creed, & Kendall, 2010). With this approach, the therapist collaboratively sets goals with the patient and works together with the patient to determine the treatment tasks and the pace of treatment. In order to do this, the therapist must listen to and validate the patient's perspective about treatment and then be flexible in how treatment is conducted with the patient (Shirk & Karver, 2011). Consistent with this, Creed and Kendall (2005) found therapist collaborativeness to predict later ratings of the patient-therapist alliance while Chu and Kendall (2009) found therapist flexibility to predict patient involvement for anxious patients. A flexible therapist could implement a treatment for an anxiety disorder but flexibly adapt

the treatment in a way to make it more interesting (e.g., changing typical manual examples or handouts), more responsive to newly presenting patient issues (e.g., use presenting issues as examples for use of skills being taught), and more applicable to patient needs and goals for the patient. These techniques can serve to more easily retain the patient's attention when implementing the treatment (Connor-Smith & Weisz, 2003). In addition, the flexible therapist would pace the treatment such that the patient is not forced to engage in a treatment component (such as exposure) before being ready.

Of course, in order for an anxious patient to feel like an equal member of the therapeutic team, the patient needs to fully understand the treatment approach that the therapist is suggesting. Thus, the therapist needs to carefully explain to the patient the rationale behind the treatment procedures, orient the patient to each of their roles, and explain to the patient how the treatment procedures will help to alleviate the patient's distressing symptoms. This is particularly important for treating anxiety disorders because exposure often sounds logically aversive to patients and not escaping also seems highly undesirable and initially can appear illogical to an anxious patient. The therapist needs to convincingly explain that these procedures, although causing short-term discomfort, will result in the patient feeling less anxious eventually. Only then will the therapist have successfully built the cognitive connection component of the alliance (e.g., willingness to engage) with the patient and an expectancy/hopefulness for change. Ahmed and Westra (2009) detail this process, where an adequately presented treatment rationale is used to increase the likelihood of anxious individuals participating in exposure to anxiety-provoking situations. Accordingly, patient acceptance of the clinician's treatment rationale has been shown to be related to treatment outcome (Addis & Carpenter, 2000). Along similar lines, patient ratings of treatment credibility (i.e., the degree to which a treatment makes logical sense in that it can be helpful) has been repeatedly shown to be related to ratings of the therapeutic alliance and/or treatment outcome in treatment of patients with anxiety disorders (e.g., Borkovec et al., 2002; Newman & Fisher, 2010).

More broadly, part of therapists' being convincing that it is worthwhile to work with them is not just their explanation of the treatment rationale but how credible the therapists appear overall. Part of appearing credible to an anxious patient is that the therapist demonstrates a combination of expertise, confidence, trustworthiness, organization, and preparedness (Karver et al., 2005). Consistent with this, Williams and Chambless (1990) found that therapists perceived as more confident were more likely to have patients approach anxiety-provoking stimuli. Perceptions of therapist expertise, self-confidence, and directiveness have also been found related to treatment outcome (Keijsers et al., 1995).

Once the patient starts to participate in treatment and take part in exposures, the therapist's engagement work is not done. In addition to forming an initial alliance, the therapist will need to maintain the alliance during exposures. This would include not only reinforcing the patient for treatment participation but also looking to repair alliance ruptures when/if they occur. Reinforcing treatment participation to maintain the alliance does not have to mean giving patients tangible rewards but can be simply praising patient effort, as this principle has been associated with participation in exposure procedures (Gosch, Flannery-Schroeder, Mauro, & Compton, 2006; Shirk, Jungbluth, & Karver, 2012).

As for dealing with alliance ruptures in the treatment of anxious patients, this has been a relatively unexplored area. Newman, Castonguay, Borkovec, Fisher, and Nordberg (2008) found in a study of treatment for anxious patients that enhancing standard CBT with a component that to some extent targeted repairing alliance ruptures resulted in better outcomes for anxious patients than has been found for standard treatment on its own. It may be that during critical treatment junctures with anxious patients, when the patients are feeling misunderstood, pushed too much and too fast, and/or overwhelmed by what they are being asked to do and/or are considering abandoning an exposure or the treatment as a whole, it may be helpful for the therapist to

explore the interpersonal relationship between the patient and therapist (bringing attention to alliance ruptures, taking responsibility for contributions to the interaction, processing patient's affective experiences relative to the therapeutic interaction, etc.; Newman et al.; Safran, Muran, & Eubanks-Carter, 2011). Unfortunately the study did not measure treatment processes, so we are unable to know if the treatment attained its results through a hypothesized mechanism of improved alliances due to repaired alliance ruptures leading to better treatment participation, which would lead to better outcomes. Kendall et al. (2009) suggest that future research is needed to get a better understanding of rupture and repair sequences that occur in the treatment of anxious patients.

Finally, many researchers have suggested that similar engagement strategies (e.g., attentive listening, empathy, collaborating on goal and task agreement, clarifying treatment rationale and expectations especially during exposures) are likely necessary to engage and maintain the parents of anxious youths in treatment (Chu et al., 2004; Nevas & Farber, 2001). However, this is a severely neglected area of research in which suggestions for engaging parents are more based on clinical lore rather than actual research.

Treatment Approaches to Address Issues with Motivation for Behavior Change

The evidence base is more robust for addressing patient motivation for behavior change than for alliance formation. In addressing patient motivation, the most prominent intervention is known as motivational interviewing (MI). Motivational interviewing is not a set of circumscribed techniques but rather consists of a presentational style consisting of an empathetic, accepting, and nonjudgmental style based in patient-centered therapy but then incorporates a directive but collaborative approach toward enacting patient change (Lundahl & Burke, 2009). It does not serve to coerce an unwilling patient to participate but rather capitalizes on patient desire for positive change. This focus is based on the assumption that many patients are well intentioned and want to change for the better but often have difficulty identifying and overcoming barriers and ambivalence in enacting such change (Miller & Rollnick, 2002).

Evidence has accumulated over the past 30 years to support the efficacy of MI in a variety of conditions, and preliminary evidence for its conjunctive role with CBT for anxiety disorders has been accumulating over the past decade. Multiple meta-analyses have indicated support for the overall efficacy of MI (for a review, see Lundahl & Burke, 2009), and it has been observed that MI often exerts additive effects on other psychological treatments, providing a swift improvement in treatment adherence and session attendance (Hettema, Steele, & Miller, 2005). Additionally, MI has been successfully used to improve adherence to medical management for a variety of conditions (e.g., AIDS, diabetes; Rubak et al., 2005). Motivational interviewing focuses first on increasing motivation for change (through examining ambivalence for change, evaluating the relative importance of change in contrast to other goals, and evaluating and fostering self-efficacy) and then consolidating commitment to such change (through considering change options, goal setting, and planning to meet these goals). It is often very brief and can be provided in as few as 1–2 sessions. While full delineation of MI is beyond the scope of this chapter (see Miller & Rollnick, 2002 for more detail), we evaluate its components in the context of complications in treatments for anxiety disorders.

Basic MI tenets include expressing empathy and validation, developing discrepancy between life goals and behavior, rolling with resistance (i.e., identifying reasons for patient resistance and evaluating such reasoning, instead of directly combating it), and fostering self-efficacy. These concerns are particularly salient in treatment for anxiety disorders, as Issakidis and Andrews (2002) found that in evaluating reasons for abstaining from treatment among patients with anxiety disorders who had not sought therapy, 58% indicated that such refusal was reflected in a desire to manage their disorder without help,

20% indicated a reason in avoiding treatment was the fear of seeking help, and 14% indicated a rationale that clinical improvement was unlikely. In the context of treating anxiety disorders, MI principles such as developing discrepancy between life goals and behavior directly address those who express a desire to manage their pathology without intervention, empathy and validation are particularly pertinent to those who indicate a fear of seeking help, and fostering self-efficacy is relevant for those who believe that clinical improvement is unlikely. Patients may never have considered how feasible exposure is, as the habit of avoidance has been in practice for so long that they may consider exposure as impossible, despite the fact that that numerous patients with similar problems have successfully faced such feared situations with great benefit. It is important to foster such self-efficacy, as a mediational model has been supported where positive expectations for anxiety change at baseline predict homework completion, which subsequently predicts initial CBT improvement for anxiety disorders (Westra et al., 2007).

With regard to its application in anxiety, MI first appeared in the research literature in the form of case series (Westra, 2004; Westra & Phoenix, 2003). Subsequently, MI was applied as a part of a pilot trial by Westra and Dozois (2006) involving 55 patients whose principal diagnoses consisted of panic disorder (45%), social phobia (31%), and GAD (24%), where patients were randomized to CBT with or without a pretreatment MI intervention. Those in the MI condition displayed better homework adherence, higher expectancy for anxiety control, and a higher rate of treatment response than CBT alone, as the effect size for treatment response in the MI condition had an effect size of $d = 0.38$ compared to the control group (i.e., those who received CBT without pretreatment MI). Subsequently, Westra, Arkowitz, and Dozois (2009) found that MI increased homework compliance and outcome for GAD in a similar RCT for GAD where groups were randomized to conditions where they received with MI or no MI at pretreatment, while both received CBT. Buckner and colleagues (Buckner, Ledley, Heimberg, & Schmidt, 2008; Buckner & Schmidt, 2009) have indicated that MI interventions improve the likelihood of those with social phobia to seek treatment, which is particularly notable given that treatment process itself involves the feared condition of these patients (i.e., social engagement) and thus is of particular interest for this population. Motivational interviewing has also shown results in increasing enrollment in ERP treatment for OCD (Maltby & Tolin, 2005) and has helped with treatment adherence and treatment response with this condition (Merlo et al., 2009; Meyer et al., 2010).

To some degree, motivational interviewing stands in contrast to CBT for anxiety disorders, which often uses psychoeducation as a factual-based argument in convincing the patient to change, and certainly many patients are eager for information benefit from this approach. However, some patients may be reactant, where they feel as if a didactic approach can be an authoritative stance, which can lead to resistance (Brehm, 1976), and some research has indicated that people with a propensity for such reactance do not fare as well with CBT (Beutler, Rocco, Moleiro, & Talebi, 2001). Therapist behavior has been indicated to increase or decrease resistance (Patterson & Forgatch, 1985), and a therapist's relational style can indeed impact patient motivation (Norcross, 2002). The focus on psychoeducation in CBT for anxiety disorders can be greatly helpful but may not be a "one-size-fits-all" approach. In contrast, motivational interviewing approaches this issue by advising the patient to consider the pros and cons of changing. For example, many patients with anxiety want to improve but are unwilling to leave behind strategies which previously have been very protective for them (i.e., avoidance). Instead of providing more evidence for the success rate of treatment, an approach more consistent with MI principles would be to consider the pros and cons of retaining the current behavior pattern vs. changing. Many patients in this case can then recognize that while avoidance provides short-term relief (a reason for maintaining the status quo), it actually leads to longer-term discomfort (a reason for abandoning the status quo), whereas engaging in exposure provides short-term discomfort (a reason for

resisting change) but allows one to live a life unencumbered by excessive anxiety (a reason for enacting change). On the other hand, simply providing further evidence in support of CBT to a moderately reactant patient may simply elicit verbalization in defense of the status quo, which only solidifies commitment to avoidance and ambivalence toward full treatment participation.

A further consideration is that responsibility to address motivation is not restricted to pretreatment interventions. Given the observation from therapists that a substantial proportion of patients often experience reduced motivation after initial relief (Goldfried, 2011), a therapist must continually monitor a patient's motivation to fully engage throughout treatment, as it may fluctuate. For example, a patient may be more motivated to participate at easier stages of an exposure hierarchy but may have more ambivalence about approaching the more difficult stages of the hierarchy. A midtreatment addressing of motivation as higher points of the hierarchy are addressed may be appropriate for such patients. In this way, simply following an EST manual step by step can lead to the neglect of certain times where it is necessary to address patient motivation to continue treatment. Otherwise, partial response may be achieved (e.g., a patient achieves 50–75% of his/her hierarchy and then stops), or a patient completes his/her hierarchy in an obligatory fashion but then does not challenge him/herself outside of the therapy office both during and after the completion of treatment. Patients also may have differential motivation for varying components of treatment. For example, some may be afraid to initiate treatment, but once treatment begins, they complete homework fastidiously, while others may have perfect attendance but are not immediately willing to perform exposures at home. Still others may complete treatment but may not continue to perform exposures and revert to avoidance posttreatment, leading to relapse. Identifying specific barriers for each instance and what competing priorities may be interfering with full treatment adherence through techniques based in MI is thus appropriate. For example, one barrier when working with children can be that typical exposure exercises may be less engaging than when used with adults. These children may be initially motivated to change and participate during office sessions but find difficulty completing exposures at home. In response, one method that has been proposed to address this barrier involves employing technology in exposure protocols (e.g., video games, interactive media; Chu et al., 2004).

As can be seen, motivational interviewing principles can be used in a variety of situations to augment CBT for patients with anxiety disorders across developmental levels. For instance, rolling with patient resistance can help move patients away from the use of previously comforting and habitual patterns of avoidance. Similarly, fostering self-efficacy and continually looking for opportunities to validate patients' experiences can help them to feel empowered in making difficult behavior changes, and evaluating motivation throughout the course of treatment can prevent regression from treatment progress. The current state of research in increasing motivation for patients with anxiety disorders is nascent but growing rapidly, and traditional CBT for patients with anxiety disorders stands to benefit from its increased application in the coming years.

Treatment Approaches to Address Therapist-Patient Mismatch

While alliance and motivation are common factors that apply to treatment for all patients with anxiety disorders, idiographic variability remains for the relationships established between individual therapists and patients, which can lead to a variety of structural and behavioral mismatches. In looking to address such mismatches, the current evidence base for specific strategies in anxiety disorders is fairly sparse, despite sources suggesting that addressing therapist-patient mismatches are of substantial value in clinical practice (Castonguay & Beutler, 2006). Nevertheless, a variety of techniques can be employed to maximize match and foster personalized treatment within a manualized therapy framework. In addressing ways to address therapist-patient match and mismatch, we employ

the extant literature while acknowledging that a considerable amount of research remains to be done and that much knowledge on the topic has some basis in clinical judgment.

With regard to ethnic mismatch between patient and therapist, while it may have not been observed to directly attenuate outcome in CBT for anxiety (Newman, Crits-Christoph, Gibbons, & Erickson, 2006), a clinician must be aware of such mismatch insofar as it could affect patient receptivity to CBT procedures. For example, small cultural differences may exist which could lead to inadvertent damage to the therapeutic relationship or reduce the patient's motivation for change, possibly stemming from the clinician expressing or emphasizing views that are subtly culturally discrepant from that of the patient (e.g., minimizing a patient's focus on "stomach aches" and instead emphasizing cognitions as "classic anxiety," when the former topic is the patient's culturally acceptable way of expressing anxiety; Spendlove, Jackson, & Borrego, 2010). To address this, one recommendation is that therapists assess their own cultural value systems with regard to both overarching beliefs and their views toward various presentations of abnormal behavior, with a particular focus on how their views might affect treatment (American Psychological Association, 2003). This can facilitate rapid and effective in-session validation of the patient's experience of anxiety, which can be of great value in treatment for anxiety disorders (Welch, Osborne, & Pryzgoda, 2010). A complementary recommendation is to develop a cursory knowledge of the customs and beliefs associated with the patient's culture through reading relevant research articles (e.g., cross-cultural treatment studies of anxiety assessment and intervention), talking to local cultural experts, and evaluating other written and electronic sources while taking into account the evidence that within-culture variability is often greater than between-culture variability (Triandis, 1997) and evaluating the role, if any, such cultural values play with a specific patient. This approach can be also applied to a variety of structural mismatches, such as when working with a patient of the opposite sex or of an alternate sexual orientation. While evidence is lacking for direct intervention strategies for structural mismatches in the context of clinical trials for anxiety disorders, these converging sources provide a foundation for clinical practice.

With regard to behavioral mismatches, one common issue that arises involves matching the procedures delineated in a treatment protocol to the particular symptom presentation of an anxious patient. Indeed, a common complaint from clinicians (and perhaps patients also) is that treatment manuals can stifle creativity insofar as matching standardized procedures to the specific patient at hand (Addis & Krasnow, 2000). Two major approaches to address this issue are known as "flexibility within fidelity" (Kendall & Beidas, 2007) and modular treatment approaches (e.g., Weisz & Chorpita, 2012). The former approach espouses a philosophy of holding true to tenets of CBT (i.e., fidelity) while working within these basic parameters to adapt them to the specific patient at hand (i.e., flexibility). Modular treatment approaches address this issue by breaking down CBT treatments into varying components that can be delivered flexibly as opposed to sequentially with each patient who presents for treatment. Both frameworks aim to match core CBT tenets to patient-specific problems. For example, some patients may need specific examples or idiographic techniques in order to engage them in exposure. For instance, presenting the case of a favored celebrity who engaged in exposure to overcome stage fright can serve as a role model for efficacy, or playing a board game with a shy youth before beginning exposures can serve to build a therapeutic relationship. Such strategies may not be delineated within a treatment manual but are small appropriate deviations in protocol that can be flexibly integrated with the core treatment protocol while maintaining fidelity to its tenets. A modular framework also provides the opportunity to match the pace and intensity of varying components of treatment to a specific patient's state of functioning or motivation. To illustrate, consider the example of Simpson et al. (2010), who found no improvement in outcomes despite adding MI to treatment for OCD. After close inspection, they identified their sample as

having relatively high scores on readiness for change at pretreatment, indicating that they were already motivated and had a lesser need for a MI intervention. In this case of such patients with stronger motivation for change, a MI module may be less appropriate (as it would be ignoring the issue of match), while the cognitive restructuring and exposure modules would likely still remain pertinent. This matching of modules to a specific patient's level of functioning can extend further. For example, a patient who is apprehensive with regard to engaging in exposures could have the therapist spend more time with him regarding the sources of such ambivalence in MI and psychoeducation modules, while another who understands why she is having panic attacks and wants to change immediately could spend little time on these modules and more quickly get to an exposure module. Many patients may benefit from an even greater acceleration of treatment, as single-session exposure protocols have demonstrated efficacy (e.g., Ollendick et al., 2009).

Another point of emphasis in matching treatments to specific patients involves the consideration of where patients' anxiety fits in the context of their overarching priorities. For example, suboptimal homework completion has been partially attributed to clinicians not matching the assignment of topics to life domains that are pertinent to the patient; thus, one recommendation to improve the effectiveness of homework implementation involves matching homework assignments to short- and long-term patient goals and to issues of current relevance to the patient (Bryant, Simons, & Thase, 1999). Some patients may be better than others in expressing which domains are higher priority for them, so spending extra time to identify such priorities can yield extended benefits. Another consideration in matching treatments to patients' overall functioning is comorbidity, as there often is some ambiguity as to which EST to apply at which time in such situations. Further complicating matters is the fact that patients with anxiety have been observed to primarily focus on symptom relief, while depressed patients may have more heterogeneous treatment goals (Grosse Holtforth, Wyss, Schulte, Trachsel, & Michalak, 2009). Given the high comorbidity rates among these disorders (Kessler et al., 2005), some conflict among multiple goals for treatment and the order of addressing them can be quite common. Methods to address this scenario include establishing if one disorder is primary relative to any others (e.g., the anxiety directly preceded the depression), identifying if treating one disorder might reduce symptomology in other comorbid conditions (Craske et al., 2007), and incorporating patient preferences in which problem to first address. In these ways, clinicians can match the order of treatment delivery to the specific patient in order to maximize outcomes. This situation extends to concurrent life problems, where some patients may be more ready to simply proceed on addressing presenting symptomology, while others might first want to consider other factors. A resolution to such a situation can include identifying if the anxiety is the cause or the effect of the personal interference and subsequently addressing the originating source of distress. For example, some patients may wish to discuss only vocational interference during sessions, despite such interference being largely the product of an anxiety disorder. With these patients, developing insight that the anxiety is a cause of the interference at work and establishing consensus that the anxiety has to be resolved can be useful while remaining attentive to the emotional implications of a difficult work environment (e.g., negative affect due to dealing with a supervisor who is unhappy with suboptimal production resulting from the patient's anxiety). This could also be addressed by directly incorporating the life issue at hand into session plans, which may require adjusting the pace of treatment to accommodate discussion and validation of the patient's extramural problems while still making continued progress with the anxiety treatment.

With regard to matching on preferences for treatment, a therapist is not often in a position to have a treatment aside from CBT to choose from when behaviorally treating anxiety disorders. Although psychodynamic approaches for panic disorder have recently displayed preliminary indications of efficacy (Milrod et al., 2007), CBT remains the contemporary gold standard.

However, within a CBT framework, therapists can still accommodate patient preferences. For example, some patients may prefer massed exposures over a few weeks, while others may prefer traditional weekly treatment. Given evidence where it is available for such massed procedures (e.g., Storch et al., 2007), matching the pace of implementation fosters patient participation with the treatment by validating the patient's preferences. In this way, a clinician can match patient preferences for treatment processes while retaining the core CBT elements.

While the concept of matching the pace of treatment involves the therapist modifying treatment to match patient preferences, another important consideration is how to match therapist and patient expectations for the procedures within treatment sessions. Sometimes match can be accomplished when the therapist orients the patient to treatment expectations. A well-done orientation can bring the patient to match the therapist's views of treatment processes. Current recommendations for CBT for anxiety disorders are to explicitly delineate what the patient will be expected to do in treatment. For example, Abramowitz (1996) found that patients receiving strict instructions in response prevention for OCD had better outcomes than ones receiving no or partial instructions to engage in response prevention—that is, being very direct on the expectations of treatment led to better outcomes. Patients may come in with a variety of expectations for therapy, and unambiguous explanations can help to facilitate participation in the procedures employed in CBT protocols. Moreover, the techniques mentioned in the section on fostering a therapeutic alliance can also serve to improve the match on expectations between patients and therapists during anxiety treatment, as these constructs are intertwined (Greenberg, Constantino, & Bruce, 2006). In many ways, establishing agreement on the tasks to be performed and the goals to be achieved in therapy (essential components of alliance) is a procedure to improve match on patient expectancies for treatment process. Another match issue that arises with CBT for anxiety disorders that can potentially threaten the therapeutic alliance involves therapist directiveness. At first glance, a directive CBT therapist may not match well with reactant patients. However, a flexible therapist, aware of the importance of matching, can identify when to push and when to back off on exposures based on patient reactance. For example, a more directive approach may be appropriate for patients who are looking for change and eager for guidance (i.e., low in reactance), while a supportive and collaborative style is appropriate for those who may show resistance to engaging in exposure (Neziroglu, Forhman, & Khemlani-Patel, 2011). Such reactance may even change throughout treatment; for example, some patients may be initially reactant at first but, after seeing initial gains, become more amenable to a higher level of therapist directiveness. These principles also apply throughout other components of treatment (e.g., psychoeducation, cognitive restructuring; Beutler et al., 2011). In this way, it is not just what a clinician delivers (i.e., CBT for anxiety) but also how he/she delivers it that leads to effective therapist-patient match.

When working with children and adolescents with anxiety disorders, several further considerations arise with regard to behavioral matching. An important consideration is that the style of treatment delivery may need to be modified to match the developmental level of youth patients. For example, it has been found that being "overly formal" with youths receiving child anxiety treatment predicts poorer therapeutic alliance (Creed & Kendall, 2005). To address this concern, several techniques can be used, including reduced formality in presenting the treatment; utilization of youth-oriented props, toys, or dolls; and avoiding delivering the treatment in a formal "sitting in chairs" fashion. This less formal approach is more likely to get the youth patient interested in participation. One method to accomplish this can be done by using the environment proximal to the clinic; for instance, for youth who are afraid of speaking with adults, walking to a nearby cafeteria or convenience store and striking up a brief conversation with the cashier can be an exposure that is more engaging and also more generalizable to the real world. Similarly, matching on social developmental level

can further facilitate treatment engagement, where younger children may frequently work better with a nurturing parental-type figure, while with adolescents, clinicians can be cognizant of avoiding an authoritarian stance. Furthermore, matching on the cognitive level of the youth (e.g., using age appropriate language, using developmentally relevant examples) can further facilitate treatment engagement. The importance of such matching with youth on a variety of developmental aspects has been reflected by an increasing focus on these aspects in newer treatment protocols such as the FRIENDS for Life program (Barrett, Webster, & Turner, 2004). These points are vital considerations given that midtreatment child involvement in treatment (which is often related to alliance) has predicted symptom reduction in CBT for pediatric anxiety disorders (Chu & Kendall, 2004).

Another novel consideration in pediatric psychological treatments for anxiety involves matching treatments and their presentation to parents. While interventions for pediatric anxiety disorders have successfully incorporated family involvement (e.g., Kendall & Hedtke, 2006), few empirically based guidelines for matching to parental characteristics currently exist. This is unfortunate, as parents maintain and improve therapeutic gains at home, and the child patient is wholly dependent on them for treatment attendance. Indeed, indications are that parental engagement in treatment improves youth treatment outcome for anxiety disorders (Podell & Kendall, 2011). To improve communication of the treatment rationale (with the intent of facilitating engagement and active parental involvement in the treatment), it has been recommended to give parents a companion document that describes the treatment in depth (Kendall, Podell, & Gosch, 2010). This can help to further convey the message that parents are co-therapists at home, as well as working to ensure that parents understand the treatment rationale and the cognitive behavioral model of anxiety. In these ways, clinicians can work to find a match with the parent on expectations for treatment, where they are not passive participants who simply bring their child to treatment but rather should expect to be actively involved in enacting the protocol.

Case Example

To illustrate the principles addressed in this chapter, we present a deidentified case example. In this instance, the application of the aforementioned engagement techniques successfully resolved barriers to completing CBT for symptoms of anxiety.

Johanna was a 32-year-old married female with a 5-year-old son who presented to our clinic with a fear of recurrent panic attacks. These attacks had been occurring off and on for 5 years, with an exacerbation in symptoms over the past 9 months. She had been prescribed benzodiazepines by her primary care physician to manage the attacks 5 months before presenting for CBT, which she took at a low dose approximately twice per week to manage panic-related sensations. While the medication provided some immediate relief for her attacks, they did not prevent their frequent recurrence. A thorough intake assessment revealed that she had panic attacks that were characterized by heart palpitations, heavy breathing, dizziness, a perceived loss of control over herself, and a sense of impending doom. She had worked as a cashier at a local grocery store but discontinued her employment due to fear of unexpected panic attacks. Johanna also displayed substantial agoraphobia, as when she presented for treatment, she reported that to avoid having panic attacks she had only left the house five times in the prior 2 months. She reported that this was highly frustrating both for her as well as for her husband and child.

Initially, Johanna had a great deal of ambivalence about therapy. She was hesitant to even leave her house and come to therapy, but she wanted relief from her symptoms and from the strain she was putting on her family. In fact, at first, she was only able to leave the house and travel to therapy accompanied by her family. Thus, even starting during the intake assessment with Johanna, the first task of the clinician was to form an alliance, as the absence of a strong working

relationship could have precluded her from fully engaging in treatment. Given Johanna's initial ambivalence about treatment, the therapist's first efforts were to listen carefully with interest and concern and then validate Johanna's fears and show warmth and empathy regarding her suffering. One way to demonstrate empathy was to use her specific language when discussing her panic, as Johanna had several idiographic labels for her symptoms. For example, she referred to a wave of panic sweeping over her as "the feeling." Thus, the clinician often referred to oncoming sensations of panic as "the feeling" taking hold, in order to match her terminology and work within her conceptualization of the problem.

The clinician realized that he could not even move to explaining the treatment rationale until the patient was ready. The patient would be ready to listen to the clinician only when she felt understood. The clinician's listening and validating skills (making statements about how her fear of leaving the house made sense given her fear of reexperiencing "the feeling") would provide the initial reasons why Johanna would view the therapist as someone who had credibility and was "safe" and trustworthy.

The second focus in alliance formation involved empathetically working collaboratively to find a match on the goals of therapy. For finding a match on goals, the therapist asked the patient about her short- and long-term goals for treatment. She mentioned that her short-term goal was to feel some sense of immediate relief and her longer-term goals included returning to work and spending more time at outside activities with her husband and son. The therapist knew that emphasizing these patient goals throughout treatment and tying these goals to any planned treatment would be critical in keeping the patient focused and willing to work on the challenging tasks of therapy.

The planned treatment consisted of psychoeducation, cognitive restructuring, and exposure. During the initial therapy session, the clinician explained how the proposed treatment could get Johanna to her goals, thus creating positive expectancies for change. Throughout psychoeducation, the therapist carefully oriented the patient to each component of treatment and each task that would be performed with the patient. The therapist also presented the treatment with expected roles for the therapist and the patient. In addition, the therapist carefully explained the rationale for the choice of treatment and for the specific roles involved with the treatment. The therapist carefully made sure that Johanna understood and agreed with the treatment rationale and the involved roles. This would be necessary for patient and therapist to have agreement on the tasks of therapy and for her to view the therapist as credible. In order to ensure a match and commitment to treatment tasks, the therapist aimed to explore the patient's view of the pros and cons of engaging in the proposed treatment. In particular, the therapist helped to identify Johanna's specific barriers relative to engaging in facing her fears. While Johanna was eager for relief, she presented some ambivalence with regard to exposure treatment. On one hand, she was excited to begin treatment, but on the other, she was quite afraid of doing the tasks she had avoided for so long. She frequently verbalized this fear with statements such as, "you're not going to make me leave the house alone, are you?" The clinician responded by validating her view that it is normal to feel fear and reluctance to expose herself to feared situations but also presenting expectations that Johanna could and would succeed in facing her fears. During this discussion, the clinician assured her that she was not the first to experience these symptoms and that many other patients had similar fears which were successfully resolved by the proposed treatment. The therapist also directly addressed Johanna's fears that the exposures might be unsafe. After the clinician explained in detail how the exposures would not put her in actual dangerous situations, Johanna recognized that she avoided feared situations almost as a reflex and she estimated the likelihood of any actual harm as very remote, which helped tip her decisional balance in favor of exposure. In addition, the therapist explained how the treatment was flexible in that an exposure hierarchy could be constructed to match Johanna's short- and long-term goals in sequence, with easier tasks

occurring first. Johanna noted that she liked the structured but flexible guidance relative to the proposed treatment and that this gave her confidence in the sense of direction for the treatment. She also appreciated that there were behavioral explanations for what she was feeling, as she previously had difficulty identifying the nature of her sensations. This validating, normalizing, orienting, flexibility, collaborative matching, discussing the pros and cons of engaging in the proposed treatment, and presenting an expectancy of success seemed to help alleviate her reluctance to participate, and thus Johanna committed to participate in the proposed treatment.

The next two sessions focused on cognitive restructuring and further orientation to exposure tasks. One particular technique of cognitive restructuring that proved useful for Johanna was estimating the actual odds of harm—while at first Johanna mentioned terror about her panic symptoms, when she challenged her thoughts about if actual harm would be incurred, she identified that she was at very little risk of actual harm. After practicing identifying automatic thoughts, she also learned to develop the habit of asking herself "is this true?" when she had thoughts about her anxiety. Johanna found this challenging of her automatic thoughts as providing some comfort, as previously she had simply assumed that her thinking was accurate. These techniques also increased her self-efficacy with regard to engaging in exposure, where she still expressed some ambivalence, but such ambivalence was much reduced compared to our initial assessments. The therapist and Johanna then collaboratively constructed a hierarchy of feared situations. At first, she was only able to produce a few situations, but after exploring a variety of situations, she was able to come up with 15 possible situations for exposure, with a range of tasks that spanned the range of low, medium, and high levels of anxiety. For example, leaving the office alone without carrying her prescribed benzodiazepines for 5 min produced mild anxiety, being alone at home and going shopping alone without benzodiazepines available would cause a moderate level of anxiety, and going to work without benzodiazepines available would produce very strong feelings of anxiety.

In session four, Johanna was asked to practice her first exposures in the office. Despite some trepidation, with therapist encouragement, she engaged in exposures including interoceptive exposures of spinning in a chair until dizzy and breathing through a coffee straw for 1–2 min. During these exposures, she reported initial elevations in anxiety followed by anxiety reduction and perceived mastery of her fear in these limited situations. The therapist was careful to reinforce/praise Johanna's efforts in participating in these initial exposures. After her initial successes, it was decided that her first exposure outside of the office would include shopping alone at a local grocery store without having benzodiazepines available.

At our next session, we evaluated how her first outside of office exposures went. Johanna reported that she experienced much distress because she was only able to remain at the grocery store for 5 min and returned home after feeling a panic attack coming on. In discussing why she left so abruptly, Johanna indicated that she became very scared and reverted to what had helped in the past (avoidance). She expressed doubt that this treatment could actually help for symptoms that were "actually physical." Additionally, she mentioned frustration with the therapist. Although she felt that the exposure in the office was easy for her, she stated that she should have been warned about how difficult this new exposure would be for her.

Instead of being defensive or taking an overly technical perspective of engaging in further psychoeducation regarding the physiological changes that can be produced by exposure (which had been already presented earlier in treatment), the therapist worked on repairing the alliance. To repair the alliance, the therapist validated the patient and took responsibility by acknowledging that he may have pushed too hard and too fast. The therapist expressed empathy that these tasks are difficult to accomplish and that many others have difficulty generalizing exposure work to outside the office. Showing flexibility and a collaborative attitude, the therapist told Johanna

that treatment could be adjusted to a pace that works for her. This calm, flexible, and validating approach improved patient-therapist match and made the therapist come across as more genuine and respectful while maintaining a sense of credibility and competence. By the therapist's calm response that the patient's reaction was not uncommon, the clinician increased patient confidence as Johanna perceived that the therapist had an understanding of the process at hand. Johanna appreciated this response and felt validated in that she was not alone in having difficulty and that she had not "failed" treatment. Additionally, the therapist invited her to feel free to directly address any concerns she ever has in their therapeutic relationship and to inform him if she did not feel ready for future exposures so as to facilitate open conversations and trust. This would also help to accurately match the difficulty of exposures to her level of readiness, as well as providing an opportunity to work through any barriers or further therapeutic ruptures together.

Given Johanna's continued hesitation relative to engaging in further exposure, the therapist utilized motivational interviewing methods. This included not confronting but rather rolling with Johanna's resistance and listening carefully and reflecting her concerns. The therapist then discussed with Johanna the pros and cons of her current methods of dealing with anxiety and the pros and cons of engaging in exposures. The clinician was careful to be accepting and nonjudgmental relative to the patient's discussion of the pros of not changing and the cons of engaging in exposures. However, the clinician highlighted the discrepancies between Johanna's current behavior and the subsequent likelihood of achieving her goals. In addition, the therapist took a gentle paradoxical position (devil's advocate) with Johanna by mildly amplifying some of Johanna's reasons for not changing. This resulted in Johanna taking the opposite position and arguing that she really did want to change. Then, the clinician was able to highlight Johanna's desire for positive change and the connection of change to her treatment goals. The result was that Johanna had renewed motivation and commitment to try exposure again. When Johanna expressed that the plan seemed reasonable but she did not think she could do it, the clinician responded by indicating that many others similar to her had overcome these initial roadblocks and that she was just as capable as these others. This encouragement and support from the therapist helped Johanna to believe that she could succeed with the exposures.

In approaching the exposures that followed, the clinician was very clear in instructing that she must remain in each setting without avoiding the situation in order for exposure to be successful. This focus on clarity facilitated a clinician-patient match on the expectations necessary for the treatment process to achieve symptom relief. In addition, the therapist carefully explored and collaboratively problem solved ahead of time potential obstacles to success or questions Johanna had relative to each exposure. Further, after checking that the patient felt ready to attempt an exposure, the clinician elicited a commitment from Johanna that she would indeed do the exposure assignment. This preparatory work helped Johanna to feel more capable and committed to each exposure that she attempted, thus increasing the likelihood that she would fully engage correctly in her subsequent exposure assignments. After each attempted exposure assignment, the therapist started each session with checking in on the assignment and reinforcing her efforts and successes, in order to strengthen the clinician-patient alliance and to increase the likelihood that she would be motivated to continue to attempt exposures in the future.

Over the next eight sessions, Johanna reported success in completing each step of her exposure hierarchy. In addition, she reported that she had the confidence to expose herself to previously feared situations that were not even listed on her exposure hierarchy. As a result, she reported that her functioning across numerous domains (family, work, etc.) was dramatically improving and that she no longer needed benzodiazepines to be available when she left her house. At her final posttreatment assessment, Johanna had a score of 7 on the Beck Anxiety Inventory (where it was a 51 before treatment). Johanna expressed appreciation for how the therapist had helped with her anxiety as well as with her personal

goals, thus validating his efforts to foster a match between her life priorities as well as her goals for symptom reduction.

Conclusion and Future Directions

CBT has demonstrated great success in treatment for anxiety disorders, but many patients often do not achieve maximal therapeutic benefit. In considering the reasons for suboptimal outcomes, a body of evidence points to limited patient motivation, mismatch of patient and therapist, and poor therapeutic alliance as three likely contributors. While many successful treatment protocols for anxiety disorders have been established, if patients do not participate in their use, they cannot benefit from them. In the past decade, greater focus has been placed in research and its dissemination to clinical practice to motivate, better match, and to foster better alliances with patients with anxiety disorders in order for them to better engage in what are often highly effective treatments for these conditions.

When considering the roles of these variables in treatment, it is important to consider that they do not function in respective isolation but rather work in tandem. Of particular note is a possible role played by therapeutic alliance, where motivation and mismatch affect alliance which in turn affects engagement. A mismatch between patient and therapist can attenuate the alliance, while a strong match and minimization of deleterious elements of mismatch can serve to improve it. With regard to motivation, a bidirectional effect is likely, where a motivated patient may more easily form an alliance with the therapist, but also a therapist who can establish an alliance with a patient who has lesser motivation can work to improve patient motivation and subsequent treatment outcome.

While it may seem natural to attribute treatment failures to unmotivated patients who were resistant or did not want to form an alliance, clinicians have empirical guidance in addressing these domains. As Hollon (2008) has noted, a more self-efficacious attitude is to consider that patients do not fail treatment, but rather treatments must adapt to serve all patients. It is incumbent on researchers and practitioners to identify reasons for treatment failures in anxiety disorders and to work to resolve them, and the aim of this chapter has been to identify a set of factors that are common stumbling blocks. While some researchers have sometimes portrayed "common factors" as being in conflict with CBT for anxiety disorders, we see no reason to believe that they do not compliment CBT well and can help enhance its efficacy. Acknowledging a variety of patient and therapist variables in tandem with fresh approaches to future research can continue to help build upon the marked success of CBT for anxiety disorders, in order to more efficiently and effectively serve a broad array of patients.

Unfortunately, research in treatment processes and idiographic patient care in anxiety disorders remains a relatively sparse body of work (Newman et al., 2006) with many unanswered questions. For example, many contemporary treatment manuals for anxiety disorders recommend forming a therapeutic relationship with the patient, but such manuals often give little guidance as to how to form such a relationship. This is of particular relevance when working with patients who may show more resistance and/or less motivation for treatment participation. At present, much guidance is limited to clinical conjecture, which unfortunately is subject to a variety of pitfalls such as confirmatory bias. More research is needed to identify empirically supported engagement techniques for CBT therapists working with patients with anxiety disorders to help them to engage patients with more complicated presentations. Longitudinal investigations could serve to disentangle issues such as how to match specific alliance-building procedures to specific patient characteristics at specific points in treatment. While ambitious, such hypotheses are certainly not inaccessible, as they could be evaluated through either already existing RCT data or by making small additions to future clinical trials (e.g., using a brief measure of alliance at early, mid-, and late treatment and coding therapy session tapes originally created for protocol adherence evaluation to investigate specific alliance-building behaviors).

In looking to future research in patient engagement for anxiety disorders, innovative approaches to research might prove fruitful. For example, Kiesler (2004) has suggested the notion of a "process diagnosis," where various facilitative and counterproductive treatment processes are identified and then specific interventions are developed to address them. Some of these various treatment processes might be somewhat unique to anxiety, and we currently have limited knowledge of such specific treatment process patterns and factors that predispose them. These sentiments are echoed by DeRubeis, Brotman, and Gibbons (2005), who note that the patient-therapist dyad may be a better unit of analysis to evaluate therapeutic alliance compared to either party in isolation. They suggest that instead of assessing solely patient characteristics to address the likelihood of benefit from therapy, to rather evaluate which dyads and associated processes might prove fruitful.

In evaluating these new approaches, methodological and analytic procedures may have to be adjusted as well. For example, traditional between-subjects pre-post designs may be somewhat insensitive to matching effects due to limited power and the difficulties involved in recruiting sufficient numbers of patients and therapists in order to detect matching effects. Methods such as the actor-partner interdependence model (Kenny, Kashy, & Cook, 2006) can more precisely partition variance and maximize the amount of information extracted from paired data. Additionally, frameworks such as random effects modeling (e.g., Singer & Willett, 2003), latent class analysis, and cluster analysis can serve to identify various subgroups of treatment responders and nonresponders, which can help identify specific characteristics of patients, therapists, and dyads who may perform better with one intervention or another. Finally, the actual nature of the data generated merits addressing. Much intervention research focuses on measures of symptom severity and demographic factors and self-reports of treatment processes, but less focus has been placed on what behaviors actually occur during treatment. Methods of approaching this issue include recording sessions and using structured methods of coding in-session behavior (e.g., McLeod & Weisz, 2005) in a quantitative fashion, as well as employing structured qualitative research (e.g., Bernard & Ryan, 2010) with session transcripts and narratives from both patient and therapist, which can provide fresh approaches to old problems while maintaining methodological rigor. While much progress has been made with traditional research methods and many insights have served to improve patient engagement, further progress in understanding and improving treatment of anxiety-disordered patients with complicated presentations may require alternative means of approaching such problems. We have a burgeoning base of research to guide current clinical practice, as well as a roadmap for further progress in order to improve therapy outcomes for anxious patients.

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Substance Abuse and Anxiety Disorders: The Case of Social Anxiety Disorder and PTSD

18

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According to the National Epidemiological Survey on Alcohol and Related Conditions (NESARC), the prevalence rate of substance use disorders (SUDs) is at least 50% higher for those with an independent anxiety disorder diagnosis than individuals without an anxiety disorder (Grant et al., 2004). Further, the odds of having an independent anxiety disorder are doubled for those with an SUD compared to those without an SUD (Grant et al.). Comorbidity rates nearly quadruple when considering only the more severe form of SUDs, substance dependence. Consequently, it is critical to identify efficacious interventions to treat these common and complex conditions. This chapter aims to provide clinicians, researchers, and students with a background in anxiety disorder/SUD comorbidity and its treatment.

The present chapter focuses on two anxiety disorders: social anxiety disorder (SAD) and posttraumatic stress disorder (PTSD). The chapter begins by providing a description of SUDs, SAD, and PTSD and an overview of the models explaining the comorbidity between these disorders. Next, we highlight factors that contribute to the complexity of the SAD/SUD and PTSD/SUD comorbidity. The bulk of the chapter describes interventions that have been developed to address these complex comorbid conditions, including a review of data examining treatment efficacy and a case study. Finally, we present conclusions and future directions.

Substance Use Disorders

The *Diagnostic and Statistical Manual—fourth edition, text revision* (*DSM-IV-TR*; American Psychiatric Association [APA], 2000) includes two broad types of SUDs: substance abuse and dependence. Substance abuse is a pattern of substance use that interferes with the person's life, manifested by one or more adverse consequences related to the substance use (e.g., failing to fulfill major obligations, social or legal problems) occurring within a 12-month period. In contrast, the essential feature of *DSM-IV-TR* substance dependence is a cluster of physiological (e.g., tolerance, withdrawal) and psychological (e.g., loss of control over use, significant effort and time spent seeking, using, and recovering from the substance) symptoms occurring within a 12-month period. The concept of "addiction" aligns most closely with substance dependence.

Lifetime prevalence rates for SUDs are estimated to be approximately 14% (Kessler, Chiu, Demler, Jin, & Walters, 2005). In 2009, an estimated 8.9% of the United States population aged

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12 years or older was classified with a past-year SUD (SAMHSA, 2010). Of these individuals, 14% were classified with dependence on or abuse of both alcohol and illicit drugs, 17% illicit drugs only, and 69% alcohol exclusively (SAMHSA). Specific illicit drugs associated with the highest past-year dependence or abuse rates were marijuana (19%), pain relievers (8%), and cocaine (5%) (SAMHSA). The annual cost of SUDs in the United States was estimated at $180.9 billion in 2002 and has been increasing annually since 1992 (ONDCP, 2004). While research on natural recovery from SUDs (primarily focused on alcohol) has shown that a significant number of substance users recover without treatment (see Smart, 2007 for a review), the majority do not. Further, relapses are more common among those who recover without treatment than those who recover with treatment (Moos & Moos, 2006). Unfortunately, only approximately 11% of individuals with an SUD received needed treatment in the past year (SAMHSA, 2010).

Social Anxiety Disorder and Substance Use Disorders

Social anxiety disorder, also known as social phobia, is a common and debilitating condition involving a marked and persistent fear of one or more social (e.g., conversations, dates) and/or performance (e.g., public speaking, writing in front of others) situations in which the person faces possible scrutiny and/or embarrassment (APA, 2000). The feared social situations are avoided or endured with intense anxiety. SAD symptoms must result in significant functional impairment and/or marked distress, which often include lower educational attainment, as well as higher rates of work and social impairment, comorbidity, and suicidal ideation (Grant et al., 2005; Ruscio et al., 2008). SAD typically begins by adolescence, has a chronic course, and does not remit without treatment (Grant et al., 2005).

Epidemiological reports have estimated that SAD has a lifetime prevalence of 12.1% (Kessler et al., 2005). Among individuals with a lifetime SAD diagnosis, 48.2% had an alcohol use disorder and 22.3% had a drug use disorder (excluding nicotine; Grant et al., 2005).

Individuals with co-occurring SAD/SUD have more severe cases, including higher psychiatric comorbidity rates, than individuals with SAD alone (Grant et al., 2005; Thomas, Thevos, & Randall, 1999). Further, people with comorbid SAD/SUD appear more likely to relapse after alcoholism treatment compared to people with an SUD alone (Kushner et al., 2006). Overall, findings suggest that individuals with comorbid SAD/SUD have more severe and complicated presentations and suffer from poorer prognoses than individuals with SAD or SUD alone.

Posttraumatic Stress Disorder and Substance Use Disorders

PTSD is another chronic and debilitating condition (Kessler, 2000) which can develop following a variety of traumatic events, including natural disasters, combat, and interpersonal violence (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). The vast majority of people exposed to a traumatic event develop acute symptomatic reactions, and while the majority of people recover without intervention, a substantial minority will continue to experience posttraumatic stress symptoms (Gilboa-Schechtman & Foa, 2001). PTSD is diagnosed when an individual experiences an event characterized by perceived threat that elicits overwhelming fear, helplessness, or horror, followed by persistent (lasting for at least 1 month) posttraumatic stress symptoms that result in functional impairment. Four types of symptoms characterize PTSD: (1) reexperiencing aspects of the traumatic event (e.g., intrusive thoughts, recurrent dreams, flashbacks); (2) avoidance of traumatic event cues (i.e., avoiding thoughts and reminders of the event); (3) emotional numbing (e.g., detachment, loss of interest, restricted affect); and (4) hyperarousal (e.g., sleep disturbance, irritability, difficulty concentrating; Elhai et al., 2009; King, Leskin, King, & Weathers, 1998).

Among nationally representative samples, Kessler et al. (2005) found a lifetime estimate of

7.8% for PTSD. When examining Veteran populations, PTSD lifetime estimates increase to 18.7% (Dohrenwend et al., 2006). A number of cross-sectional studies have documented elevated odds of substance use among individuals with a diagnosis of PTSD compared to individuals without PTSD (Blumenthal et al., 2008; Chilcoat & Breslau, 1998; Kessler et al., 1995).

Comorbid PTSD/SUD is associated with increased impairment compared to PTSD or SUD alone. Individuals with co-occurring PTSD/SUD experience greater emotional difficulties and receive less social support than individuals with only SUD or SUD and another Axis I disorder (Ouimette, Ahrens, Moos, & Finney, 1997). Compared to individuals who do not use drugs/alcohol to manage emotions, individuals who report active substance use to medicate their PTSD symptoms have reduced health-related quality of life (Leeies, Pagura, Sareen, & Bolton, 2010). Further, PTSD and SUD appear to be independent predictors of suicidal ideation and attempts (Ullman & Brecklin, 2002) and comorbid PTSD/SUD report a higher number of lifetime suicide attempts than either PTSD or SUD alone (Moylan, Jones, Haug, Kissin, & Svikis, 2001). Additionally, studies support the notion that individuals with co-occurring PTSD/SUD fare worse in treatment compared to people with either disorder alone (Brown, Read, & Kahler, 2003; Brown, Stout, & Mueller, 1996; Ouimette et al., 1997).

Models of Anxiety Disorder/Substance Use Disorder Comorbidity

Three models are presented that propose: (1) anxiety disorders promote SUD (i.e., self-medication hypothesis), (2) SUD promotes anxiety disorders (i.e., high-risk model), or (3) a common variable promotes both SUD and anxiety disorders (i.e., common etiology). While the models presented here are condensed to provide a general overview, a more in-depth analysis is available in Kushner, Abrams, and Borchardt (2000) and Stewart and Conrod (2008).

Self-Medication

According to the self-medication hypothesis (Khantzian, 1985), similar to tension reduction (Conger, 1956) and stress-response dampening (Sher & Levenson, 1982) models of alcohol use, substance use functions as a means to reduce distress and manage unpleasant psychological states. Self-medication and related models assume a causal model where the presence of a disorder leads to the development of the other. When applying the self-medication hypothesis to anxiety disorder/SUD comorbidity, one would predict that individuals suffering from a primary anxiety disorder, such as SAD or PTSD, develop an SUD after repeated attempts to relieve or reduce their anxiety through substance use (Quitkin, Rifkin, Kaplan, & Klein, 1972).

High-Risk

The high-risk model, commonly referred to as the substance-induced anxiety hypothesis, states that pathological substance use serves to promote the development of an anxiety disorder (see Kushner et al., 2000 for a review). One explanatory mechanism for such a pathway is that substance dependence and withdrawal symptoms, particularly when considering alcohol, can cause neurobiological changes (e.g., reduced levels of γ-aminobutyric acid benzodiazepine receptors in the case of alcohol and depressants) that result in acute or chronic anxiety symptoms (e.g., George, Nutt, Dwyer, & Linnoila, 1990).

Common Etiology

In the common variable theory, it is assumed that a third variable may account for an individual's increased risk for developing both disorders and that no causal relationship exists between the disorders (see Kushner et al., 2000; Stewart & Conrod, 2008 for reviews). Possible candidates for such common variables include personality predispositions or a common genetic basis underlying the two disorders.

Data Supporting Models

Supporting the self-medication hypothesis, National Comorbidity Survey follow-up data confirmed that baseline panic, specific phobia, separation anxiety, PTSD, and SAD were each predictive of at least one form of substance dependence 10 years later (Swendsen et al., 2010). Several longitudinal studies based on epidemiological samples have found that baseline SAD symptoms (e.g., Buckner et al., 2008; Swendsen et al., 2010; Zimmerman, Wittchen, Pfister, Kessler, & Lieb, 2003) or PTSD symptoms (e.g., Cottler, Compton, Mager, Spitznagel, & Janca, 1992; Leeies et al., 2010; Swendsen et al., 2010) in particular predict later SUD diagnoses. For example, the presence of PTSD more than tripled the odds of developing alcohol dependence (OR = 3.2) and illicit drug dependence (OR = 3.9) across a 10-year follow-up period (Swendsen et al.). In people with SAD, epidemiological estimates suggest that self-medication with substance use is present between 7.9% (public speaking subtype) and 21.2% (Bolton, Cox, Clara, & Sareen, 2006; Robinson, Sareen, Cox, & Bolton, 2009). Individuals who report frequently using alcohol to cope with social anxiety also drink more and have greater alcohol dependency symptoms than those who do not use alcohol to cope with social anxiety (Carrigan, Ham, Thomas, & Randall, 2008). Further, experimental data have found that socially anxious and non-anxious individuals demonstrate greater levels of alcohol use (see Battista, Stewart, & Ham, 2010 for a review) following a social stressor, suggesting that social stressors confer a higher risk for alcohol use. When considering PTSD, Leeies et al. (2010) have shown that approximately 20% of individuals with PTSD use substances (alcohol, drugs, or both) in an attempt to self-medicate. Overall, these data suggest that individuals diagnosed with SAD or PTSD commonly self-medicate and are more likely to develop an SUD.

Data also support a high-risk (substance-induced anxiety) model. Studies indicate that the onset of SUD symptoms often precedes anxiety disorder onset (Breslau, Novak, & Kessler, 2004; Semple, McIntosh, & Lawrie, 2005). The disparate findings in directionality could be explained by considering the age of onset and temporal ordering for specific anxiety disorders and SUDs. For instance, Falk, Yi, and Hilton (2008) found that while the onset of SAD and specific phobia tended to precede the onset of alcohol dependence or alcohol abuse, the age of onset for panic disorder and generalized anxiety disorder was much later than the onset for both types of alcohol use disorders. Therefore, self-medication and high-risk models may play more or less prominent roles depending upon anxiety disorder type.

Support for the common etiological model has been found in research examining a shared genetic basis or personality predispositions in comorbid anxiety and SUDs. For example, Merikangas, Stevens, and Fenton (1996) found that relatives of individuals, who were alcohol-dependent and/or had an anxiety disorder, were at an increased risk (ORs = 2.0–3.7) for developing alcohol dependence (but not alcohol abuse) and/or an anxiety disorder themselves. It has been suggested that anxiety sensitivity, a personality predisposition characterized by fear of the consequences of anxiety symptoms (Taylor, 1999), might be important in the development of both anxiety disorders and SUDs (Stewart & Kushner, 2001; Stewart & Conrod, 2008). Despite support for the common etiology model, additional research is needed to better understand the impact of personality predispositions and genetics on the comorbidity of anxiety and SUDs.

Importantly, the processes involved in *perpetuating* a comorbid anxiety disorder/SUD condition may not be those implicated in the *onset* of the comorbid condition. Instead, both anxiolytic (i.e., self-medication) and anxiogenic (i.e., high-risk) processes may be at play in *maintaining* comorbid anxiety and SUDs, as proposed by Kushner et al. (2000; "feed-forward" model) and Stewart and Conrod (2008; "mutual maintenance" model). While processes consistent with either the self-medication or high-risk models might be key in the development of the co-occurring condition (e.g., self-medication in SAD and PTSD), both model processes eventually interact in a reciprocal nature and maintain both substance use and anxiety symptoms. Thus, it is important

to recognize that both processes may be occurring in these comorbid conditions, contributing to the complexities in treating a person with an anxiety disorder and SUD.

Factors That Contribute to Complexity

An individual with a comorbid SAD/SUD or PTSD/SUD presents a particularly complicated picture when considering assessment and treatment. As described previously, people with the comorbid condition present with more severe cases (e.g., Grant et al., 2005; Ouimette et al., 1997) and have poorer prognoses (e.g., Brown et al., 2003; Kushner et al., 2006) than people diagnosed with one disorder. This section highlights several aspects contributing to the complexities in assessment and treatment of individuals with comorbid SAD/SUD or PTSD/SUD.

Assessment

The presence of an anxiety disorder might be overlooked in individuals completing treatment in a substance treatment facility and vice versa. For example, El-Sayegh, Fattal, and Muzina (2006) found that SAD went unrecognized in a psychiatric evaluation for 94% of addiction treatment-seeking patients with substance dependence. One way to address this problem in SUD contexts is to provide training and education in anxiety disorders as well as to encourage assessment of anxiety symptoms and motives for drinking (e.g., self-medication-related motives) in any client presenting with an SUD. Similarly, clinicians in anxiety disorder treatment contexts should be aware of the possibility of a co-occurring SUD and routinely assess substance use, including substance use as a safety behavior. While a lack of awareness among clinicians about the need to assess for the co-occurring disorders is one factor contributing to problems in identifying individuals with the comorbid condition, there are many additional factors that complicate assessment in cases involving an anxiety disorder and an SUD. Challenges related to differential diagnosis, cognitive deficits, and secondary gain are reviewed below.

Differential diagnosis. First, symptoms of SUDs (particularly withdrawal) and substance intoxication can overlap with symptoms of anxiety disorders. For instance, the effects of substance intoxication and withdrawal from a substance may mimic anxiety symptoms or include unpleasant symptoms that influence anxiety levels. Intoxication, withdrawal, and adaptation to abstinence may have temporary or permanent effects on psychological, cognitive, and psychomotor functioning (Clark, 1999). Indeed, it may be difficult to ascertain whether or not anxiety symptoms are in fact temporary (e.g., substance-induced anxiety disorder) or independent anxiety syndromes. For example, effects of intoxication (e.g., stimulants) and withdrawal (e.g., alcohol, depressants) from several substances result in hyperarousal and increased anxiety. These effects could appear similar to hyperarousal symptoms present in PTSD. Further, individuals experiencing withdrawal might appear to meet some criteria for SAD due to concerns about appearing anxious and/or being negatively evaluated when experiencing observable withdrawal symptoms (e.g., tremors, shaking). Given these difficulties in distinguishing substance-induced anxiety from an independent anxiety disorder, a minimum of 4 weeks of abstinence is recommended prior to giving an individual a diagnosis of an anxiety disorder in addition to an alcohol use disorder (APA, 2000; Clark, 1999). The abstinence period for other substances has not been established empirically, but there is agreement that a drug-free period is necessary to determine whether anxiety symptoms represent intoxication, withdrawal symptoms, a substance-induced anxiety disorder, or an independent anxiety disorder.

Determining length of sobriety may be accomplished using self-report, collateral report(s), behavioral observation, and/or a biological drug test. Self-report information from substance users is generally reliable and accurate when collected in a confidential setting while the individual is sober (e.g., Sobell & Sobell, 1990). Thus, ensuring

sobriety at the time of assessment is critical. A biological test, such as urine drug screening, blood tests, or alcohol breath tests (e.g., Allen & Litten, 2001; Goldberger & Jenkins, 1999) is preferred, particularly if there are concerns that the individual is intoxicated at the time of the assessment and/or is dishonest about his or her last use. However, there are potential drawbacks to this method (e.g., cost, varying drug metabolite half-lives). Generally, the most cost effective and least invasive methods for assessing sobriety is through the use of self-report, collateral reports (e.g., a significant other, family member), and behavioral observations of signs of use (e.g., unsteady gait, pupil dilation) or withdrawal (e.g., tremors, sweating).

For many reasons it may be difficult to attain the goal of sobriety prior to or during the assessment process. Achieving this goal can be particularly challenging when the anxiety disorder and SUD interact reciprocally (i.e., the "feed-forward" or "mutual maintenance" models; Kushner et al., 2000; Stewart & Conrod, 2008) making it difficult for the client to abstain from the substance without another method to cope with fear-related substance using cues and anxiogenic withdrawal effects. Often, assessment occurs before the client has achieved the recommended period of sobriety. When working with an individual with an SUD who is currently using or has recently initiated a period of abstinence, it is important to consider the potential intoxication and withdrawal effects of the abused substance(s) when evaluating anxiety symptoms. Furthermore, the clinician should establish a chronology in the development and maintenance of symptoms, which is best accomplished through the combination of client and collateral reports (e.g., Lingford-Hughes, Potokar, & Nutt, 2002). The use of multimodal assessments (e.g., previous medical records, substance and psychological history, self-report assessments, self-monitoring, collateral reports, behavioral observations, and biological substance use tests), multiple assessment points, and medical examinations to assess for withdrawal effects are strategies that can be implemented to improve diagnostic accuracy.

Cognitive abilities. Cognitive deficits may interfere with assessment and treatment participation. Individuals with an SUD in particular are likely to experience cognitive deficits given the neurotoxic effects of the substance, traumatic brain injury occurring while intoxicated, or severe malnutrition associated with SUDs (Bates, Bowden, & Barry, 2002; Tarter & Kirisci, 1999). Though some neurocognitive deficits related to chronic alcohol use may improve over time (Goldman, 1986), permanent deficits have been observed even in heavy social drinkers (e.g., Oscar-Berman, Shagrin, Evert, & Epstein, 1997). Therefore, a clinician assessing a substance-using client must consider the possibility of short-term or long-term cognitive deficits. In the case of PTSD, for example, substance use-related cognitive deficits could be overlooked if memory problems are attributed to avoidance of trauma-related memories. As such, substance users may require a neuropsychological evaluation, with consideration of time since last use, length and pattern of use, medical history, as well as history of anxiety and SUD symptoms.

Secondary gain. There are several possible secondarily reinforcing factors, also referred to as secondary gain, for clients undergoing an assessment for co-occurring anxiety disorder and SUD. For example, assessment results could have implications for compensation related to level of functional impairment and distress (e.g., disability claims, worker's compensation), in which the individual may be motivated to overreport symptoms. In some situations, the likelihood of receiving compensation and/or insurance benefits for treatment of psychological diagnoses might be increased if the individual has an anxiety disorder (or other non-SUD diagnosis) compared to an SUD. In addition, the assessment might be motivated by a desire to avoid or reduce the severity of legal consequences. Conversely, some individuals may be motivated to avoid receipt of diagnoses. In addition to concerns about the general social stigma of the label(s), individuals may be concerned about whether the diagnoses affects eligibility for certain types of employment (e.g., military and other governmental service), future insurance coverage, or access to a treatment

program that excludes individuals with co-occurring anxiety or SUDs. Given these potential motivators for over- or underreporting symptoms, it is important that the clinician asks the client (and collateral sources or agencies as applicable) about his or her expectations and beliefs about the assessment process as well as the desired outcomes.

Treatment Barriers

Only about 24% of NESARC respondents with comorbid SAD/alcohol use disorder sought treatment for SAD and 26% for an alcohol use disorder (Schneier et al., 2010). This leaves a large proportion of individuals with the comorbid condition without treatment for SAD, an alcohol use disorder, or both. Recent reports suggest that only 13.3% of individuals with PTSD had ever received treatment (Davis, Ressler, Schwartz, Stephens, & Bradley, 2008). Further, previous findings suggest that PTSD is underdiagnosed in SUD samples (Dansky, Roitzsch, Brady, & Saladin, 1997) resulting in numerous individuals not receiving adequate treatment. Reasons for the lack of treatment can be classified as client-related, systemic, and clinician-related factors.

Client-related factors. Avoidance behaviors present in anxiety disorders generally, and for SAD and PTSD in particular, may impact treatment-seeking and participation in both anxiety and SUD treatment. Fear avoidance could result in avoidance of specific therapy components or the therapy process more generally. The prospect of engaging in feared situations as part of exposure-based interventions may lead many clients to avoid anxiety-related treatments, particularly when the individual is also attempting to abstain from substances that have been used to cope with such fears. In the case of a client with SAD, the prospect of placing a telephone call to make an appointment, social interactions in the waiting room, and social interactions in treatment settings might deter treatment-seeking. Similarly, PTSD-related hypervigilance and difficulty with trust (which may make the prospect of engaging in therapy and potentially feeling vulnerable in a therapeutic setting threatening) may promote avoidance of therapy.

Treating a client with comorbid anxiety disorder/SUD may be impacted by his or her willingness to participate in common aspects of addiction treatment. For example, individuals with SAD may avoid SUD treatment or attend but not actively participate in treatment, which often involves group settings. Indeed, socially anxious substance abusers report that social anxiety symptoms interfere with willingness to talk to a therapist, speak in a group setting, attend 12-step programs, and ask someone to be a 12-step sponsor (Book, Thomas, Dempsey, Randall, & Randall, 2009).

Withdrawal from substance use often leads to increased anxiety and arousal. Thus, another consideration in the treatment of individuals with comorbid anxiety/SUD is problems in discontinuing substance use or refusal to stop using anxiolytic medications during exposure-based treatments for anxiety disorders. Research suggests that alcohol and other depressants (e.g., benzodiazepines) interfere with certain learning processes, which is particularly critical in exposure-based therapies (Morissette, Spiegel, & Barlow, 2008). Furthermore, depressant use among individuals receiving exposure-based treatment for PTSD has been associated with higher dropout rates (van Minnen, Arntz, & Keijsers, 2002). A related problem involves intoxication in therapy. Clinicians should discuss rules about intoxication in the clinical setting before therapy begins so that the client is aware of the consequences of arriving to therapy intoxicated.

Systemic factors. One critical barrier to treatment in individuals with comorbid anxiety disorder and SUD is the lack of availability or access to efficacious treatments focused on the comorbid conditions. This often leaves clients with both an anxiety disorder and an SUD to seek treatment that focuses on just one disorder. Such treatments typically do not include consideration of the co-occurring disorder and the functional relations between the two disorders. Many treatment programs/providers require that potential clients obtain treatment for the SUD prior to anxiety

disorder treatment. This is unfortunate because some individuals may not desire SUD treatment if substance use is viewed as a coping mechanism for anxiety, rather than an independent problem. Secondly, SUD treatment may not be as effective when disorder-specific triggers for substance use remain (Ouimette, Moos, & Finney, 2003). Third, disorder-specific fears might interfere with SUD treatment participation (as discussed above). Finally, there is evidence that psychosocial or pharmacological treatment targeting SAD only in individuals with SAD/SUD may experience a clinically significant reduction in SAD with limited changes in substance use (Book, Thomas, Randall, & Randall, 2008; McEvoy & Shand, 2008). Thus, the standard practice of requiring SUD treatment prior to anxiety disorder treatment may be problematic in certain cases.

In the context of treatment for an anxiety disorder and an SUD separately, it is also possible that one type of treatment could interfere with another form of treatment. For example, benzodiazepines, often prescribed for anxiety symptoms, can interfere with the learning process within exposure-based therapies for anxiety and increase the likelihood of attrition (Morissette et al., 2008; van Minnen et al., 2002). Benzodiazepines also have a high potential for abuse and could be dangerous if mixed with other substances (Back & Brady, 2008).

Clinician factors. Clinicians may be hesitant to implement exposure-based therapies in an individual with an SUD due to concerns of relapse or attrition from therapy as a result of introducing a potential substance use trigger (i.e., a feared situation). Indeed, some clinicians may be reluctant to use exposure therapy in general (see Richard & Gloster, 2007 for a review), and in individuals with PTSD in particular (see Ruzek & Rosen, 2009 for a review), due to concerns that the anxiety-inducing techniques central to exposure may cause excessive distress for the client. Clinicians treating an anxiety disorder may avoid treating an SUD due to concerns about client attrition because the client might not share the goal of treating the SUD. Further, clinicians may not have sufficient training or feel competent in providing treatment for the co-occurring disorder, leading them to ignore the co-occurring disorder or refer the client elsewhere.

In sum, several factors complicate the picture for clinicians when encountering an individual with comorbid SAD/SUD or PTSD/SUD. Challenges related to differential diagnosis, cognitive abilities, and secondary gain may make identification of individuals with these comorbid conditions difficult. Furthermore, client-specific, systemic, and clinician-related barriers contribute to the difficulties in treating clients with a co-occurring anxiety disorder and SUD.

Treatment Approaches

After presenting treatment elements common to SUD treatments more generally, this section will cover specific treatment approaches for SAD and SAD/SUD as well as treatment approaches for PTSD and PTSD/SUD. A case study is presented for each set of comorbid conditions to illustrate treatment implementation for these co-occurring disorders.

Treatment for SUDs often involves group settings in which social support is an important aspect of the therapeutic environment. Clinicians often encourage clients with an SUD to participate in 12-step groups such as Alcoholics Anonymous (AA) or Narcotics Anonymous (NA), which may be accomplished using a manualized treatment (Twelve-Step Facilitation [TSF]; Nowinski, Baker, & Carroll, 1992). Motivational enhancement therapy (MET), cognitive-behavioral therapy (CBT), and combined behavioral intervention (CBI) are currently the leading psychosocial interventions involved in the treatment of alcohol use disorders (Randall, Book, Carrigan, & Thomas, 2008). MET refers to an approach in which the therapist employs motivational strategies, such as active listening techniques, to encourage the client to develop his or her own plan for changing drinking behaviors (Miller, Zweben, DiClemente, & Rychtarik, 1995). CBT is founded on the integration of both behavioral and cognitive interventions, which are each based on the assumption that prior learning is having maladaptive consequences.

Accordingly, CBT aims to reduce distress or unwanted behavior by undoing learning or by proving new, more adaptive learning experiences (Kadden et al., 1995). CBI incorporates techniques from both MET and CBT in addition to encouraging clients to participate in support groups and including family members in the treatment plan (Longabaugh, Zweben, LoCastro, & Miller, 2005). Although this chapter does not cover pharmacological treatment, it should be noted that pharmacotherapy continues to be an important part of SUD treatments (Vocci, Acri, & Elkashef, 2005).

Social Anxiety Disorder and Substance Use Disorder Comorbidity

SAD treatment aims to reduce social fears and avoidance of feared situations. Several forms of cognitive-behavioral approaches (i.e., cognitive therapy, exposure therapy, CBT, social skills training, and relaxation training) appear effective in the treatment of SAD (Book & Randall, 2002; Clark et al., 2006; Randall et al., 2008; Rapee, Gaston, & Abbott, 2009); however, exposure therapy has been argued to be the critical component in reducing SAD symptoms (Clark et al., 2006). Exposure therapy is an approach in which the client is encouraged to expose themselves to the feared situation while using skills learned in therapy to reduce anxiety symptoms. Cognitive therapy is largely focused on challenging erroneous beliefs (e.g., cognitive restructuring) after the client identifies his or her own problematic thoughts, behaviors, or emotions. Relaxation training refers to a technique in which the client is taught to control the amount of physical tension in his or her body in order to reduce anxiety. SAD may be associated with deficits in social performance; therefore, social skills training targets behavioral skills for social interactions. With the integration of each of these approaches, CBT has consistently shown significant and long-lasting treatment effects for anxiety disorders (see Stewart & Chambless, 2009 for a review).

Many well-controlled clinical trials have highlighted the efficacy of both psychosocial and pharmacologic (primarily selective-serotonin reuptake inhibitors [SSRIs]) interventions for the treatment of SAD (Hofmann & Smits, 2008; Randall et al., 2008; Stewart & Chambless, 2009); however, these studies have largely excluded participants with an SUD. Consequently, the literature provides few evidence-based treatment options for individuals presenting with co-occurring SAD/SUD. Despite this gap in the literature, previous studies have highlighted important characteristics of treatment that increase the likelihood of a positive outcome when treating an individual with co-occurring SAD/SUD. Research on the treatment of SUDs and its co-occurrence with SAD has largely focused on alcohol use disorders specifically; however, treatment options for SUDs more generally will be discussed when data are available.

There are currently three general approaches to the treatment of co-occurring SAD/SUD, namely, sequential, concurrent, and integrated methods. Traditionally, clinicians have utilized a sequential treatment approach for treating individuals with comorbid SAD/SUD, treating the substance use problem first and then treating the SAD. Concurrent treatment is an approach in which the client receives treatment for both disorders simultaneously; however, there is no consideration given to the relations between the two disorders. With strong evidence-based treatment options for each disorder individually, Kushner et al. (2000) argued that the lack of success when treating clients with comorbid SAD/SUD may suggest that together these disorders form a "hybrid condition" that requires an entirely different treatment option than that seen for either condition alone. As such, it is possible that a "hybrid" or integrated treatment model would be a more efficacious approach. In an integrated model, both SAD and SUD are treated and monitored simultaneously by a single individual qualified to treat both disorders. This model attempts to demonstrate to clients how problems are interrelated rather than approaching them separately, as done in sequential or concurrent models.

Efficacy. One clinical case study examined a sequential approach to treating SAD/SUD.

Buckner et al. (2008) examined the utility of combining MET with CBT in treating a client with comorbid SAD and an alcohol use disorder. The authors found that the use of a brief MET intervention prior to individual CBT for SAD provided a useful skill (e.g., alcohol-related change plan) that decreased the risk of using alcohol to cope with the increased anxiety levels experienced in CBT treatment. Following 19 sessions of MET and CBT, and at 6-month follow-up, the client no longer met criteria for SAD or the alcohol use disorder.

Two studies examined a concurrent treatment approach to treating SAD and alcohol dependence. In one randomized controlled trial, researchers utilized a concurrent individual treatment model in the treatment of alcoholics with SAD (Randall, Thomas, & Thevos, 2001). Participants were randomly assigned to alcohol treatment only ($n=44$) or combined alcohol and SAD treatment ($n=49$). Both 12-week treatments were individual, manual-guided, and CBT oriented. Interestingly, the group receiving CBT for alcohol dependence yielded slightly better alcohol-related and equivalent SAD treatment outcomes when compared to the group that received concurrent SAD/alcohol dependence CBT treatment. A high dropout rate in the concurrent treatment group is a possible explanation for findings that were inconsistent with hypotheses (i.e., the dual-treatment approach would lead to less SAD and drinking, compared to the alcohol only treatment). It is possible that exposure to feared situations without the aid of alcohol or alternative coping strategies could have led to greater anxiety levels and thus lower treatment adherence (Randall et al., 2008). Methodological care concerning attrition and third variable effects may be beneficial in future clinical trials examining treatment for SAD/alcohol use disorder.

Schade et al. (2005) demonstrated similar results while examining a concurrent treatment for individuals with comorbid alcohol dependence and an anxiety disorder (SAD or agoraphobia). In this randomized controlled trial, participants were assigned to either a combined intensive psychosocial alcohol relapse prevention program with anxiety-focused CBT and optional SSRI pharmacotherapy ($n=47$) or a psychosocial relapse prevention treatment only ($n=49$). Results indicated that the concurrent treatment was effective in reducing anxiety symptoms; however, it had no significant effect on alcohol relapse rates. Concurrent treatment approaches for comorbid SAD/SUD may reduce anxiety symptoms, but not alcohol use.

In a preliminary study, Courbasson and Nishikawa (2010) found that a modified SAD cognitive-behavioral group therapy (CBGT) resulted in decreased social anxiety-related symptoms from pre- to posttreatment among 26 clients with co-occurring SAD and SUDs. Though the protocol was primarily based on Heimberg and Becker's (2002) CBGT for SAD, it also included explicit discussions of the link between SAD and substance use, making it an integrated treatment to a degree. The effect size of the social anxiety reduction ($d=0.85$) was similar to that of studies consisting of clients with SAD only. Unfortunately, substance use was not assessed, so treatment outcomes related to substance use are unknown. Other important limitations of the study include the lack of a control group and the high (56%) attrition rate. Nonetheless, the results provide evidence for additional research to explore the specificity of these effects in this population. Importantly, this is the only known study to examine treatment of SAD/SUD for substances other than alcohol (i.e., cocaine, cannabis, opiates, or prescription drugs).

Finally, one study has provided support for an integrated treatment approach. In a recent National Institute on Alcohol Abuse and Alcoholism (NIAAA)-funded study, researchers examined a new integrated treatment, the Brief Intervention for Socially Anxious Drinkers (BISAD), which combined CBT strategies for social anxiety and hazardous drinking (PI: Tran; Grant No. R21AA014014). A pilot study provided evidence for the efficacy of BISAD ($n=21$) in reducing heaving drinking, social anxiety, and alcohol-related negative consequences at 1- and 4-month follow-ups, compared to alcohol psychoeducation ($n=20$) (Tran, 2008). The success of this integrated approach may be attributed to the focus on the links between anxiety and alco-

hol use. Future well-controlled, randomized treatment outcome studies are necessary to examine the efficacy of such interventions for individuals with coexisting SAD/SUD.

Taken together, these studies have been limited by small sample sizes and a paucity of work examining treatment of substance use problems other than alcohol. Nonetheless, based on the case study examining a sequential treatment for SAD and alcohol use disorders, it seems that this treatment might be efficacious in reducing symptoms of SAD and alcohol-related outcomes (Buckner et al., 2008). Evidence from two randomized controlled trials does not support concurrent SAD and alcohol dependence treatments in improving alcohol-related outcomes, and the findings related to SAD reduction were mixed (Randall et al., 2001; Schade et al., 2005). Finally, two studies examined integrated treatments for SAD/SUD. The uncontrolled (Courbasson & Nishikawa, 2010) and the randomized controlled pilot study (Tran, 2008) of integrated interventions found successful reductions in anxiety symptoms. There were also successful reductions in alcohol-related outcomes in the pilot study (Tran); however, it is unknown whether the integrated intervention used in the Courbasson and Nishikaway (2010) study affected substance use outcomes as these dependent variables were not assessed. Overall, it seems that the results related to integrated treatments for SAD/SUD are the most promising.

Clinical case study. What follows is a case example of a client diagnosed with co-occurring SAD and alcohol dependence. "Alex" is a 27-year-old, White man who was treated in an addiction therapeutic community setting. His entry into the therapeutic community was precipitated by a driving under the influence (DUI) legal charge and short-term inpatient treatment primarily focused on detoxification. Three days following admission into the therapeutic community, Alex experienced a drinking "lapse." The facility's addiction counselor referred Alex to the therapeutic community's mental health provider to address co-occurring anxiety symptoms identified as relapse triggers. Specifically, he reported difficulty abstaining from alcohol while working in his job in a factory assembly line. Alex reported that, prior to this treatment episode, he typically drank before work, during breaks, and during the lunch hour on a daily basis to control his anxiety (in addition to evening drinking, for a total of 12–15 standard drinks on a typical day). On the day he lapsed, Alex reported that he was informed that a supervisor would be evaluating the assembly line employees over the next week. Alex reported that his anxiety about being scrutinized at work increased substantially and he felt he needed to drink to "calm [his] nerves." Subsequently, Alex drank three 12-oz beers at lunch. He noted that some of his co-workers regularly drank 1–2 beers over the lunch hour.

Assessment included a set of self-report measures, portions of a well-established semi-structured diagnostic interview, an unstructured clinical interview, and a review of an assessment report from a previous addiction-focused evaluation. Alex previously attended court-ordered Alcoholics Anonymous (AA) meetings related to a DUI charge 2 years earlier. He reported no previous treatment for SAD. Related to SAD, Alex reported that he experienced SAD symptoms for "as long as [he] could remember." Alex reported that he first consumed alcohol at age 14 and began abusing alcohol around age 16. Alex noted that, as a teenager, he began using alcohol to cope with anxiety about social interactions, particularly interactions with women that he found attractive. After graduating from high school, his drinking escalated. Alex reported he moved from a small, rural community to a larger community where he experienced increased anxiety as he was repeatedly faced with interacting with unfamiliar people. At the time of the initial assessment, Alex reported that his SAD symptoms interfered with his ability to engage in treatment as well as in daily activities inherent in living with others in the therapeutic community setting. He reported that, in the past, he figured out ways to mask his drinking so that he could go to work and attend AA meetings while intoxicated. Alex was particularly concerned about negative evaluation at work, but also reported that he avoided dating situations and interactions with members of

the opposite sex due to his SAD symptoms. The initial evaluation supported a diagnosis of comorbid SAD and alcohol dependence.

Alex completed the therapeutic community's standard addiction treatment, primarily comprised of a 12-step facilitation approach. Concurrent to addiction treatment, he was offered weekly individual treatment to target SAD and the relations between SAD and drinking behavior. The SAD intervention was based on the *Managing Social Anxiety* manual (Hope, Heimberg, & Turk, 2006), which includes psychoeducation about SAD, cognitive restructuring, and graduated exposure to feared situations. Three components were integrated into the manualized SAD treatment to address comorbidity: (1) psychoeducation focused on the association between SAD and alcohol use (and its reciprocal nature), (2) self-monitoring of alcohol use urges, and (3) cognitive restructuring examining Alex's biased beliefs about his ability to perform and cope with anxiety in social situations without the use of alcohol. Special attention was paid to feared situations that interfered with addiction treatment (e.g., AA meetings, interacting with housemates, refusing to drink alcohol with co-workers) in planning and implementing exposures.

Following completion of 20 cognitive-behavioral treatment sessions, Alex completed self-report measures and a targeted semi-structured clinical interview. The interview revealed that Alex still experienced SAD symptoms, but that these symptoms were in the subclinical range. Self-report measures suggested a clinically significant reduction in SAD symptoms. Alex remained abstinent from alcohol according to self- and collateral reports. He reported increased attendance and involvement at AA meetings, including active involvement with an AA sponsor. At discharge from the therapeutic community and 2 months after treatment completion, Alex continued to experience improvements in SAD symptoms and had not consumed alcohol. This case study illustrates the importance of considering both SAD and the alcohol use disorder in treating the comorbid condition, as well as the need to assess for SAD in addiction settings.

PTSD and Substance Use Disorder Comorbidity

As above for comorbid SAD/SUD treatment, arguments have been made for sequential, concurrent, and integrated treatment methods. Historically, clinicians have considered sequential treatment of co-occurring PTSD/SUD as the most clinically indicated method. Treatment of the SUD is typically considered first and then PTSD treatment if clinically indicated. This method can be advantageous in that reduced substance use may result in increased emotional stability and greater ability to benefit from PTSD treatment. Moreover, PTSD symptoms may remit following successful SUD treatment (Dansky, Brady, & Saladin, 1998). However, such patients may be medicating their PTSD symptoms with drugs. In this scenario, delaying PTSD treatment in favor of SUD treatment may increase the probability of an addiction relapse as PTSD-related triggers for substance use remain (Ouimette et al., 2003). Alternatively, congruent with the self-medication hypothesis of co-occurring PTSD/SUD (see Stewart & Conrod, 2003), if individuals are using drugs to manage PTSD symptoms, it seems rational to first treat PTSD and then offer SUD treatment if clinically indicated. This option also has a number of potential faults. Effective treatments for PTSD require rational examination of trauma memories and experiencing negative emotions. Increased exposure to such stressors could trigger increased substance use. Previous research has found that individuals who continue alcohol use or benzodiazepine use during PTSD treatment are more likely to drop out of treatment early compared to individuals abstinent from anxiolytic substances (van Minnen et al., 2002). Additionally, continued substance use during PTSD treatment could block or limit learning, which is a key mechanism in the effective treatment of PTSD. Such findings suggest that PTSD treatment without consideration of SUD complexities may hinder treatment effects.

Second, clinicians can consider treating both disorders concurrently. For example, a patient might receive prolonged exposure (PE) for PTSD, while at the same time receive cognitive-behavioral coping skills training targeting the SUD. This method may be limited in that each treatment is provided without consideration of the other. Considering evidence suggesting a strong functional relation among co-occurring disorders (Coffey, Stasiewicz, Hughes, & Brimo, 2006; Smith, Feldner, & Badour, 2011), concurrent treatment without consideration of the functional relations between the two conditions may be limited.

Third, clinicians can provide integrated treatment for co-occurring disorders. In this method, the same provider treats the PTSD and SUD at the same time. Specific focus is paid to the functional relationship between the co-occurring disorders. This option matches well with client preferences (Brown, Stout, & Gannon-Rowley, 1998) and addresses a more comprehensive set of intertwined functional relations within a client.

Efficacy. Since the mid-1990s, researchers have been designing and testing programs focused on treating the complexities associated with co-occurring PTSD/SUD. A variety of procedures have been implemented; however, there have been several key elements common in these programs including education, SUD treatment, PTSD treatment, and relapse prevention. Education typically focuses on increasing understanding of the basic components of SUD and PTSD and how they relate to each other. Coping skills training for SUD focuses on increasing efficacy and ability to manage emotions and behaviors related to addiction (Kadden et al., 1995). Elements of coping skills training include craving management, assertiveness training, relaxation training, anger management, and management of time and social life. Effective PTSD treatments examined thus far in the co-occurring PTSD/SUD literature typically include exposure procedures. Currently there are a limited number of studies examining effective methods of treating co-occurring PTSD/SUD.

Early investigations combined treatment procedures found to be effective for each disorder to address concurrent disorders. For example, in an uncontrolled study, Seidel, Gusman, and Abueg (1994) examined the effect of inpatient treatment incorporating cognitive modification, exposure, and coping skills training for Veterans with co-occurring PTSD/alcohol abuse. Cognitive modification appears to have targeted general belief systems as opposed to precise dysfunctional beliefs related to a specific traumatic event. They also applied an exposure-based therapy; however, the description of the exposure elements suggests that the participants engaged in a more general discussion of military experiences as opposed to targeting a particular memory for repeated prolonged exposure. Finally, relapse prevention strategies were applied. The authors reported that greater than 60% of the participants remained abstinent at 3 months posttreatment. Change in PTSD-relevant functioning was not reported. Although it is difficult to draw any firm conclusions about treatment mechanisms, this study provided a base from which future studies could expand upon.

In 2000, Triffleman reported findings of a small controlled examination of *Substance Dependence PTSD Therapy* (*SDPT*), an integrated treatment designed to address the unique needs of individuals with co-occurring PTSD/SUD. Participants were randomly assigned to either SDPT or TSF. TSF focuses specifically on eliminating substance use, but does not directly address PTSD symptoms. In the active treatment condition, participants first engaged in abstinence-focused SUD treatment (i.e., coping skills training). During this phase, they also received education on the interaction of PTSD symptoms and addiction. The second phase applied stress inoculation therapy (Meichenbaum & Cameron, 1983) while continuing to address addiction concerns. Cognitive modification, in vivo exposure (as long as can be tolerated), and coping skills training were the primary treatment components. All participants improved equally across groups indicating no difference between SDPT treatment protocol and TSF in treating co-occurring PTSD/SUD. Small sample size ($n = 19$) may have limited the ability to detect true differences, or perhaps mechanisms present in each treatment (e.g., common factors

such as regular sessions, empathetic therapist) may have driven the null outcome.

Donovan, Padin-Rivera, and Kowaliw (2001) provided preliminary data from an uncontrolled study showing that "Transcend," a group-based treatment program for co-occurring PTSD/SUD, can have a positive effect on relevant symptom profiles. "Transcend" included a variety of treatment procedures stemming from a diverse collection of theoretical backgrounds including constructivist, psychodynamic, and cognitive-behavioral. Coping skills training and TSF were provided for addiction concerns and unstructured exposure to memories of traumatic events was included for PTSD. Participants reported statistically significant decreases in all PTSD symptoms and in addiction severity. However, as noted by Riggs and Foa (2008), the magnitude of treatment gains was marginal, suggesting limited clinical impact. Further, it is difficult to determine the active mechanisms responsible for treatment effects given the variety of program components.

In an attempt to investigate more specific treatment mechanisms, Back, Dansky, Carroll, Foa, and Brady (2001) designed an integrated treatment for co-occurring PTSD and cocaine dependence. Substance use treatment focused on coping skills training, whereas PTSD symptoms were targeted through PE therapy. Treatment occurs across 16, 90-min sessions, twice weekly. The first five sessions focused on coping skills training to provide some stabilization and education concerning the functional relation between PTSD and addiction. PE is initiated in session six. In an uncontrolled study, Brady, Dansky, Back, Foa, and Carroll (2001) reported that treatment completers ($n = 15$ out of 39 initially enrolled) experienced statistically significant reductions in intrusive, avoidance, and hyperarousal symptoms from baseline measurements. Additionally, depression difficulties improved, as did addiction symptoms. Effect size data suggested clinically significant improvement in PTSD (Glass's delta = 1.80) and substance use (Glass's delta = 1.26) symptoms at posttreatment and 6-month follow-up.

Riggs and Foa (2008) summarize data from a recent NIAAA-funded study examining the effect of concurrent treatment of PTSD/alcohol dependence comorbidity (PI: Foa; Grant No. RO1 AA012428). Preliminary data from this randomized controlled trial was presented previously at meetings of the International Society for Traumatic Stress Studies (Riggs et al., 2003) and the Association for Advancement of Behavior Therapy (Riggs, Pai, Volpicelli, Imms, & Foa, 2004). Cognitive-behavioral treatment and medication management were provided for alcohol dependence while PE was provided for PTSD. Participants were randomly assigned to PE/No PE for PTSD and Naltrexone/Placebo for alcohol dependence. Findings suggested that individuals receiving active treatment components reported significant reductions in PTSD and SUD symptoms compared to control participants. Similar to the findings reported by Coffey et al. (2006), individuals receiving PE reported reduced alcohol cravings. These preliminary data suggest that in addition to the findings from Brady et al.'s (2001) integrated treatment study, concurrent treatments are also a promising avenue for treatment of co-occurring PTSD/Alcohol dependence.

Najavits (2002) developed an integrated treatment package for co-occurring PTSD/SUD entitled *Seeking Safety.* This package purposely omits exposure therapy elements for PTSD to limit the potential for relapse triggered by exposure-related negative effect. *Seeking Safety* incorporates cognitive, behavioral, and interpersonal elements to increase ability to manage and cope with PTSD and substance use difficulties. While this therapy has shown promise in terms of client acceptance, reduced suicidal threat, improved emotion management, and reduced substance use (Najavits, Weiss, Shaw, & Muenz, 1998), treatment outcome investigations have generally produced equivocal results in terms of PTSD outcomes. Previous reports have failed to find differences in PTSD symptoms compared to educational and relapse prevention control groups (Hien, Cohen, Miele, Litt, & Capstick, 2004; Hien et al., 2009).

Researchers have suggested that PE may be contraindicated for individuals with severe anger difficulties and suicidal/self-harm tendencies (e.g., Foa, Hembree, & Rothbaum, 2007) or individuals for whom exposure becomes emotionally

overwhelming (Coffey, Dansky, & Brady, 2003). As a preliminary attempt to address some of these concerns, Najavits, Schmitz, Gotthardt, and Weiss (2005) applied *Seeking Safety* to address difficulties in emotion regulation and included an exposure element in the treatment co-occurring PTSD/SUD ($n=5$). Findings indicated improvements in addiction and PTSD symptoms, in addition to suicidal risk. Such findings suggest that the inclusion of Seeking Safety with exposure therapy for PTSD may be a viable treatment option to address the complexities associated with co-occurring PTSD/SUD.

Current data provide promising avenues of exploration to address the clinical complexities associated with PTSD/SUD co-occurrence. Brady et al. (2001) and Riggs and Foa (2008) both provide preliminary data supporting concurrent and integrated treatment models utilizing cognitive-behavioral coping skills training and exposure therapy for PTSD. Additionally, preliminary evidence suggests that Seeking Safety may be an effective adjunct to exposure therapy in the treatment of PTSD/SUD. Although we have detailed studies resulting in promising treatments, there are numerous areas to examine further. The National Institute on Drug Abuse (NIDA) has funded a randomized controlled study to replicate and extend previous findings (Brady et al.) examining the impact of concurrent CBT for SUD and PE for PTSD in OEF/OIF Veterans (PI: Back, Grant No: RO1 DA030143). NIDA has also funded a randomized controlled project focused on examining the impact of PE in the concurrent treatment of PTSD/SUD which incorporates biological and emotion regulation elements in hopes of uncovering specific mediators of treatment change (PI: Hien, Grant No: RO1 DA023187). Additionally, in response to relatively high attrition rates (e.g., Brady et al.), the NIAAA has funded a randomized controlled trial examining the impact of MET on treatment retention for exposure-based treatment of co-occurring PTSD/SUD (PI: Coffey, Grant No: RO1 AA016816).

Currently, the co-occurring PTSD/SUD treatment literature has focused mostly on exposure-based treatment for PTSD. However, cognitive processing therapy (CPT: Resick & Schnicke, 1992) has also received considerable empirical support as an effective treatment for PTSD. In a direct comparison, Rizvi, Vogt, and Resick (2009) compared PE with CPT for women with PTSD. Interestingly, they found that younger women appeared to benefit more from CPT, whereas older women received more benefit from PE. Such findings suggest that specific treatment procedures for PTSD may evidence differential efficacy as a function of client characteristics. The debilitating effects and complex nature of this relatively common co-occurrence have provided necessary support for continued large-scale clinical trials examining various treatments targeting co-occurring PTSD/SUD. Future well-controlled treatment outcome studies are necessary to rigorously examine the effects of such interventions in order to determine what treatments work best for which clinical scenarios.

Clinical Case Study

The following is a case example of a Veteran with co-occurring PTSD, cannabis dependence, alcohol dependence, and cocaine dependence in full remission. "Joe" is a 55-year-old, African American combat Veteran who was treated in a VA PTSD/addiction treatment program. He served in Vietnam for 12 months. During this time, he experienced several combat-related traumatic events and exhibited functional difficulties stemming from PTSD symptoms for years. Joe had been unable to maintain regular employment and had difficulties related to family and friends due to his PTSD symptoms. He avoided crowds and other social activities due to anxiety stemming from trauma reminders. Joe frequently lost his temper, was constantly irritable, and had trouble sleeping due to frequent, disturbing trauma-related nightmares.

Although Joe experienced several events meeting the PTSD criteria for a trauma, he was able to identify the memory that intruded most frequently and had the greatest impact on current daily functioning. This particular event occurred when he was serving on a ship off the coast of

Vietnam. He and several friends were originally ordered to participate in a specific operation. However, at the last minute he was ordered to stay back while several of his friends boarded the helicopter and departed the ship. He then witnessed a missile strike the helicopter down. Just prior to and during this incident, he reported extreme fear. Following this incident, Joe reported feelings of helplessness, anger, disgust, and sadness. In the years following, he kept considering things he could/should have done to prevent this incident. He has repeatedly blamed himself for this incident for approximately the past 40 years.

Joe was referred to the PTSD/addiction treatment program following his most recent relapse. Joe completed an initial evaluation, which consisted of several questionnaires, an unstructured interview, and a drug screen. Joe reported two previous addiction treatments. Each lasted 1 month and occurred in a residential treatment facility focused specifically on treatment of SUDs. Following each treatment, Joe reported several months of sobriety; however, he stated that he eventually returned to substance use in order to cope with his trauma memories. Joe had not received treatment for his PTSD symptoms previously. Joe's responses on self-report measures were suggestive of PTSD and depressive symptoms in the moderately severe range. Joe was drinking 12 12-oz beers and smoking 3–4 marijuana joints daily. He reported a history of regular cocaine use; however, he denied use in the past 7 years. His initial drug screen was positive for cannabis. Baseline results yielded initial diagnoses of PTSD, cannabis and alcohol dependence, depressive disorder NOS, and cocaine dependence in full remission.

Given Joe's initial level of substance use, it was determined that immediate PTSD treatment would offer limited benefit. Therefore, our initial goal was to reduce substance use to a level that would allow for effective PTSD treatment. He was offered treatment in a 4-week dual diagnosis residential treatment program, which integrates educational elements of the relation between SUD and PTSD and provided cognitive-behavioral coping skills training for SUD treatment. Cognitive-behavioral coping skills training included education about addiction and PTSD, craving management, cognitive modification, problem solving, substance refusal skills, lapse management, assertiveness training, anger management training, and motivational enhancement training.

Following completion of the residential treatment phase, Joe transitioned into an integrated CPT/cognitive-behavioral coping skills outpatient treatment program aimed at treating PTSD and preventing relapse. The coping skills relapse prevention training focused on management of social life, time management, and managing internal and external triggers in addition to motivational enhancement. This treatment occurred in a group setting for 1 h each week. Consideration of the relation between substance use and PTSD symptoms was incorporated into group discussions. Additionally, Joe participated in individual outpatient CPT, which aims to correct inaccurate interpretations concerning the selected trauma event. Joe completed all 12 CPT sessions. In addition to directly examining faulty beliefs surrounding Joe's trauma, CPT skills were used to target and modify biased beliefs concerning Joe's perception of the connection between PTSD symptoms and his substance use.

Following completion of all 12 CPT sessions, there was a clinically significant drop in PTSD symptoms and depressive symptoms, as well as no substance use. These gains were maintained at a 3-month follow-up assessment. His drug screens confirmed his self-report. This case study illustrates the successful use of CPT combined with cognitive-behavioral coping skills training in the treatment of co-occurring PTSD/SUD. These findings also lend support for the integration of PTSD and SUD elements across treatment.

Conclusion and Future Directions

Though there is considerably more work focused on interventions for PTSD/SUD than SAD/SUD, there remain important gaps in the assessment and treatment knowledge base for both combinations of diagnoses. It also is notable that anxiety disorder/SUD comorbidity rates are

dramatically higher when considering drug dependence (OR = 6.2) in comparison to alcohol dependence (OR = 2.6; Grant et al., 2004). Given this backdrop, it is surprising that most researchers have focused on evaluating treatments for co-occurring anxiety disorders and alcohol dependence. There is a paucity of research examining interventions for co-occurring anxiety disorders and drug dependence. Future research should address this gap.

Further, more research is warranted that examines the efficacy of integrated treatment protocols for co-occurring anxiety disorders and SUDs. This is particularly lacking in the case of SAD/SUD, despite evidence that integrated treatments might result in improved SAD and drinking outcomes (PI: Tran; Grant No. R21AA014014; Tran, 2008) and that efficacious integrated treatments are available for treating individuals with co-occurring PTSD/SUD (e.g., Brady et al., 2001; Riggs et al., 2003, 2004).

Future research also needs to examine if different combinations of anxiety-focused and SUD-focused treatments might be more effective for certain types of clients. As previously discussed, it is possible that CPT and PE, both effective treatments for PTSD, could result in differential outcomes depending on client characteristics (Rizvi et al., 2009). In examining data from Project MATCH, a large-scale clinical trial comparing three major types of alcohol dependence treatments (i.e., CBT, MET, and TSF), Thevos, Roberts, Thomas, and Randall (2000) found that there were better alcohol use outcomes for socially anxious women (but not socially anxious men) treated with CBT compared to TSF therapy. Furthermore, it might be the case that a sequential model might be preferred to a concurrent or integrated model for some clients. For example, an SUD-focused treatment might be indicated as the first step in a client with severe substance dependence that rarely uses substances to self-medicate anxiety symptoms. Perhaps the level of interaction between substance use and anxiety symptoms might be examined in terms of treatment outcomes to determine whether this is an important consideration in treatment planning for individuals with co-occurring anxiety disorders and SUDs.

Despite the high prevalence, the resulting negative impact, and the complicating factors involved in the assessment and treatment of these comorbid anxiety and SUD conditions, relatively little is known about the optimal ways to serve individuals with co-occurring SAD/SUD or PTSD/SUD. Additional empirical attention to developing efficacious treatments for clients with these co-occurring conditions is clearly warranted.

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Part IV

Cross Developmental Complexities

Treatment of Comorbid Anxiety Disorders Across the Life span

19

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The fourth edition of the *Diagnostic and Statistical Manual for Mental Disorders* (DSM-IV; American Psychiatric Association [APA], 1994) lists 12 diagnostic categories for anxiety disorders, leading to over 25 distinct anxiety diagnoses on Axis I (Norton, 2008). Decades of research on these disorders have found very high rates of comorbidity (i.e., co-occurrence of disorders within an individual), both for mood disorders and other anxiety disorders (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Kessler, Chiu, Demler, & Walters, 2005). In fact, a substantial body of research suggests that presentation of a single anxiety disorder, with no comorbidity, is the exception and not the norm (Brown, Di Nardo, Lehman, & Campbell, 2001; Davis, Barlow, & Smith, 2010), even for those disorders with the lowest comorbidity rates (Goisman, Goldenberg, Vasile, & Keller, 1995). This information is not completely surprising, given the overlap in both etiology and diagnostic symptoms across these disorders (see Lawrence & Brown, 2008 for a review).

The high rates of overlap among anxiety disorders have numerous implications across the research and practice realms. One of the most important is that many clinical trials for treatment of disorders restrict the types of participants, particularly excluding those with highly complex comorbid conditions (e.g., psychosis or bipolar disorder) and instead focusing on "pure" clients. As a result, our current empirically supported treatments may not be generalizable to a large portion of clients—those with comorbid conditions (Goldenberg et al., 1996). Over the last 15 years, considerable effort has been put forth to examine how comorbidity influences treatment, as well as how treatment can influence comorbidity. Does the presence of a comorbid disorder make treatment more difficult? Will treating a primary disorder impact the comorbid disorder? The focus of the current chapter will be to review this research, particularly focusing on how such knowledge can allow psychologists and other mental health practitioners to more effectively treat clients who present with multiple, comorbid anxiety disorders.

Anxiety Disorders and Comorbidity

As mentioned, epidemiological- and community-based research have found high rates of comorbidity for persons with an anxiety disorder, even when compared to other categories of mental disorders (Toft et al., 2005). In terms of specific rates, research has shown differential patterns of comorbidity for

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the different anxiety disorders. For the below information, the first disorder named would be considered the primary diagnosis, with the following being the most typically comorbid diagnoses.

Generalized anxiety disorder (GAD) has shown high rates of comorbidity with several other anxiety disorders, with estimates from 66 to 83% (Goldenberg et al., 1996; Wittchen, Zhao, Kessler, & Eaves, 1994). For persons with primary GAD, the most commonly seen anxiety disorders are social and specific phobias (Borkovec, Abel, & Newman, 1995; Davis et al., 2010), panic disorder, and posttraumatic stress disorder (PTSD; Kessler et al., 2005). For panic disorder (with or without agoraphobia), the most commonly co-occurring anxiety disorders are specific phobias, social phobias, and generalized anxiety (Brown, Campbell et al., 2001; Kessler et al., 2005). In persons with PTSD, high rates of social phobias are seen (Zayfert, Becker, Unger, & Shearer, 2002), as well as panic disorder (Kessler et al., 2005), although the most commonly co-occurring disorders are non-anxiety ones (e.g., major depression and substance abuse; Brady, Killeen, Brewerton, & Lucerini, 2000).

Social anxiety disorder (also known as social phobia) is highly comorbid with GAD, obsessive-compulsive disorder (OCD; Davis et al., 2010), agoraphobia, specific phobias, and panic disorder (Kessler et al., 2005). Obsessive-compulsive disorder has some of the highest comorbidity rates, with almost half of persons being diagnosed with a co-occurring anxiety problem (Weissman et al., 1994), most commonly panic disorder, phobias, or generalized anxiety (Brown & Barlow, 1992; Davis et al., 2010; Kessler et al., 2005). For those with a primary diagnosis of a specific phobia, much lower rates of comorbidity exist compared to other anxiety disorders (Brown, Di Nardo et al., 2001); however, for those with a different primary anxiety disorder, specific phobias are the most common co-occurring disorder (Sanderson, Di Nardo, Rapee, & Barlow, 1990). Given the low incidence rates of persons with only a specific phobia presenting for treatment (Silverman & Kearney, 1992), though, it is more likely that a specific phobia will be a comorbid, non-primary diagnosis in someone presenting for treatment.

Impact of Comorbidity on Disorder Severity

General clinical and research consensus is that, compared to someone presenting with only a single anxiety disorder, those persons who meet diagnostic criteria for multiple anxiety disorders tend to have more severe symptoms. In terms of course, single anxiety disorders have a later onset and are more likely to remit on their own than co-occurring disorders (Bruce, Machan, Dyck, & Keller, 2001; Goldenberg et al., 1996). Research examining social phobia has found more severe pretreatment symptoms in both the primary (social phobia) and comorbid disorders (Erwin, Heimberg, Juster, & Mindlin, 2002; Mennin, Heimberg, & Jack, 2000). Similar findings have been seen with generalized anxiety (Belzer & Schneier, 2004; Nutt, Argyopoulos, Hood, & Potokar, 2006; Wittchen et al., 1994). In one study examining PTSD in a noncombat population, comorbid anxiety disorders were not related to PTSD severity (Zayfert et al., 2002), although a later study found that those with PTSD and a comorbid diagnosis of social phobia were more clinically severe than those with PTSD or social phobia alone (Zayfert, DeViva, & Hofmann, 2005).

There are, however, mixed outcomes in primary panic disorder diagnoses, with some studies finding higher symptoms (Allen et al., 2010) and some finding no differences in symptoms between persons who did and did not have comorbid disorders (Tsao, Mystkowski, Zucker, & Craske, 2005). One study in particular that studied comorbid GAD and panic disorder found that those with both had significantly less satisfaction in personal relationships, lower functioning, and lower emotional health (Massion, Warshaw, & Keller, 1993).

Anxiety Comorbidity and Treatment Impact

There has been a substantial body of research devoted to examining the treatment implications of comorbidity. Overall, the presence of comorbidity does not appear to diminish the benefits of

empirically supported treatments. For example, Brown, Antony, and Barlow (1995) found that the presence of comorbid conditions in primary panic disorder did not impact short- or long-term outcome for panic symptoms. Most research has yielded similar results about panic symptoms (e.g., Tsao et al., 2005), but not all studies have confirmed such findings. One replication instead found comorbidity was associated with less likelihood of treatment response (Tsao, Lewin, & Craske, 1998).

Results are more mixed with other anxiety disorders. Treatment outcome for primary GAD with or without any comorbid conditions has shown no differences in short-term treatment response (Mennin et al., 2000), although others have shown comorbid anxiety disorders to decrease remission rates in the long term (Bruce et al., 2001). In contrast, GAD comorbid with OCD or panic disorder has been linked to attenuated treatment response (Steketee, Chambless, & Tran, 2001). Primary social phobia treatment outcome has not been negatively impacted by comorbid anxiety disorders (Brown et al., 1995).

Research on OCD is also mixed. Having primary OCD with comorbid PTSD has been found to decrease response rate (Gershuny, Baer, Jenike, Minichiello, & Wilhelm, 2002), while OCD and comorbid GAD were shown to increase dropout rates and decrease treatment response (Steketee et al., 2001). In contrast, others have shown no negative impact on OCD treatment from comorbid anxiety problems in adults (Steketee, Eisen, Dyck, Warshaw, & Rasmussen, 1999; Storch et al., 2010) or children (Storch et al., 2008).

Treatment Approaches for Comorbid Anxiety Disorders

As discussed above, research has begun to examine the impact of treatment approaches for comorbid anxiety disorders with mixed results. As reviewed below, recent literature has demonstrated decreases in symptom severity and number of comorbid conditions following treatment for certain disorders, but not for others. Two distinct approaches to treating comorbid anxiety disorders have been developed and will be outlined below: transdiagnostic treatments and treatments designed to specifically treat specific combinations of disorders.

Transdiagnostic Approaches

Transdiagnostic treatments are predicated on three notions (Norton, 2008). First, that anxiety disorders may not be independent from one another; second, that treatments for specific anxiety disorders are robust; and finally, that some diagnoses may lead to other diagnoses. Given these ideas, treatment geared toward the primary diagnosis should then generalize to the comorbid diagnoses as well. Due to the fact that there is no one "transdiagnostic treatment," but are instead treatments for specific disorders, below are summaries of research into transdiagnostic therapies, arranged by the primary diagnosis treated.

Social Phobia: One example of transdiagnostic treatment is cognitive-behavioral group therapy (CBGT; Juster & Heimberg, 1994; Heimberg, 1991; Heimberg & Juster, 1994). This type of treatment was first developed for use with individuals with social phobia and has been adapted for transdiagnostic groups as discussed below. Cognitive-behavioral group therapy begins with providing individuals with a cognitive framework for understanding the link between faulty beliefs about and anxiety during social interactions. Restructuring activities are then employed to help participants identify negative automatic thoughts, begin to classify them by cognitive distortion type, dispute negative thoughts using Socratic questioning and challenging of underlying assumptions, and finally developing coping self-statements. The behavioral component follows cognitive therapy. Individuals develop concrete goals (i.e., measurable, observable, attainable) for exposures. Further, participants are given "homework" assignments to complete exposures between sessions. Since CBGT is conducted in a group setting, this improves access to care and helps normalize symptoms of social phobia.

Mennin et al. (2000) examined the efficacy of CBGT in 122 adults with social phobia with and

without GAD. Results indicated significant improvement in social phobia symptoms as well as the comorbid GAD symptoms. They proposed that pretreatment GAD symptoms exacerbate the avoidance, fear, and worry associated with social phobia, thus treatment of social phobia may generalize to the overlapping symptoms in GAD (Mennin et al., 2000). This seemed to be confirmed by their findings.

Specific Phobias: A one-session cognitive-behavioral treatment has been developed for specific phobias (Ost, 1989, 1997). It is predicated on the belief that individuals experience catastrophic thinking related to the feared stimuli which leads to avoidance. Therapy uses exposure to allow the individual to have positive experiences with the feared stimuli which allow individuals to collect experiences that contradict their faulty assumptions about the stimulus. Treatment is conducted in a single session that may last up to 3 h. Participants are gradually exposed in vivo to the feared stimuli. Cognitive strategies are not used by the therapist during session, but participants are encouraged to draw conclusions based on the positive experiences. With each step in the graduation, the participant must experience a reduction in anxiety of at least 50%.

In two studies, Ollendick, Ost, Reuterskiold, and Costa (2010) and Ost, Svensson, Hellstrom, and Lindwall (2001) report that youth undergoing this therapy experienced not only a significant drop in symptoms of the primary anxiety disorder (specific phobia) but also reductions in clinical severity of other comorbid phobias and anxiety disorders. They attribute this to increased self-efficacy following treatment of and "conquering" a specific phobia.

Generalized Anxiety Disorder: Different combinations of treatments have shown success in treating GAD and comorbid anxiety disorders. However, some common elements of treatment are shared by those found to be the most effective in reducing GAD and other comorbid diagnoses. Each of the proposed treatments involve self-monitoring of symptoms and cues, employing active strategies to reduce anxiety, and practice using skills between sessions (Newman & Borkovec, 1995). For example, self-control desensitization therapy (Goldfried, 1971) requires individuals to practice relaxation skills in response to exposure to anxiety-producing stimuli and worries. Participants are taught to use pleasant imagery, diaphragmatic breathing, and progressive muscle relaxation. They are then instructed to use these skills when cued to by an anxiety provoking situation. Additionally, cognitive therapy also been examined. As discussed previously, cognitive therapy for anxiety involves identification of negative automatic thoughts, systematic evaluation of anxious thoughts, and restructuring techniques to make thoughts more adaptive. Specific to GAD, cognitive therapies typically include discussion of intolerance of uncertainty, poor problem-solving, and avoidance. Finally, CBT adds behavioral exposure to cognitive training. Following cognitive training, participants are encouraged to use skills in real-world situations with clear goals for each exposure.

Newman, Przeworski, Fisher, and Borkovec (2010) compared a treatment combination of self-monitoring, active anxiety reduction, and homework assignment in a sample with GAD and comorbid anxiety disorders. Results indicate that psychotherapy for primary GAD led to a reduction in number and severity of comorbid anxiety disorders at both posttreatment and follow-up. Borkovec et al. (1995) reported similar results in a sample of adults with GAD. Participants experienced a reduction in primary GAD as well as significantly fewer comorbid diagnoses following successful treatment of GAD. Ladouceur et al. (2000) examined a cognitive treatment for GAD. Results suggest that focusing on GAD specific elements of cognitive therapy lead to reductions in comorbid anxiety disorders. Specifically, they reported that participants had more than a 50% decrease in number of comorbid illnesses which were maintained at follow-up 1 year later.

Panic Disorder: Panic disorder treatment has also been shown to impact comorbid conditions. Typically, treatment of PD uses cognitive-behavioral methods developed by Craske and Barlow (Panic Control Treatment, 1993). First, individuals are provided with a physiological explanation for panic symptoms; then, they are taught cognitive

restructuring techniques to combat overestimations of risk related to these symptoms. Next, participants learn breathing retraining for use when hyperventilation occurs during a panic episode. Individuals are then gradually exposed to internal and external cues to panic using interoceptive (internal) and in vivo (external) exposure. Self-monitoring of anxiety and home practice are also required between treatment sessions.

Several authors have examined the effect of this treatment in PD with comorbid diagnoses (e.g., Brown et al., 1995; Tsao et al., 2005). Brown et al. (2001) reported a dramatic decrease in number and severity of comorbid conditions at posttreatment. However, in contrast to other research, participants had returned to pretreatment levels of comorbidity at 2-year follow-up and were more likely to seek additional treatment between follow-ups. Tsao et al. (2005) found that participants experienced significant reductions in both PD symptoms and comorbid diagnoses. Interestingly, the reduction in comorbid diagnoses in this sample was not significant until follow-up (9 months), which is a departure from other research. However, severity of comorbid diagnoses did decrease from pre- to posttreatment.

Specific Therapies

In contrast to transdiagnostic therapies, there are two therapies that have been designed to treat specific clusters of anxiety disorders. For example, multichannel exposure therapy (MCET) was developed to treat posttraumatic stress disorder (PTSD) and comorbid panic attacks (Falsetti, Resink, & Davis, 2005). This treatment combines aspects of cognitive processing therapy (Resick & Schnicke, 1993) and panic control treatment (Barlow & Craske, 1994). Treatment in MCET targets symptoms of both PTSD and panic attacks through physiological, cognitive, and behavioral channels. Initially, participants in MCET are introduced to the idea that panic attacks and symptoms are conditioned responses to conditioned stimuli related to the initial unconditioned trauma. Following from this explanation, treatment includes (1) cognitive restructuring techniques to respond to catastrophizing and overestimation of trauma, (2) interoceptive exposure to physiological symptoms of panic and in vivo exposure to reminders (cues) to trauma, and (3) additional cognitive work involving exposure to the trauma via writing and reading aloud about the initial trauma.

Research has shown MCET to be effective over a wait list control (Falsetti, Erwin, Resnick, Davis, & Combs-Lane, 2003) as well as a minimal attention control (Falsetti et al., 2005). Falsetti, Resnick, Davis, and Gallagher (2001) used the above model in 22 women in a group format. They reported reductions in the number of women meeting diagnostic criteria for PTSD, panic attacks and symptoms, fear of having another attack, as well as reduction in the interference associated with attacks that were present and symptoms of depression. Additionally, Falsetti et al. (2003) reported improved function in a sample of female trauma victims following MCET. Participants experienced reduced symptoms of PTSD and panic attacks and experienced improvement in functioning in work, marriage, and general functioning. Reductions were maintained at 3- and 6-month follow-ups. Treatment gains have been shown to extend past the 6-month follow-up as well (Falsetti, Resnick, & Davis, 2008).

A second specific therapy has been developed for individuals with GAD and comorbid panic disorder with agoraphobia (PDA; Labrecque, Dugas, Marchand, & Letarte, 2006). This treatment method is based on the notion that available cognitive-behavioral therapies of the individual disorders shared common components that could be used in treating both disorders together. Labrecque et al. (2006) combined features from other available treatments for panic (Craske & Barlow, 1993; Marchand & Letarte, 1993) and GAD (Dugas & Ladouceur, 2000; Ladouceur et al., 2000). One major tenant of combining the treatments is to help individuals tolerate the uncertainty/discomfort that accompanies both GAD and PDA. Treatment components include providing psychoeducation to participants about anxiety and specific diagnoses, information regarding symptoms and maintenance of panic, breathing

retraining, cognitive restructuring, confronting beliefs, interoceptive and in vivo exposures, exposure to worries, training in problem orientation, and relapse prevention. Results of preliminary studies have demonstrated a reduction in symptom presentation of both GAD and PDA at post-treatment and follow-up (Labrecque et al., 2006; Labrecque, Marchand, Dugas, & Letarte, 2007).

Case Study

Leah (a pseudonym) was a 16-year-old Caucasian female referred by her psychiatrist for evaluation and treatment of obsessive-compulsive disorder, panic disorder without agoraphobia, and major depressive episode, recurrent. Specific symptoms of both anxiety disorders are listed below. Leah received her initial diagnoses from her psychiatrist according to the DSM-IV-TR (APA, 1994) at the age of 14 years. Leah was referred for treatment due to increased anxiety in numerous situations, ritualistic behaviors (i.e., excessive cleaning and organizing), and impairment in social and academic functioning. Based on her symptoms, impairment, and results of clinical evaluation, Leah was given the above diagnoses. Leah was otherwise typically developing aside from hypothyroidism which was managed by a pediatric endocrinologist and stable at treatment entry. She was in a regular classroom and during periods of lower anxiety was performing above average academically and had several friends.

Assessment of Anxiety Symptoms

Leah began exhibiting a significant number of ritualistic and avoidant behaviors around the age of 14 years. Specifically, these included contamination concerns, avoidance of contaminated items (e.g., public areas), excessive cleaning/washing (i.e., items in the home such as books as well as her person), concerns about organization, excessive list making, and other just right phenomena (i.e., evening things out, repeating tasks until they felt just right). Leah reported feeling increasingly anxious when prevented from engaging in rituals. Her symptoms began interfering with academic, social, and family functioning at this time. Specifically, teachers noticed that assignments were completed only after long delays and that she was not spending time with peers during free periods. At home, Leah spent considerable amounts of time organizing and straightening her room. For example, each time she needed to put clean laundry away, she would refold all clothes in the same container/drawer.

In terms of panic symptoms, Leah began having panic attacks (PA) once per month after her 15th birthday. She experienced her first PA at home while relaxing. This attack lasted approximately 15 min and involved increased heart rate, sweating, shaking, nausea, and derealization. In response to the PA, Leah became concerned with having another panic attack and worried that she was "going crazy." Hence, she limited social interactions due to these concerns. Leah also experienced symptoms of MDD, likely as a result of OCD and PD. Specifically, Leah reported feeling depressed mood and anhedonia. These mood disturbances appeared to result from her combination of OCD and PD, specifically her functional impairment (e.g., lack of social interactions and limited range of pleasurable activities). She was sleeping more than 11 h per night, felt lethargic, and reported extreme indecisiveness. This was differentiated from indecisiveness related to anxiety by the nature of content (i.e., minor decisions rather than related to organizing or just right phenomena).

Treatment

Leah presented for 14 sessions of CBT with exposure and response prevention. Therapy was based on the model proposed by March and Mulle (1998) and validated in several studies (e.g., POTS, 2004; Storch et al., 2007). Treatment was conceptualized as a transdiagnostic approach and included additional sessions in which physiological symptoms of panic were targeted using CBT principles.

Sessions one and two focused on an introduction to therapy, psychoeducation, and fear hierarchy construction. Leah was asked to begin monitoring her thoughts between sessions and

recording them using a thought record. Leah completed this easily and brought back a significant number of thought records between sessions. Hierarchy items included contamination related items (e.g., showering and cleaning excessively) and numerous organization/just right phenomena (e.g., list making, reorganizing bedroom and clothes). Leah's final hierarchy consisted of 25 items, the majority of which were related to avoidance and organizing. Initial cognitive work was begun in the second session; this involved having Leah begin to differentiate between "typical" and "OCD" worries and concerns.

Sessions three through eight primarily consisted of exposure and response prevention work. As an example of an exposure, Leah was required to "disorganize" her wallet, backpack, or computer files while in session. Exposures were continued until habituation to the feared stimuli was achieved and her anxiety was greatly decreased. Leah initially resisted some of the exposures, stating that they were not relevant to her treatment. However, over the course of treatment, Leah began to acknowledge that habituation in session and during homework was generalizing to more naturalistic, real-world situations and, thus, was valuable. Exposures were conducted during each session with increasing difficulty. Due to Leah's PA symptoms, particular attention was given to the physiological symptoms of anxiety during exposures. Other tasks during this portion of treatment involved reviewing homework from between sessions, which typically included additional exposures in the home as well as continued cognitive work. Leah also reviewed and practiced coping statements and cognitive restructuring in session.

Sessions 9–12 focused on continued exposure with the introduction of interoceptive exposures for panic symptoms. As Leah had been primed to attend to physical symptoms in earlier exposures and to actively use coping statements, she was receptive to this type of exposure. Leah was assigned additional interoceptive exposures in places and at times which were related to her PA (e.g., at school, the mall). For example, one homework assignment was to go to a shopping center in the area and walk up and down the steps between floors until her heart rate was elevated at least three times during a shopping trip and remain at mall for a minimum of 30 min following exposure or until habituation occurs. We also continued to review cognitive strategies at the end of each session.

Relapse prevention and preparation for termination were covered in session 12. Psychoeducation was provided regarding possible triggers for Leah's symptoms to return, for example, increased stress or less attention paid to returning symptoms. The importance of continued exposure and cognitive work at home was reinforced. Leah was asked to prepare any questions to be discussed with the therapist in the final session. Session 14 was conducted 3 weeks after session 13 to allow Leah to have additional time to work on symptoms at home. In the final session, termination was discussed and relapse prevention reviewed.

Assessment

Leah completed the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS; Scahill et al., 1997) at pre- and posttreatment (session 13). At pretreatment, Leah received a severity rating of 20, indicating moderately severe OCD symptoms. At the final session, Leah's CY-BOCS score had reduced to 3, which is well within normal limits. Although Leah has not returned for a follow-up assessment, per her psychiatrist she continues to maintain posttreatment levels of anxiety. Prior to treatment, Leah was experiencing monthly PA with worry about having one on a daily basis. After the first month of treatment, during which time Leah had one PA, Leah did not experience any further PAs. Worry about her attacks decreased over the course of treatment, so that at her 14th appointment Leah reported only minimal worry (1 out of 10) and noted that this only occurred occasionally. Although there was no specific measure of MDD symptoms, anecdotally, Leah reported increased engagement in activities with friends, improved mood, decreased sleep, and she obtained a job at a coffee shop which she enjoyed.

Future Directions

Although much has been learned over the last 20 years regarding comorbidity in anxiety disorders, many questions are still unanswered. Currently, the most pressing need for research concerns finding the most effective and efficient treatment. As mentioned above, many studies investigating the treatment of anxiety disorders use participants without comorbid or complex presentations, undermining the ability of the results to generalized to the majority of clients (Goldenberg et al., 1996). Evidence suggesting that frequency and severity of comorbidity does not predict CBT treatment outcome (Davis et al., 2010; Storch et al., 2010) implies that exclusion criteria for CBT research studies no longer need to include comorbid anxiety disorders.

Future research needs to establish whether focusing treatment on the primary diagnosis is superior to combining treatments for primary and comorbid diagnoses into one treatment (Labrecque et al., 2007). Studies have found treatments focusing on the primary diagnosis not only effectively treat the primary diagnosis but also significantly decrease the frequency and severity of comorbid disorders (Newman et al., 2010; Ollendick et al., 2010). There is, however, evidence that combining treatments for both the primary and comorbid diagnosis is effective (Labrecque et al., 2007), which has led to the development of transdiagnostic treatments (such as those as reviewed above) that emphasize commonalities across disorders to treat the primary and comorbid diagnoses concurrently (McEnvoy, Nathan, & Norton, 2009). Future research should directly compare diagnosis-specific (e.g., MCET) and transdiagnostic treatments to determine each method's effect on outcome variables such as diagnosis-specific symptoms, higher-order and common factors (e.g., negative affectivity, locus of control, emotion regulation), nonspecific factors (e.g., therapeutic alliance, group cohesion), treatment compliance and attrition, and duration of treatment effects and remission rates on primary and comorbid diagnoses (Storch et al., 2010). Research also needs to be undertaken to determine if treatments addressing both disorders simultaneously are superior to those treating disorders separately in discrete sessions (Reinecke & Hoyer, 2010).

A thorough understanding of the mechanisms that facilitate improvement of comorbid disorders may assist in determining whether diagnosis-specific or combined treatments are more effective. The answer may depend on the type and relatedness of the comorbid disorders (Reinecke & Hoyer, 2010) or the components within the treatment (Labrecque et al., 2007). Approaches that emphasize commonalities across disorders significantly reduce comorbid disorders (McEnvoy et al., 2009). For example, Labrecque et al. (2007) found that treatments that identified intolerance of bodily sensations for PDA and intolerance of uncertainty for GAD as vulnerability factors were preferred by participants. The authors suggest treatment models that address intolerance may be effective regardless of which disorder is the main diagnosis. Another study found that the more perceived control participants reported on a post-treatment anxiety control questionnaire, the greater the decrease in comorbidity (Craske et al., 2007). It is also possible, however, that decreases in comorbid disorders occur because clients apply techniques learned in treatment to their comorbid disorders (McEnvoy et al., 2009). Given that effective treatments for various anxiety disorders tend to share common features (e.g., exposure with response prevention, cognitive restructuring, relaxation training), this seems likely.

The optimal intensity of treatment also remains unclear, as one study reduced the clinical severity of comorbid anxiety disorders, which are typically treated with 12–16 sessions of CBT, using only one 3-h session to treat youth with specific phobias (Ollendick et al., 2010). It is unknown if other intensive treatments (e.g., multiple times weekly, extended therapy sessions) for different primary disorders would have the same impact. Future research also needs to identify the mechanisms leading to optimal outcomes and ascertain if the same effects occur for these disorders when they are more severe or when they are the primary disorder rather than the comorbid one.

Despite all that is known, more advances in the understanding of comorbid anxiety disorders

need to be made. Najavits et al. (2008) speculate that future research will result in advances in the types of studies conducted (e.g., an increase in randomized controlled trials), increases in training and dissemination, and a deeper understanding of the comorbidities themselves (e.g., rates, causal relationships, and prognosis).

Conclusions

Over the last two decades, our knowledge regarding anxiety disorders has grown immensely, especially in our understanding of issues surrounding comorbidity. We are now beginning to understand how different disorders present comorbidly, how those presentations impact both each other and treatment, and how to treat comorbid disorders. At the current time, there is strong evidence to support both transdiagnostic and specific therapies as effective, although much research remains before we have a full understanding of which works better. It may be that a transdiagnostic approach will work better for certain comorbid combinations while a specifically developed therapy works better for others, or it could be that both are equally effective.

Regardless, today's practitioners should be aware that both methods have been shown to be useful in treating numerous combinations of anxiety disorders, as shown in both the above reviewed literature and the case study. Undoubtedly, given the enormous rates of comorbidity seen in those with anxiety disorders, most practitioners treating those with anxiety are likely to already be using some type of transdiagnostic or personally developed specific therapies. It can be immensely beneficial to both researchers and clinicians to share this type of information with each other, particularly if more private practice clinicians were able to share knowledge gain from clinical experience and expertise. This could be done by engaging in systematic case studies regarding treatment methods and outcomes on typical, outpatient populations. Researchers should attempt to reach out to practitioners and form collaborations, supplying their expertise in data collection and analysis and possibly helping to secure grant funding so that the extra work involved in doing research in the context of a private practice would not be financially burdensome on practitioners.

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Family Conflict and Childhood Anxiety

20

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Most clinicians and researchers agree that child adjustment be viewed through a contextual lens, with the family environment being the first, and potentially most formative, backdrop affecting children. Heriditability estimates suggest familial aggregation of anxiety (for review, see Hettema, Neale, & Kendler, 2001), but genetic contributions are not large enough to account for all variability, suggesting family environment also be considered (Murray, Creswell, & Cooper, 2009). Anxious children are more likely than less anxious peers to have a dysfunctional family (Pagini, Japel, Vaillancourt, Côté, & Tremblay, 2009), and family factors such as cohesion, adaptability, parenting, stress and social support, and marital quality have been associated with various pediatric anxiety disorders (Côté et al., 2009; Lange et al., 2005; Peleg-Popko & Dar, 2001, 2003). Moreover, family conflict and dysfunction may partially account for interfamilial transmission of anxiety (e.g., Drake & Kearney, 2008; Du Rocher Schudlich & Cummings, 2003) and family dysfunction predicts anxiety treatment outcomes (e.g., Crawford & Manassis, 2001). Yet, family conflict is often neglected in theoretical formulations of anxiety development, and family conflict studies too infrequently consider anxiety apart from depression. As no review has singularly focused on family conflict and pediatric anxiety, the chapter synthesizes the two literatures in hopes of inspiring increased attention to family conflict and its implications for pediatric anxiety.

Parenting practices, particularly psychological control and intrusiveness, are implicated in anxiety development and maintenance (McLeod, Wood, & Avny, 2011). Some recent reviews of family matters in pediatric anxiety suggest that other aspects of family life, including family conflict, are also relevant (Bögels & Brechman-Toussaint, 2006; Bögels & Phares, 2008; Chorpita & Barlow, 1998; Elizabeth et al., 2006; Hughes & Gullone, 2008; Murray et al., 2009; Weich, Patterson, Shaw, & Stewart-Brown, 2009). For example, family chaos at age four predicts anxiety in middle childhood (Asbury, Dunn, & Plomin, 2006). Conflict within families is ubiquitous, normative, unavoidable, and when infrequent and effectively resolved, typically is not harmful (Adams & Laursen, 2007; Montemayor, 1983). However, frequent, intense, and poorly resolved conflict can be detrimental for families and children (see Fincham & Osborne, 1993). Given that families are complex and transactional (Eichelsheim, Deković, Buist,

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& Cook, 2009), conflict can pervade families such that dysfunction and chaos spreads throughout. Thus, while we examine dyadic conflict separately, namely, interparental conflict, parent–child conflict, and the role of siblings, we highlight reciprocal effects and potential mechanisms of influence, including the interrelationship between interparental conflict and parent–child conflict. Following a brief consideration of family conflict beyond adolescence, we conclude with recommendations for the next generation of research, discussion of treatment considerations, and an illustrative case study.

Interparental Conflict

Various conceptualizations and examinations of interparental conflict (IPC) exist, ranging from domestic violence to everyday squabbles between couples. Although IPC is associated with a wide range of adjustment difficulties, including but not limited to internalizing symptoms, results from meta-analyses suggest that not all children who witness IPC are negatively impacted (see Table 20.1 for summary of meta-analyses). However, witnessing domestic violence, an extreme form of IPC, is associated with pediatric anxiety and posttraumatic stress symptoms (for reviews, see Guille, 2004; Margolin & Gordis, 2000). A recent meta-analysis suggests a moderate effect between family violence and internalizing symptoms and a slightly stronger association with posttraumatic stress symptoms (Chan & Yeung, 2009). Moreover, an earlier meta-analysis found children exposed to interparental violence experience levels of internalizing symptoms similar to physically abused children (Kitzmann, Gaylord, Holt, & Kenny, 2003). Meta-analytic results specific to IPC indicate a small-to-moderate relationship between marital conflict and internalizing symptoms and a slighter strong relationship with externalizing disorders (Beuhler et al., 1997). As anxious and depressive symptomology regularly co-occur, meta-analysis of these effects specific to anxiety has yet to be conducted.

While meta-analyses suggest a moderate association between IPC and internalizing symptoms (see Table 20.1), they do not provide information regarding the nature or direction of the relationship. Interparental conflict may serve to produce, maintain, or exacerbate anxious impairment in children. Longitudinal evidence suggests that the marital relationship, and IPC in particular, can lead to later internalizing symptoms (e.g., Cummings, Schermerhorn, Davies, Goeke-Morey, & Cummings, 2006; Harold, Shelton, Goeke-Morey, & Cummings, 2004; Nomura, Wickramaratne, Warner, Mufson, & Weissman, 2002). For example, IPC has been found to predict internalizing symptoms 5 years later (Gerard, Krishnakumar, & Buehler, 2006). Specifically considering anxiety, a distressed marital relationship has been associated with adolescent-reported anxiety 8 years later, even after controlling for maternal psychopathology (Spence, Najman, Bor, O'Callaghan, & Williams, 2002).

In a prospective study, family discord (family cohesion, divorce, marital, and parent–child relationships) was associated with the child developing an anxiety disorder within 10 years (Nomura et al., 2002). Results demonstrated a fourfold increase in anxiety disorders when family discord was present in families without parental depression. This study may be the strongest evidence that the family factors and marital relationship are related to clinical anxiety. However, the study did not examine IPC specifically or potential associations between family discord and parental depression. Hence, family conflict may mediate the relationship between parental psychopathology and internalizing symptoms. In support of this possibility, Drake and Kearney (2008) found that family environment and conflict mediated the relationship between parent and child anxiety sensitivity. Similarly, a depressive conflict style observed during a martial conflict problem-solving task mediated parent's depressive symptoms and child's internalizing symptoms (Du Rocher Schudlich & Cummings, 2003).

While meta-analysis specific to child anxiety is pending, longitudinal data suggest a directional link from the marital relationship and IPC to child internalizing symptoms. While most studies examine anxiety and depressive symptoms

Table 20.1 Summary of family conflict meta-analyses

Citation	Family conflict	Outcome	Effect size (*r*)
Beuhler et al. (1997)	IPC	Internalizing symptoms	0.15
		Externalizing symptoms	0.19
Chan and Yeung (2009)	Family violence (IPC and child abuse)	Internalizing symptoms	0.22
		Posttraumatic stress symptoms	0.35
Erel and Burman (1995)	IPC	Parenting behaviors	0.22
Kitzmann et al. (2003)	Interparental violence	Internalizing symptoms	0.17
		Posttraumatic stress symptoms	0.25
Krishnakumar and Beuhler (2000)	IPC	Parenting behaviors	0.30
Rhoades (2008)	Affective reaction to IPC	Internalizing symptoms	0.31
	Cognitive reaction to IPC		0.34
	Self blame		0.36
	Percieved threat		0.40
	Physiological reaction to IPC		0.14
	Behavioral reaction to IPC		0.24
	Child involvement in conflict		0.29
	Avoidance		0.26
	Affective reaction to IPC	Externalizing symptoms	0.15
	Cognitive reaction to IPC		0.21
	Self blame		0.28
	Perceived threat		0.21
	Physiological reaction to IPC		0.11
	Behavioral reaction to IPC		0.14
	Child involvement in conflict		0.15
	Avoidance		0.04

Note. Some effect sizes were converted from Cohen's *d* to *r* for ease of comparison
IPC interparental conflict

conjointly, some do report significant associations between IPC and anxiety specifically (e.g., El-Sheikh & Elmore-Staton, 2004; Dewit et al., 2005; Kerig, 1998). Yet, anxiety and depression may be difficult to disentangle as they frequently co-occur, with 25–50 % of depressed youth also having an anxiety disorder (Axelson & Birmaher, 2001). As such, incremental effects specific to anxiety may be difficult to detect, especially if both anxiety and depression are related to family conflict. While the field may still be short of a definitive link between IPC and child anxiety, process-level research provides a more specified and nuanced understanding.

Interparental Conflict and Child Anxiety: Process-Level Research

Children are not merely passive receivers of their environment, so it is important to consider how children's processing of conflict is associated with internalizing symptoms. A recent meta-analysis (Rhoades, 2008) found moderate associations between children's internalizing symptoms and their affective, cognitive, behavioral, and physiological reactions to IPC (see Table 20.1). Cognitive, affective, and behavioral, but not physiological, reactions had greater associations with internalizing than externalizing

problems. Regarding internalizing symptoms, the effect sizes were larger for cognitive and affective, compared to behavioral and physiological, reactions. Thus, children's reactions to IPC likely determine whether internalizing symptoms develop. Viewed differently, children with internalizing symptoms may be more likely to experience maladaptive reactions to IPC. We group our discussion of mechanisms into three broad categories: (1) emotional processes and physiological reactions, (2) cognitive processes and behavioral reactions, and (3) family processes and parenting. One additional mechanism, the interplay of interparental and parent–child conflict, will be discussed in the later section focusing on parent–child conflict.

Emotional processes and physiological reactions. The emotional security hypothesis (Davies & Cummings, 1994; see also the specific emotions model Crockenberg & Langrock, 2001) posits that negative emotional reactions to IPC over time result in a more chronic state of emotional insecurity. Emotional security refers to a child's "felt security," or perception of safety, with respect to their family. Both concurrent and longitudinal support has been provided for the emotional security hypothesis as an explanatory mechanism for the development of internalizing symptoms in the context of IPC (e.g., Cummings et al., 2006; El-Sheikh, Cummings, Kouros, Elmore-Staton, & Buckhalt, 2008; Harold et al., 2004; Shelton & Harold, 2007).

The emotional security hypothesis suggests implications for understanding child anxiety. First, anxiety may result from witnessing IPC if children are less able to manage their emotional and physiological reactions (Davies & Cummings, 1994). The ability to regulate physiological arousal, as indicated by vagal tone, has buffered children exposed to IPC from developing internalizing symptoms (El-Sheikh, Harger, & Whitson, 2001). Moreover, physiological reactivity is a stable trait of anxiety (Olatunji & Cole, 2009), and anxious children experience and express heightened negative emotions in response to negative events (e.g., Suveg et al., 2008). Thus, anxious children will likely have difficulty regulating physiological reactions to IPC, which could exacerbate anxious symptoms. Second, repeated exposure to IPC may sensitize a child such that emotional responses, and felt insecurity, are heightened (Davies & Cummings, 1994). Similarly, posttraumatic stress disorder includes heightened sensitivity to trauma-related cues and diminished regulation ability (Meiser-Stedman, 2002), and emotional security has been found to mediate the relationship between IPC and posttraumatic stress symptoms (El-Sheikh et al., 2008).

Cognitive processes and behavioral reactions to conflict. The cognitive-contextual framework recognizes the role of emotion but suggests that children's cognitive appraisals of conflict are more proximally responsible for child adjustment (Grych & Fincham, 1994). The theory postulates that *threat perceptions,* coupled with low coping efficacy, are related to anxiety and feelings of helplessness, while *self-blame* is associated with guilt, shame, and sadness. Several studies have implicated appraisals of self-blame and perceived threat in the relationship between IPC and internalizing symptoms (e.g., El-Sheikh & Harger, 2001; Gerard, Buehler, Franck, & Anderson, 2005), affective well-being (e.g., Xin, Chi, & Yu, 2009), and anxiety specifically (e.g., Kerig, 1998). Meta-analysis reveals internalizing symptoms are moderately associated with self-blame and perceived threat (Rhoades, 2008; see Table 20.1). A relationship between internalizing symptoms and children's cognitive processing of IPC seems clear, but less is known about how self-blame and threat perceptions are linked to anxiety specifically.

Similar to the cognitive-contextual model of IPC, cognitive models of anxiety development posit that prolonged, repeated, and early exposure to events perceived as uncontrollable leads to a diminished sense of control (e.g., "uncertain helplessness") resulting in a consistent affective state of anxious arousal (Chorpita & Barlow, 1998). Interparental conflict could certainly represent an early, and likely repeated and prolonged, negative life event that could be perceived as uncontrollable, and thus lead to anxiety. Pediatric anxiety is associated lower coping efficacy and cognitive bias to interpret ambiguous, and even benign, situations as threatening (e.g., Bögels & Zigterman,

2000; Creswell & O'Connor, 2006; Kortlander, Kendall, & Panichelli-Mindel, 1997; Lester, Seal, Nightingale, & Field, 2010). It follows that anxious children will be especially likely to interpret threat to less ambiguous events, such as IPC, that could pose an imagined or real threat to their well-being (e.g., separation from parent, divorce, or change in schools), since anxious children display hypervigilant attention to external threat cues (e.g., Schultz & Heimberg, 2008; Meiser-Stedman, 2002). Children, more so if anxious, may look to their parents to determine whether an IPC poses a threat (e.g., Creswell & O'Connor, 2006; Lester et al., 2010). Anxious parents, and parents of anxious children, may be more likely to inadvertently display threat cues or overtly convey threat information (e.g., Murray et al., 2009). Thus, IPC could contribute to anxiety development if the child interprets the event as uncontrollable and threatening. Moreover, anxious children are especially likely to perceive IPC as threatening.

While appraisals of self-blame are thought to be more related to depression than anxiety, they remain significantly correlated with anxiety, albeit to a lesser degree, when depressive symptoms are controlled (Matheson & Anisman, 2003) and general self-blaming tendencies have been associated with adult anxiety (e.g., Kelly, Tyrka, Price, & Carpenter, 2008). Children are more likely to self-blame when IPC is child-centered (Grych & Fincham, 1994), and child-centered conflict has been tied to internalizing symptoms (e.g., Gordis, Margolin, & John, 2001) and anxiety specifically (Snyder, Klein, Gdowski, Faulstich, & LaCombe, 1988). Parenting an anxious child can be stressful for families (Kalra, Kamath, Trivedi, & Janca, 2008; Lange et al., 2005; Storch et al., 2009), and this stress could potentially result in increased family conflict. For example, parents' stress over accommodating obsessive-compulsive symptoms and child distress over refusal to accommodate are associated with increased family conflict (Peris et al., 2008). Thus, the presence of an anxious child in a family may contribute to increased stress and child-related conflict, which could in turn exacerbate child anxiety, especially if the child blames themselves.

Some children try to distract parents in conflict by diverting the attention onto themselves. Meta-analysis reveals a moderate effect between children's involvement in conflict and internalizing symptoms (Rhoades, 2008; see Table 20.1). While acting out is perhaps more characteristic of externalizing disorders, its use in the context of IPC has been associated with increases in later internalizing symptoms (Schermerhorn, Cummings, & Davies, 2005). While anxious children may not regularly display these behaviors, children that do employ behavioral dysregulation in the context of IPC develop internalizing symptoms. It is possible, but not yet considered, that anxious children attempt other distraction strategies (e.g., rituals, somatic complaints). Conversely, anxious children may be more likely to withdraw and anxiously ruminate on the potential threat the argument poses for the family unit (Riskind, 2005). Meta-analysis reveals a moderate association between avoidance and internalizing symptoms (Rhoades, 2008; see Table 20.1). While both avoidance and involvement are associated with internalizing symptoms, more specific research is needed to determine the frequency or impact of these strategies for anxious children.

Family processes and parenting. Interparental conflict is embedded in a larger family context so that other family factors influence the impact of IPC. Early researchers supposed that parental separation and divorce would result in internalizing difficulties. Yet, findings suggest that the effects of divorce are largely a by-product of IPC (for review, see Amato & Keith, 1991). For example, family conflict (combined couple, parent–child, and sibling) predicts anxiety symptoms 1 year later, but divorce does not (Noller, Feeney, Sheehan, Darlington, & Rogers, 2008). The relationship between family conflict and child anxiety is likely multifaceted and family factors, such as parental psychopathology, attachment, family cohesion, and relationship quality, are supported as process-level mediators (e.g., El-Sheikh & Elmore-Staton, 2004; Owen, Thompson, Shaffer, Jackson, & Kaslow, 2009). Illustrative of the interplay of multiple family factors, Shelton and Harold (2008) found that parent depressive

symptoms increased IPC, which was associated with the child perceiving rejection by their parent and development of internalizing symptoms. While multiple family factors likely influence the impact of IPC, we focus on parenting, social learning, and dysfunctional family roles or alliances, namely, parentification and triangulation.

Parenting and spillover effects. By far, the family factor that has received the most attention regarding IPC and internalizing symptoms is parenting. The "spillover" hypothesis suggests that disagreement between parents may result in dysfunction in the parent–child relationship, which is the proximal force related to child adjustment (see Erel & Burman, 1995). Interparental conflict may be emotionally draining and result in diminished ability or desire to engage with and parent children. For example, maternal emotional reactivity (as measured by cortisol) to IPC is subsequently related to suboptimal parenting (Sturge-Apple, Davies, Cicchetti, & Cummings, 2009). Meta-analysis (Erel & Burman, 1995; Krishnakumar & Buehler, 2000) provides support for the "spillover" hypothesis, finding a moderate association between IPC and parenting practices (see Table 20.1). Several studies find parenting "spillover" effects from IPC to internalizing symptoms (e.g., El-Sheikh & Elmore-Staton, 2004; Kaczynski, Lindahl, Malik, & Laurenceau, 2006). In fact, parenting practices important to anxiety development, namely, psychological control and intrusiveness, mediate the relationship between IPC and internalizing symptoms (e.g., Benson, Buehler, & Gerard, 2008; Buehler, Benson, & Gerard, 2006). Maritally distressed fathers, more than their female partners, may withdraw from children and parenting responsibilities, which is unfortunate as fathers seem to play a specialized role in pediatric anxiety by facilitating child autonomy (for a review, see Bögels & Phares, 2008).

Social learning. In addition to indirect effects through parenting, social learning, modeling, and information transfer can occur. Regarding modeling, the type of conflict resolution employed by parents might influence whether anxiety is experienced. For example, parents' use of withdrawn, or avoidant, strategies reported by daily diary predicted children's fear reactions, and children's distress was then associated with child internalizing symptoms (Cummings, Goeke-Morey, & Papp, 2003; Davies, Sturge-Apple, Winter, Cummings, & Farrell, 2006). If parents use avoidant or withdrawn strategies to cope with conflict in their marriage, children may model these strategies and generalize their use to other situations, particularly if a parent's avoidance of conflict results in de-escalation (Bussell et al., 1999; Crockenberg & Langrock, 2001). In fact, adolescents' conflict resolution style with siblings is predicted by methods employed by their parents during IPC, and modeling of parent's avoidance is associated with internalizing symptoms (Dadds, Atkinson, Turner, Blums, & Lendrich, 1999).

Parent's interpretations of, and comments during, IPC represent information transfer and could increase a child's threat perception and anxious reactions. For instance, parents' explanations absolving the child of blame may reduce fear, whereas explanations implicating the child may result in more distress (see Fincham & Osborne, 1993). Parents of anxious children, regardless of parental anxiety levels, make more catastrophizing comments and engage in less explanatory discussion of emotion during conversational tasks with their children (Moore, Whaley, & Sigman, 2004; Suveg et al., 2008; Whaley, Pinto, & Sigman, 1999) and may also do so during disagreements with their spouse. Moreover, anxious children may be especially tuned into their parent's interpretations of events (e.g., Creswell & O'Connor, 2006; Lester et al., 2010) and may integrate their parents' interpretation into their own understanding of the situation, in turn creating or perpetuating a fearful or anxious pattern of responding.

Parentification and triangulation. The "compensatory" hypothesis, an alternative to the "spillover" theory, posits that parents may become over-involved and particularly invested in the parent–child relationship due to their dissatisfaction with their marriage (Erel & Burman, 1995). While the compensatory hypothesis may not be pertinent for most families, it may hold relevance for a subset of more dysfunctional families or

families characterized by anxious parenting practices (e.g., intrusiveness). For example, internalizing symptoms have been related to feeling closer with one parent than the other (Grych, Raynor, & Fosco, 2004). Two dysfunctional patterns of parent–child relations, parentification and triangulation, may be considered compensatory processes relevant to pediatric anxiety. *Parentification*, defined as a child's felt responsibility to provide emotional support to their parent, has been linked to IPC and increased threat perceptions, as well as anxiety and overdependence, but mediation has not been tested (Mayseless & Scharf, 2009; Peris, Goeke-Morey, Cummings, & Emery, 2008). *Triangulation* refers to involving the child in IPC by forming a coalition with the child against the other parent. Triangulation has been found to mediate the impact of IPC on internalizing symptoms through children's self-blame, threat perception, and coping efficacy (Grych et al., 2004). However, one study found that triangulation was associated with a negative parent–child relationship and child maladjustment, but not internalizing symptoms (Kerig, 1995). While parentification and triangulation are less studied, future examination of compensatory processes linked to IPC in clinically anxious families may yield interesting findings.

Parent–Child Conflict

Somewhat surprisingly, relatively few studies have examined whether anxious children experience more or less conflict with their parents compared to their non-anxious peers. Parent–child conflict is thought to naturally increase during adolescence in response to adolescent autonomy seeking, especially if parents are reluctant to grant independence (for a review, see Steinberg, 2001). Parents' psychological autonomy granting protects against anxiety development (Gray & Steinberg, 1999). In fact, the converse parenting practice, psychological control or intrusiveness, is more characteristic of families of anxious adolescents (see McLeod et al., 2011). It is unclear how anxious adolescents respond to psychological control during the teen years. If anxious adolescents resist overcontrolling parents, they might experience higher levels of parent–adolescent conflict. However, anxious hesitation and doubting may debilitate anxious adolescents, such that they are less likely or unable to seek typical levels of autonomy. While anxious adolescents may or may not combat parental overcontrol, efforts aimed at independence are likely beneficial and could reduce anxiety, as increased autonomy seeking and independent decision-making over time improve emotional functioning (Qin, Pomerantz & Wang, 2009).

Evidence, albeit limited, suggests that parent–child conflict is related to, exacerbates, and has implications for the pathogenesis of child anxiety (e.g., Caples & Barrera, 2006; Krishnakumar, Buehler, & Barber, 2003). Supporting this connection, treatment reduction in child anxiety, whether or not conflict is specifically addressed, is associated with reduced parent–child conflict (Silverman, Kurtines, Jaccard, & Pina, 2009). Perhaps the most definitive evidence comes from a prospective longitudinal study using growth curve modeling to test a diathesis-stress model. In this study, Rueter, Scaramella, Wallace, & Conger (1999) found that over time parent–adolescent disagreements exacerbated anxiety symptoms and ultimately triggered the onset of an anxiety disorder. However, another longitudinal study found that parent–adolescent conflict was associated with life dissatisfaction, but not anxiety, in young adulthood (Overbeek, Stattin, Vermulst, Ha, & Engels, 2007). Thus, conflict with parents may exacerbate anxiety in predisposed adolescents, but not necessarily lead to increased anxiety for all adolescents. This highlights the importance of process-level research.

Parent–Child Conflict and Anxiety: Process-Level Research

While the literature has not advanced to examine many potential mechanisms related to parent–child conflict and anxiety, process-level variables would illuminate how parent–child conflict is linked to anxiety and which children are at

greatest risk for developing anxiety as a result of conflict with their parents. For example, relationship quality may be more predictive of internalizing symptoms than frequency of parent–adolescent conflict (Adams & Laursen, 2007). Similar to IPC, it is likely the child's processing and coping are important to consider. For example, parent–adolescent conflict is more predictive of externalizing symptoms unless the adolescent uses destructive conflict resolution strategies, which are then more predictive of internalizing symptoms (Branje, van Doorn, van der Valk, & Meeus, 2009). Avoidant coping strategies leave conflicts unresolved, which may induce or perpetuate anxious worry (e.g., Caples & Barrera, 2006; Riskind, 2005). An extreme form of conflict avoidance, namely, "exiting statements" (e.g., "I have told my parents I never want to talk with them again"), has been associated with internalizing symptoms (Wijsbroek, Hale, Van Doorn, Raaijmakers, & Meeus 2010). While little process-level research exists, relationship quality and conflict resolution style seem important, and conflict between parent and child is also related to IPC.

Interparental conflict and parent–child conflict intertwined. The parent–child relationship and the marital relationship are interdependent. In addition to undermined parenting ability, discord in the marriage may lead to increased parent–child conflict, especially if one parent attempts to form a coalition with the child against the other parent (e.g., Buehler & Gerard, 2002; Kerig, 1995; Grych et al., 2004). For example, parents are 50 % more likely to have negative interactions with their child the day after an IPC (Almeida, Wethington, & Chandler, 1999). This may be a result of parents feeling more irritable or emotionally drained (El-Sheikh et al., 2001). Parents may also not be supportive of each others' parenting decisions, and this lack of unity and social support may lead to more parent–child conflict. This may be especially true for families parenting anxious children, as parents of children with emotional and anxiety disorders report less social support, including less support from within the family (Lange et al., 2005).

Parent–child conflict may be either a full or partial mechanism explaining the relationship between IPC and child anxiety (e.g., Gerard et al., 2006; Krishnakumar et al., 2003). For example, Chung, Flook, and Fuligni (2009) employed a diary method and found that both IPC and parent–adolescent conflict were associated with daily increases in anxious and depressed symptoms. Moreover, parent–adolescent conflict partially mediated the relationship between IPC and internalizing symptoms. Thus, IPC may be tied to anxiety directly and indirectly as a function of conflict between parent and adolescent. Additionally, the combination of both stressors, interparental and parent–adolescent conflict, might serve to increase risk for anxiety. Parent–child conflict accounts for additional variance in child-report of internalizing symptoms after accounting for IPC (El-Sheikh & Elmore-Staton, 2004). Similarly, the combination of IPC and parent hostility towards the child represents a cumulative risk factor for boys' internalizing symptoms (Gordis et al., 2001).

Role of Siblings

Sibling conflict in the context of child anxiety has received little attention. In one cross-sectional study, anxious children were found to engage in more conflict with their siblings than non-anxious peers (Fox, Barrett, & Shortt, 2002). As a potential explanation, mothers of anxious children are reported to be much more involved, controlling, and protective of their anxious child, as compared to their other children (Barrett, Fox, & Farrell, 2005; Hudson & Rapee, 2002). This differential treatment could lead the sibling to believe he or she is being treated unfairly due to the anxious child's need for attention. As a result, the sibling could develop feelings of jealousy and resentment towards the anxious child, and conflict may develop between the two children.

In a longitudinal study of the influence of sibling relationships, sibling conflict at baseline predicted childhood anxiety 2 years later (Stocker, Burwell, & Briggs, 2002). Sibling conflict accounted for a unique and significant proportion

of the variance in child anxiety, even after controlling for maternal hostility and IPC. This important preliminary finding suggests that sibling conflict could be more influential, than interparental and parent–child conflict, in predicting later anxiety symptoms. On the other hand, some studies have shown that siblings can serve a positive function for children with anxiety. One study found that a supportive sibling relationship protected a child from developing adjustment problems that may result from IPC (Jenkins & Smith, 1990). Another study (Lockwood, Gaylord, Kitzmann, & Cohen, 2002) found that siblings may act as a buffer between family stress and peer rejection, which is also a major source of anxiety for children and adolescents (Storch, Masia-Warner, Crisp, & Klein, 2005). This evidence suggests that a child with anxiety may benefit from having a sibling to provide social support in times of stress, perhaps after exposure to interparental or parent–child conflict.

Effects Beyond Adolescence

While the focus of this chapter has been on family conflict and childhood, this is not meant to suggest family conflict only relates to children's anxiety. As previously discussed, parenting an anxious child can lead to conflict within the couple relationship. There is a vast literature, beyond the scope of this chapter, on the detrimental impact of relationship conflict, domestic violence, and divorce on each partner's health, mood, life satisfaction, and anxiety (see Howard, Trevillion, & Agnew-Davies, 2010; Kiecolt-Glaser & Newton, 2001; Robertiello, 2006; Whisman & Uebelacker, 2006). Further, family conflict experienced as a child can have long-lasting effects present in adulthood. For example, women with panic disorder retrospectively report more conflicted family environments during childhood than non-anxious adult women (Laraia, Stuart, Frye, Lydiard, & Ballenger, 1994). Similarly, higher stress reactivity is seen in adult men who experienced greater childhood conflict, and this reactivity then predicts adult onset of mood and anxiety disorders (McLaughlin et al., 2010). Lastly, both anxiety and dysfunctional family processes can be "inherited" (transmitted, modeled, or replicated) from family of origin to the nuclear family (Dadds et al., 1999; Hettema et al., 2001) resulting in a cycle of anxiety, distress, and dysfunction. In fact, approximately 60 % of men who perpetuate domestic violence experienced familial violence in their childhood (Delsol & Margolin, 2004).

Summary, Future Directions, and Implications

While the implications of family conflict for pediatric anxiety are not yet fully known, evidence supports the likely possibility of a bidirectional relationship between these two factors. Meta-analysis and longitudinal studies support an association between IPC and internalizing symptoms, but specificity to anxiety is less certain as most researchers combine anxiety and depression. As stress and caregiver burden seem to be more substantial when parenting an anxious child (Storch et al., 2009), reciprocal effects related to child-centered conflict may be present. Increased attention should be given to social learning models, including parents' modeling of avoidant coping and information transfer during conflict. Parent–child and sibling conflict have received less empirical attention, but connections and relevance to anxiety are suggested. As such, the field is in need of more rigorous process-level design and longitudinal examination of parent–child conflict and pediatric anxiety, as well as the role of siblings. For example, it remains to be seen whether anxious adolescents combat, or cede to, parental over control or whether specific conflict tactics impact anxiety course. As the sibling relationship can function as risk or protective factor for child anxiety, specific dyadic relationship qualities might clarify findings. Finally, more studies with clinical samples would be beneficial for understanding the distinct relationships between family factors and individual anxiety disorders. It is certainly possible that some anxiety disorders are more influential for, and affected by, family conflict than others or that

disorder-specific family conflict processes exist. As examples, a family's lack of accommodation of obsessive-compulsive symptoms may result in parent–adolescent conflict, and separation anxiety may be perpetuated if a child fears divorce after overhearing parents arguing.

Regardless of causal potential, as conflict occurs in all families, it is also present in many families with anxious children. As such, it is important to consider *how* IPC affects an anxious child and whether it might impede treatment or amelioration of anxiety symptoms. There is not definitive evidence that including parents or siblings in treatment of pediatric anxiety is warranted (see Lewin, 2011). However, as illustrated in the case study presented below, family intervention may be more appropriate, or even necessary, when the family also presents with conflict considering that family dysfunction has been associated with poorer anxiety treatment outcomes (e.g., Crawford & Manassis, 2001; Merlo, Lehmkuhl, Geffken, & Storch, 2009). Alternatively, there is some evidence that amelioration of child anxiety symptoms will reduce family conflict and dysfunction (e.g., Silverman et al., 2009; Storch et al., 2007), due to a bidirectional link between child impairment and family functioning. Thus, a primary consideration may be determination of family's willingness and desire to concurrently address family conflict.

Case Study

Hector (a pseudonym) was a bright, creative, and humorous 9-year-old Latino male exhibiting multiple symptoms of anxiety including excessive worry (e.g., concern about "flunking" a standardized test that he passes every year, fearing consequences of presenting an expired coupon), catastrophic thinking (e.g., fear someone would be hit by car if they walked too close to the street), perseveration (e.g., "not letting things go"), trouble falling asleep, and somatic complaints (e.g., stomach aches). Hector's mother sought evaluation and treatment for her son due to school avoidance (e.g., frequent trips to the nurse, lower attendance) and emotionality at school (e.g., breathing irregularly, "freezing up," and crying when called on in class). Hector was otherwise typically developing, successful academically, and socially adept. Hector lived at home with his mother, father, brother (7 years), and sister (3 years). According to this information obtained during the assessment process, Hector met diagnostic criteria for generalized anxiety disorder (see American Psychiatric Association, 2000).

Considering best practice, the treating clinician implemented an evidence-based treatment approach for managing symptoms of anxiety. Using a cognitive behavioral model, and the "Coping Cat" manual (Kendall & Hedtke, 2006) as reference, Hector was taught affective education, awareness of anxious thought patterns, recognition/reduction of worry, and relaxation training. Relaxation training was coupled with a biofeedback computer "game" in which "points" were awarded coupled with visual stimuli (e.g., a gray and white picture of a rainbow slowly colored in) for a relaxed demeanor (slow and steady heart rate). Hector responded positively and quickly to treatment. After nine sessions, it was determined that Hector was ready to terminate therapy. This readiness was based on Hector experiencing remission of many anxiety symptoms (including sleep difficulties and somatic complaints), as well as demonstrating competency in using coping tools to reduce anxiety, replacing catastrophic thinking with more adaptive appraisals of situations, and exposure to age-appropriate risk-taking behaviors with minimal anxiety (e.g., amusement park rides previously avoided due to fear). However, 5 months after termination, Hector's parents initiated services again at Hector's request.

Anxiety Treatment in the Context of Interparental Conflict

Upon returning to treatment, Hector reported he was experiencing school-related "stress". Although he was reporting somatic complaints (e.g., stomachaches) and emotional distress, many of Hector's anxious symptoms (e.g., sleep difficulties) were in remission, and most coping

strategies (e.g., relaxation, cognitive restructuring) were retained. In fact, since leaving therapy, Hector made a successful transition to a new school (feared event reported during initial treatment) without debilitating anxiety, sleep difficulties, or somatic complaints. Despite these gains, Hector's experience of distress was concerning enough for him to seek treatment. At this point in time, Hector also reported frustration with his younger brother and frequent sibling conflict over minor daily events (e.g., sharing). Lastly, Hector's parents reported marriage difficulties, which ultimately resulted in Hector's parents planning a temporary separation, which was achieved by one parent taking a job that required an extended period overseas. The parent's absence was explained to the children in terms of a career opportunity rather than explaining the marriage difficulties.

While the domestic disputes were not known to be physical in nature, or even intense, Hector, who was hypervigilant to potential danger (as anxious children tend to be), had observed parental disagreements, less parental cohesion, and occasional changes in his parents' sleeping locations. Hector admitted fearing a potential divorce and that some "stress" was related to his parents' arguments. However, the degree to which he attributed his stress to his family's circumstances was minimal. Similarly, both parents were most comfortable not directly addressing the IPC and were uncertain how much or what information to share with their children.

As family conflict, parental separation, and potential divorce complicated this case and likely contributed to exacerbation in Hector's anxiety symptoms, individual therapy focused solely on addressing child anxiety was insufficient. Based on evaluation of family functioning (parents' willingness and motivation to work together for the sake of their children) and Hector's symptom presentation, treatment included extensive family involvement (individual, sibling, parent [separate and joint], and family sessions), psychoeducation regarding the interplay between family conflict and anxiety, and a strength-based focus on coping ability. Due to the unfolding process of parental separation and divorce, treatment took place over the course of approximately 10 months (20 sessions) with maintenance sessions (e.g., 2–4 weeks between sessions) as symptoms improved and family circumstances stabilized. Unlike traditional family therapy, Hector remained the identified client and treatment focused on his functioning and processing of IPC. While his siblings' coping was assessed and discussed, because they did not demonstrate maladaptive coping or functional impairment, they were not primary participants in treatment. Marital discord (outside of effects on children) was not directly addressed, but the treating clinician provided a referral for couples counseling.

In collaboration with the family, the following treatment objectives were established: (1) monitor Hector's anxiety and reinforce learned anxiety reduction techniques, (2) bolster Hector's coping skills and self-efficacy beliefs, (3) ensure communication and maintain a positive relationship with the parent who was away, and (4) decrease sibling conflict and improve the sibling bond. Lastly, when a decision for divorce was made, treatment included helping parents communicate this decision and co-parent their children through, and after, this transition.

As Hector previously responded well to cognitive behavioral methods, a similar approach to individual sessions was taken to monitor symptoms, maintain previous treatment gains, and bolster anxiety reduction skills. Because Hector underestimated his coping ability and tended towards "worse case" interpretations or perceptions of threat, a positive strength-based component (e.g., identifying role models, defining successful coping, exploring own strengths, parental praise of coping efforts) was included to improve coping self-efficacy and encourage positive expectations regarding the future. Hector also created a "coping cheat sheet" listing ways to cope with IPC (e.g., remove self from situation, journal, seek support) and then recorded negative events and coping attempts (and effectiveness of strategy selected) in daily logs. Problem-solving, relationship maintenance,

and communication (e.g., daily feelings journal including things to share with the absent parent) exercises were used to ensure the maintenance of a strong relationship with the absent parent. Sibling conflict was addressed with conflict resolution training and anger management skill building.

As Hector's anxiety likely contributed to his initial inclination to avoid addressing the IPC, which in turn likely exacerbated his anxiety, it was important to facilitate open communication (e.g., encourage questions, make explicit plans on how to communicate distress and concerns to parents), normalize the experience of divorce (e.g., child education), anticipate difficulties (e.g., living in two homes) and problem-solve solutions (e.g., determining times when children wanted both parents present), and identify extra-familial sources of social support (e.g., friends with divorced parents, school counselors).

Termination

Hector's symptoms diminished with the decrease in IPC during the parental absence. Upon disclosure of plans to divorce, Hector revealed how attuned to the parental relationship he had been (e.g., "I guessed it [the topic of divorce] right away"). According to parent report, Hector coped with the news "better than expected". By directly addressing family conflict, the children were able to process, rather than avoid, the IPC, and Hector displayed age and situation appropriate worry, but not debilitating anxiety. Moreover, with the sibling relations improved, Hector maturely volunteered himself as social support for his younger siblings. In determining readiness for termination, Hector examined coping logs and graphed instances of negative moods and successful coping until he reported feeling self-efficacious. Hector was able to terminate treatment before his parents' divorce was finalized and reported feeling happy and self-confident in his abilities. With this individualized variant of treatment, Hector evidenced commendable coping, despite anxious tendencies, during an extremely difficult family transition.

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Assessment and Treatment of Comorbid Anorexia Nervosa and Obsessive–Compulsive Disorder

21

Adam B. Lewin, Jessie Menzel, and Michael Strober

In this chapter we describe the complications that ensue from the phenotypic overlap between anorexia nervosa (AN) and obsessive–compulsive disorder (OCD) and consider how to approach the evaluation and treatment of their comorbidity; to aid the discussion we present two illustrative case examples. The theoretical significance of this association lies in recent speculation that the distinguishing phenotypic characteristics of AN, phobic avoidance of normal body weight and grossly distorted appraisal of bodily image, express a broad array of heritable traits, including anxiety and fear proneness and disturbances in reward and habit circuitry, also linked to the pathophysiology and clinical features of OCD. Empirical support for the notion that AN and OCD overlap is strong, based on evidence of, first, a strikingly high lifetime comorbidity of AN with anxiety disorders, OCD in particular (Godart, Flament, Perdereau, & Jeammet, 2002; Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). Second, the common emergence of OCD and anxiety phenotypes prior to onset of weight concerns and dieting (Bulik, Sullivan, Fear, & Joyce, 1997) and persistence of anxiety states following weight restoration (Pollice, Kaye, Greeno, & Weltzin, 1997) further support of the OCD-AN link. Third, strong familial aggregation of OCD and multiple anxiety phenotypes, as well as compulsive personality, in AN (Strober, Freeman, Lampert, & Diamond, 2007) and evidence from twin studies of a common genetic architecture influencing liability to both anxiety and eating disorder (Keel, Klump, Miller, McGue, & Iacono, 2005; Silberg & Bulik, 2005) bridge these two impairing neuropsychiatric syndromes. Commonalities between the diagnostic features of AN and the behavioral phenomenology of anxiety states and obsessional illness are notable. They include anticipatory fear, hypervigilance, phobic avoidance, the incorrigible resistance of dietary restriction to reason or logic, and compulsiveness of weight checking, dieting, exercise, and counting of calories—features similar in character to the worry-driven, compulsive error checking and inability to inhibit perseveration of compensatory goal-directed action characteristic of OCD. Notably, despite these associations, AN is generally not considered an obsessive–compulsive spectrum disorder, per se (DSM-IV Workgroup; Hollander, Braun, & Simeon, 2008).

The common association of OCD and AN complicates their presentation, prognosis, assessment, and the interventions required for these often chronic, treatment refractory syndromes.

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In this regard, the presence of comorbid OCD symptoms in individuals with eating disorders confers greater severity and persistence of eating disorder symptoms (Jimenez-Murcia et al., 2007; Milos, Spindler, Ruggiero, Klaghofer, & Schnyder, 2002) as well as increased overall levels of associated anxiety and depression (Sallet et al., 2010).

Anorexia Nervosa (APA, 2000)

Anorexia nervosa is an illness of undetermined etiology whose onset, typically during adolescence, confers high risk of medical and psychological morbidity, premature early death, and extreme economic burden. The illness is characterized not simply by an avoidance of normal body weight (at or less than 85% of normal or ideal body weight for age or height) but rather an inexplicable indifference to the emaciation that results and steadfast resistance to its correction because of the intense fear of being "fat." Accompanying these features is an apparent disturbance in how weight is perceived and judged (believing oneself to be overweight even when emaciated), an undue influence of weight or shape on self-worth, and amenorrhea in postmenarchal females (APA, 2000). The full syndromic presentation of AN is not common; its lifetime occurrence estimated to be under 1% (Hudson, Hiripi, Pope, & Kessler, 2007), occurring preponderantly in females. However, the public health significance of AN is significant, as it is among the most lethal of all psychiatric disorders (Hudson et al.) with a mortality rate of approximately 5% per decade (Sullivan, 1995).

Phenomenological Overlap (Palmer & Jones, 1939)

The overlap in cognitive, affective, and behavioral aspects of OCD and AN has long been noted (Altman & Shankman, 2009). Palmer and Jones (1939) noted that rigidity, compulsive perfectionism, and obsessional personality were inherent to AN. Both disorders are distinguished by perseverative thoughts centered on fear- or anxiety-inducing stimuli (Altman & Shankman, 2009) with compulsive acts aimed at reducing alarm and distress occasioned by these intrusive ideas (Buree, Papgeorgis, & Hare, 1990; Rachman & Hodgson, 1980; Tynes, White, & Steketee, 1990). While the content of the most pervading aversive thoughts differs across the two syndromes, the confluence of fear and alarm and behavioral acts reinforced by their avoidance-related function figure prominently in each. Even when classic obsessions and compulsions are lacking in persons with AN, overlap with the phenomenology of OCD is striking—impaired inhibition of intrusive ideas, rigid habit behaviors that resist change, and avoidance of the fear instantiated by ingestion of food. Much like the patient with OCD, persons with AN experience these preoccupying thoughts as (at times) deeply upsetting, difficult to inhibit, and interpersonally disruptive. Accordingly, adherence to abnormally extreme rules governing food choices, frequent weighing, purging, and body checking have been likened to compulsive actions which function to reduce anxiety related to eating or weight gain (Kaye, 2004). Potentially a contrast to obsessions in OCD is the degree perfectionism and rigid standards for oneself can appear ego-syntonic in AN (vs. an ego-dystonic presentation, at least typically, in OCD). Anorexia nervosa is likely to have a strong temperamental foundation as perfectionism accompanied by intolerance for personal imperfections, unrelenting self-standards, and harm avoidance which often appear early in development (Hildebrandt, Bacow, Markella, & Loeb, 2012; Strober, 2004, 2010).

Prevalence

Evidence from comorbidity studies attests to the strength of the OCD and AN association. In clinical populations, the estimated co-occurrence ranges widely, with figures ranging from 10 to 60% (Godart et al., 2002; Halmi et al., 2003). In mixed populations of adolescents and adults with AN, the lifetime prevalence of OCD ranges from 24.3 to 35% with a point prevalence of 16.8% in adolescents (Salbach-Andrae, Lenze, Simmendinger, Klinkowski, Lehnkuhl, &

Pfeiffer, 2008) to 17.8% in older adolescents/adults (Godart et al., 2003). Conversely, in clinical samples of OCD, lifetime comorbidity with eating disorders are lower, in the range of 2.4–13% for AN but as high 18% when including subthreshold AN (du Toit, van Kradenburg, Niehaus, & Stein, 2001; LaSalle et al., 2004; Rubenstein, Pigott, L'Heureux, Hill, & Murphy, 1992; Sallet et al., 2010). For a detailed review of comorbidity studies, see Swinbourne and Touyz (2007).

Further support for phenotypic overlap is evidence of a greater number of non-eating disordered obsessive thoughts and compulsions in individuals with eating disorders compared to either psychiatric or healthy controls (Cassidy, Allsopp, & Williams, 1999; Claes, Vandereycken, & Vertommen, 2002; Halmi et al., 2003; Hirani, Serpell, Willoughby, Neiderman, & Lask, 2010; Matsunaga et al., 1999; Roberts, 2008; Sassaroli et al., 2008; Strober, 1980). For example, contamination, aggressive and somatic obsessions, as well as checking and ordering/arranging rituals are common in youth with AN (Hirani et al., 2010). Notably, some evidence also suggests that obsessional symptoms decrease with weight restoration in AN patients (Ehrlich et al., 2010) in accord with the known association between obsessional behavior and severe malnutrition (e.g., see Keys, Brozek, Henschel, Mickelson, & Taylor, 1950).

Etiology and Genetic Studies

Detailed considerations of a potential shared pathophysiology in eating disorders and OCD have been offered (Stein & Lochner, 2008). Considering the typical developmental trajectory and chronology of disease onset within this comorbidity, it can be argued that OCD may represent a risk factor for the later emergence of AN (Bulik et al., 2003; Kaye et al., 2004) as the illness precedes onset of AN in upwards of two-thirds of comorbid patients (Bulik et al., 2003; Godart, Flament, Lecrubier, & Jeammet, 2000; Kaye et al., 2004), early age of onset of OCD has been linked to later eating disorders (Fahy, Osacar, & Marks, 1993), and symptoms of OCD typically persist after weight recovery (Morgan, Wolfe, Metzger, & Jimerson, 2007; von Ranson, Kaye, Weltzin, Rao, & Matsunaga, 1999; Wentz, Gillberg, Anckarsater, Gillberg, & Rastam, 2009).

Familial studies supporting a shared underpinning of AN and OCD have shown that the lifetime prevalence of OCD is elevated among first-degree relatives of AN probands compared to relatives of non-AN controls (Bellodi et al., 2001; Lilenfeld et al., 1997, 1998; Stein et al., 1999; Strober et al., 2007) though there is also evidence to the contrary (Bienvenu et al., 2000; Nestadt et al., 2000). Twin studies of anxiety disorders and eating disorders suggest shared genetic susceptibility (Silberg & Bulik, 2005). Notably, Keel et al. (2005) found that within monozygotic twin pairs discordant for eating disorders, the prevalence of anxiety in the non-eating disordered twins was greater than in controls; conversely, for monozygotic twin pairs discordant for anxiety disorder, the non-anxiety disordered twins were more likely to express eating pathology than were control subjects (Keel et al., 2005). While these twin studies did not examine OCD specifically, a convergence of evidence on this area suggests at least some shared liability between OCD and AN.

Putative Neurobiological Mechanisms

Anorexia nervosa and OCD have been linked to common abnormalities in neurobiological substrates involving, in particular, serotonergic (5-HT) systems that regulate fear learning and extinction, habit actions, and frontal modulation of limbic emotion-generating circuits (Kaye, 2008). The attention on 5-HT function has been based on the role of this broadly distributed system in synaptogenesis, appetite regulation, and impulse regulation. In eating disorders, abnormalities in 5-HT function are not limited to acute illness alone (Jimerson et al., 1997) but are found post-morbidly as well (Frank et al., 2002; Kaye, Fudge, & Paulus, 2009; Kaye, Wagner, Fudge, & Paulus, 2011).

Evidence linking defects in 5-HT systems to OCD is better developed and has been well reviewed (Goodman, McDougle, & Price, 1992; Stein, 2000; Westenberg, Fineberg, & Denys, 2007). Notably, a wide range of studies have reported increased 5-HT metabolic activity and decreased levels of 5-HT transporter protein in OCD patients compared to non-patients (Arora & Meltzer, 1991; Insel, Mueller, Alterman, Linnoila, & Murphy, 1985; Marazziti et al., 1997; Marazziti, Hollander, Lensi, Ravagli, & Cassano, 1992), Reductions in cerebrospinal fluid levels of 5-HT metabolites have been correlated with decreases in obsessive–compulsive symptom severity (Thoren, Asberg, Cronholm, Jornestedt, & Traskman, 1980), and evidence exists for pharmacological treatment studies which consistently find that serotonin reuptake inhibitors (SRIs) produce positive treatment outcomes in patients with OCD (Abramowitz, Taylor, & McKay, 2009; Watson & Rees, 2008).

A Putative Model of Etiological Overlap

From a young age, persons with AN are uncompromisingly rigid and compulsive, show exaggerated worry about inconsequential mistakes, are distressed by the anticipation of change, avoid novelty, and their life decisions are governed by the avoidance of even the slightest possibility of threat (Strober, 2010). Strober and colleagues recently proposed a neurodevelopmental model of AN emphasizing a heritable predisposition towards obsessional anxiety- and stress-engendered impairment of circuitry involved in fear learning and the regulation of affective arousal as core elements (Strober, 2004; Strober et al., 2007). These neuroatypicalities (also associated with obsessional illness) are offered as a mechanistic explanation for the sudden onset of perceived threat and avoidance of weight change that emerge in concert with physical, hormonal, and social/developmental changes that accompany pubertal maturation. Preclinical and clinical studies are supportive of this speculative model, showing that chronic stress, highly associated with anxious states generally, alters neural morphology mediating fear learning, emotional memory consolidation, and the regulation of emotion (Duman, Malberg, & Thome, 1999; Kaufman, Plotsky, Nemeroff, & Charney, 2000; Sapolsky, 2003; Vyas, Jadhav, & Chattarji, 2006), thus suggesting that individuals prone to early onset of anxious states may acquire a hyperresponsiveness to signals of novelty and become impaired in the ability to discriminate safe from threatening environments well in advance of the emergence of puberty. These characteristics, also prominent features of OCD, may thus confer risk for the eventual development of AN (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003; Kaye et al., 2004; Olatunji, Tart, Shewmaker, Wall, & Smits, 2010) and are consistent with the antecedence of anxiety and OCD in persons with AN (Hsu, Kaye, & Weltzin, 1993; Kaye et al., 2004; Kaye, Weltzin, & Hsu, 1993).

Assessment and Treatment

The phenotypic overlap between AN and OCD has important assessment and treatment implications. In the following sections, we offer some considerations for how to view differential diagnosis as well and how to approach treatment options.

Differential Diagnosis

As anxiety and compensatory behavior are central psychological features of AN and OCD alike, dietary restriction, ritualistic eating patterns, and eating-induced fear can be seen in both disorders. Just as the patient with AN steadfastly restricts caloric intake due to weight phobia, OCD can present as an avoidance of certain foods based on fears of contamination or disgust. As such, careful ascertainment of the core rationale for ritualistic behaviors and the underlying worry is fundamental to accurate differentiation as we have seen classic OCD accompanied by low body

weight due to food contamination fears. Generally, obsessions and rituals experienced in OCD tend to be ego-dystonic, while in eating disorders they are largely ego-syntonic (Bastiani et al., 1996). Also in contrast to patients with OCD, obsessive thoughts in the early stages of AN are not commonly experienced as intrusive or inappropriate, and attempts to neutralize/suppress/ignore intrusive thoughts are rare (Olatunji et al., 2010). Thus, insight can assist in the differential diagnosis as individuals with AN do not experience their obsessions surrounding food or the related compulsions, such as excessive exercise, calorie counting, or ritualistic eating, to be irrational (Halmi et al., 2003). By contrast, most individuals with OCD experience their obsessions and compulsions as odd, irrational, and intrusive and easily characterize them in these terms.

Descriptively and clinically, differences between the two syndromes have been noted and may assist in their separation. Specifically, AN has been associated with greater perceived ineffectiveness and poorer interoceptive awareness (Jimenez-Murcia et al., 2007), whereas contamination fears, sexual obsessions, and cleaning compulsions tend to be more common in pure OCD (Bastiani et al., 1996; Halmi et al., 2003).

Lastly, OCD has an earlier age at onset than AN and other eating disorders (Kaye et al., 2004) underscoring the importance of careful assessment of the temporal chronology of symptom development in relationship to significant weight loss bearing in mind the effect of malnutrition on obsessional thought (Keys et al., 1950; Pollice et al., 1997). Following these timelines is of critical importance in identifying true comorbidity bearing in mind that comorbid OCD usually precedes AN in upwards of 65% of cases (Speranza et al., 2001).

Treatment of the Comorbid Patient

Treatments for anorexia nervosa. The treatment of AN consists of a blend of treatment modalities (individual, group, family) and approaches (interpersonal, CBT, psychodynamic) requiring a comprehensive and multidisciplinary approach (Kaplan & Howlett, 2010; Keel & McCormick, 2010). Nevertheless, there is no single, empirically well-supported approach at this time. Pharmacological interventions have also been widely used, but without robust findings. Two studies examined the use of fluoxetine in treating AN and neither found significant effects on weight gain or eating disorder psychopathology relative to placebo (Attia, Haiman, Walsh, & Flater, 1998). Overall, AN is largely considered resistant to pharmacological interventions (Kaplan & Howlett, 2010) and pharmacotherapy in the absence of psychotherapy is not recommended for AN (Bulik, Berkman, Brownley, Sedway, & Lohr, 2007). Unfortunately, no studies from a dismantling perspective have been conducted to determine which interventions account for the greatest variance in improvement nor is there a set of guidelines for sequencing treatment by different modalities.

Neither is there strong empirical support for the broad efficacy of psychosocial interventions for AN. Psychological treatments that have been studied include various family therapies, cognitive-behavioral therapy (CBT), interpersonal therapy (IPT), and psychodynamic therapy (Bulik et al., 2007). Despite some support for CBT in maintaining normal body weight after discharge from a CBT-based inpatient treatment setting (Pike, Walsh, Vitousek, Wilson, & Bauer, 2003), one acute treatment trial (McIntosh et al., 2005) that compared CBT, interpersonal psychotherapy, and supportive psychotherapy found that after a course of 20 visits, women randomized to the nonspecific supportive psychotherapy arm fared better than those assigned to either CBT or IPT. A recent long-term follow-up (mean 6.7 years) of this sample found no differences in severity of illness across the three conditions (Carter et al., 2011). Unfortunately, few well-designed and adequately powered randomized controlled treatment studies of AN exist (see Bulik et al., 2007 for a review). The strongest support is for a behavioral family therapy approach for adolescents, which emphasizes parent control of renutrition during an initial phase of treatment (Eisler et al., 2000).

During this acute phase, health stabilization is the primary outcome with hospitalization generally advocated in cases of more extreme malnutrition (e.g., BMI < 15) or when a course of outpatient care fails to achieve weight gain (Fairburn, 2008). Anorexia nervosa is associated with a range of physical health complications such as arrhythmia, electrolyte disturbance, loss of menses, dehydration, and bone disease, and as malnutrition progresses the patient's mental state worsens, diminishing the patient's capacity to benefit from psychotherapy. Accordingly, psychotherapy has limited chances of succeeding when the body is in a starved state (Bulik et al., 2007), and frequent physician assessments and nutritional rehabilitation are necessarily central to improving health and achieving desired treatment outcomes (APA, 2006; Pomeroy, 2004).

Treatments for obsessive–compulsive disorder. There are two primary approaches for treating OCD: CBT with exposure and response prevention (ERP) and pharmacotherapy with SRIs. A discussion of these approaches is found elsewhere in this volume and in several recent reviews (Abramowitz et al., 2009; Lewin & Piacentini, 2009).

Treatment of eating disorders using OCD behavioral therapies. Unfortunately, no treatment studies have targeted individuals with comorbid OCD and AN. The need for this line of research is highlighted in a recent cross-sectional analysis of 508 inpatients with an eating disorder (half of the sample also had OCD) that suggests a bidirectional and reciprocal relationship between the symptom complexes (Olatunji et al., 2010) wherein change in one mediated improvement in the other. However, isolated reports have examined the effects of ERP techniques where the goal is to reduce anxiety associated with eating certain foods or reduce binge eating/purging. An initial model of ERP applied by Rosen and Leitenberg (1982) to bulimia nervosa had patients eat "forbidden" foods until they felt the urge to vomit while being prevented from doing so (Rosen & Leitenberg). A slightly different version of the technique involved preventing binge eating by exposing patients to "forbidden" foods through licking, touching, smelling, and ingesting only small amounts (Kennedy, Katz, Neitzert, Ralevski, & Mendlowitz, 1995).

Several studies have evaluated the ERP model by exposing patients to binge eating but preventing the compensatory vomiting behavior. Results have shown a reduction in binge and purge episodes (Gray & Hoage, 1990; Kennedy et al., 1995; Rossiter & Wilson, 1985), but the durability of these effects has never been demonstrated and two studies that compared the effectiveness of CBT with and without ERP found that the addition of ERP did little to enhance the effects of CBT alone (Bulik, Sullivan, Carter, McIntosh, & Joyce, 1998; Wilson, Eldredge, Smith, & Niles, 1991). Nevertheless, experts have argued that ERP is inherent to therapies applied to AN given the following: (1) the marked role of anxiety/avoidance in the pathogenesis and presentation of AN (Hildebrandt et al., 2012) and (2) fear-learning-based models consistent with the maintenance of AN that resemble the presentation of OCD (Steinglass & Walsh, 2006; Sysko, Walsh, Schebendach, & Wilson, 2005). This suggests that exposure and habituation to food-, eating-, and weight-related cues promote change via negative reinforcement of avoidance behaviors and fear extinction. Weekly weight checks, refeeding with a variety of foods, and naturalistic exposures (where precise caloric and fat values are not available) mirror the ERP strategies employed for OCD treatment.

Treatment of comorbid OCD and eating disorders. In treating the comorbid patient, weight must first be restored to an adequate level for medical stability and to maximize the patient's ability to both comprehend and participate in psychotherapy. Fairburn (2008) recommends that if comorbid OCD is present, the clinically more severe disorder should take precedence in prescribing the treatment approach. However, it is sometimes feasible to pursue both treatments simultaneously as psychotherapy for AN can aid the flexibility required for ERP with OCD symptoms. As discussed above, with less severely ill patients with AN, ERP can be applied to aspects of the eating pathology (Fairburn, 2008). While treatment of AN with ERP alone is not indicated (Shapiro et al., 2007), ERP can logically be

combined with cognitive therapies to treat a range of specific intrusive obsessions and compulsions (McCabe & Boivin, 2008), including obsessions concerning risky foods and the subsequent use of compensatory behaviors such as compulsive exercise, laxative use, and purging. ERP can also be used to address obsessions of weight or shape, compulsive weighing, body checking, avoidance of mirrors and revealing clothing, food avoidance, and dietary restriction.

Still, there are caveats for psychotherapy with the comorbid AN/OCD patient to keep in mind. First, when challenging the core psychopathology of AN—fear of gaining weight/fatness and the undue influence of weight/shape on self-evaluation—use of cognitive techniques is probably essential for achieving and maintaining good therapeutic outcomes (Shapiro et al., 2007). Second, it is important to take into account the age of the individual. While the addition of CBT techniques may be essential for adults and older adolescents, involvement of the family is essential in treating children and younger adolescents (Lewin, 2011; Lock, 2001; Lock & Le Grange, 2001).

We now present two cases that highlight the importance of differential diagnosis and treatment selection. In the case of Kendra, OCD symptoms are misdiagnosed as AN but subsequently respond well to ERP. In the case of Zoe, who presents with severe comorbid AN and OCD, weight restoration is the primary goal. Zoe remains treatment resistant and her history is noteworthy for several inpatient hospitalizations. Once her weight is stabilized, she is better able to participate in ERP for OCD, but dramatic character changes are unlikely.

Case Illustrations

Case 1. Kendra was a 10-year-old Caucasian female who presented for treatment of obsessive–compulsive symptoms and presumed eating disorder. The only daughter of divorced parents, she lived with her mother and grandmother, enjoyed sports, and was very diligent about her school work. At age 8 years, Kendra developed abnormal eating behaviors following a strep infection, which included tapping foods, limiting and repeating food choices (she ate only berries at breakfast), eating only half of food portions, and refusing to watch food being cleared from the table. Her abnormal eating behaviors were accompanied by other rigidities, including restricting the clothes she would wear, the development of bedtime routines, following a rigid daily schedule, and refusal to be touched by any sticky substances. Significant weight loss occurred over the period of a year, yet Kendra denied fear of weight gain, disturbance in her perception of weight and shape, or a desire to maintain her low body weight. To the contrary, she stated she wished she could eat and knew that she needed to eat more in order to be healthy, yet she was unable to explain why she was engaging in these behaviors, only that something in her head told her that she must and that she needed to eat less.

In spite of her statements, Kendra was initially diagnosed with, and treated for, AN. However, she stated that what her treatment team told her made little sense as the characteristics of people with this illness did not apply to her. After 4 weeks of treatment for an eating disorder, Kendra underwent another psychiatric evaluation which resulted in a primary diagnosis of OCD.

From this point on, Kendra's treatment included a course of pharmacotherapy with the SSRI fluoxetine, dietary support for weight gain, and behavioral therapy initially focused on her eating behaviors. Kendra's tapping and eating in halves responded rapidly to behavior therapy, but her other eating-related oddities continued. As a result, she underwent an intensive, 3-week course of CBT with ERP targeting the entire range of her obsessions and compulsions. Exposure practices included watching food being cleared from the table, participating in clearing food from the table, "contaminating" hands with leftover food from meals and food from the table, and covering hands in sticky substances. An essential part of this phase was preventing Kendra from engaging in her usual avoidance responses, such as fleeing the table and being able to wash her hands. The ERP therapy proved effective in helping her reduce her anxiety and she resumed normal

eating behaviors and was successful in restoring and maintaining a normal body weight.

Case 2. Zoe was a 27-year-old Caucasian woman with a bachelor's degree from an Ivy League university and was on medical leave from a competitive MBA program at the time she presented for inpatient treatment of AN. She had also been a superior student and was on an athletic scholarship for track while in college. Zoe was hospitalized twice before for severe emaciation, at ages 17 and 23 years. Preceding this current hospital admission, she restricted her daily intake to under 300 calories and followed a rigid exercise routine. On admission she described a marked 'fear of fat,' an extreme fear of weight gain, and marked distortion of body image. Zoe's rigidity and inflexibility extended beyond her disordered eating. She was unable to tolerate uncertainty or perceived imperfections and resisted any deviations from her usual daily routine. In addition to fears about weight gain and appearance, she worried that foods, and other people, were "contaminated." She incessantly questioned staff regarding the preparation and origin of all foods and fluids, washed her hands repeatedly, and avoided removing her raincoat due to a belief that it protected her from environmental contaminants.

Zoe's treatment initially focused on medical stability and weight restoration. As her cognitive capacity improved, intensive psychotherapy for AN was initiated. Nevertheless, her obsessive–compulsive symptoms proliferated to a degree that essentially negatively affected her ability to participate in therapy within the inpatient eating disorders unit. As a result a combined regimen of an SSRI and atypical neuroleptic was initiated as well as a course of CBT with ERP focused specifically on her OCD symptoms. However, Zoe was resistant to challenging her OCD behaviors and refused to engage in ERP but remained compliant with the refeeding program. Upon discharge from the eating disorder program, she reinitiated ERP for OCD symptoms as an outpatient. At this time, Zoe remains underweight but has increased her caloric intake to a level that is maintaining her current weight. She is now able to partially deviate from her OCD routines and has been able to return to school; she struggles socially and with family due to her obsessional fears, and her marked rigidity has persisted.

Conclusions

The frequent co-occurrence and shared phenomenology between AN and OCD, and the familiality of anxiety symptoms in AN, suggest the two syndromes may share risk factors that impact neural systems regulating emotional and habit behavior in common. Nevertheless, at this time translational and clinical evidence linking these syndromes is limited and no research-driven guidelines for managing the comorbid patient exist. Even so, several rational principles apply to responsible management: (1) medical stability must be a first aim; (2) a multidisciplinary approach is crucial for integrating the management of medical, psychological, and nutritional components of the psychopathology; (3) while research support exists for use of ERP techniques in treating OCD symptoms and these same techniques may also aid in the reduction of certain abnormal eating disorder behaviors, other psychotherapeutic techniques will be required in the core features of AN; and (4) involvement of the family cannot be ignored when treating eating pathology in children and adolescents.

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Cluster C Personality Disorders and Anxiety Disorders

22

Nicole M. Cain, Emily B. Ansell, and Anthony Pinto

Over the past two decades, tremendous strides have been made in the treatment of anxiety disorders, with both psychopharmacological and cognitive-behavioral therapies (CBT) demonstrating significant efficacy (e.g., Barlow & Lehman, 1996; Lydiard, Brawman-Mintzer, & Ballenger, 1996; Mennin & Heimberg, 2000). However, despite a number of positive outcomes, many individuals with anxiety disorders continue to exhibit chronic impairment with low rates of recovery that appear to be worsened by certain comorbid psychiatric conditions (Bowen, Senthilselvan, & Barale, 2000; Bruce et al., 2005; Yonkers, Bruce, Dyck, & Keller, 2003). This chapter reviews the treatment complexities associated with having an anxiety disorder and a comorbid cluster C personality disorder (PD), which includes avoidant personality disorder (AVPD), obsessive–compulsive personality disorder (OCPD), and dependent personality disorder (DPD). We discuss the impact of these PDs on the presentation, clinical course, and treatment outcome for specific anxiety disorders as well as review two personality models that help to clarify the underlying mechanisms that contribute to treatment complexity. We also review treatment approaches that address the nuances associated with having comorbid cluster C personality features and use a clinical case presentation to illustrate the challenges of treating an anxiety disorder along with comorbid OCPD. Finally, we conclude with recommendations for future research to address these treatment complexities.

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Cluster C Personality Disorders

The *Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition Text Revision* (*DSM-IV*; American Psychiatric Association, 2000) diagnostic criteria for AVPD describe a pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation via four or more of the following characteristics: avoids occupational activities that involve interpersonal contact due to fears of criticism, disapproval, or rejection; is unwilling to develop relationships unless certain of being liked; is restrained in intimate relationships due to fears of being shamed or ridiculed; is preoccupied with being criticized or rejected; is inhibited in new interpersonal situations due to feelings of inadequacy; views self as socially inept, unappealing,

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or inferior; and/or is reluctant to take personal risks or engage in new activities because they may result in embarrassment. Prevalence in outpatient settings is around 15% (Zimmerman, Rothschild, & Chelminski, 2005) and in the general population is between 2 and 5% (Grant et al., 2004; Torgersen, Kringlen, & Cramer, 2001). Individuals with an AVPD diagnosis present as aloof, apprehensive, and guarded while internally experiencing feelings of inadequacy (Ansell & Grilo, 2007).

OCPD (APA, 2000) is a pattern of orderliness, perfectionism, and rigid control that interferes with efficiency, task completion, and social interactions. Diagnostic criteria require four or more of the following: preoccupation with details, rules, lists, schedules, and organization to the extent that the major point of the activity is lost; perfectionism that interferes with task completion; excessive devotion to work to the exclusion of leisure activities; is overconscientious, scrupulous, or inflexible about morality, ethics, or values; inability to discard worn-out or worthless objects that have no real or sentimental value; reluctance to delegate tasks; a miserly spending style toward self and others; and/or rigidity and stubbornness. Prevalence in outpatient settings is estimated between 8 and 9% (Zimmerman et al., 2005) and in the general population between 2 and 8% (Grant et al., 2004; Torgersen et al., 2001). The need for interpersonal control in OCPD can lead to hostility and occasional explosive outbursts of anger at home and work (Villemarette-Pittman, Stanford, Greve, Houston, & Mathias, 2004).

DPD (APA, 2000) is described as an excessive need to be taken care of and fears of autonomy expressed through submissive and clinging behaviors and fears of separation as indicated by five or more of the following criteria: difficulty making everyday decisions without an excessive amount of advice or reassurance from others; needing others to assume responsibility for major areas of his or her life; difficulty expressing disagreement with others due to fears that support or approval will be withdrawn; difficulty initiating projects or carrying out tasks autonomously; going to excessive lengths to obtain nurturance and support from others, including volunteering for unpleasant or aversive tasks; feeling discomfort or helplessness when alone due to excessive fears of being unable to take care of oneself; urgent seeking of a new relationship to provide care and support when a previous relationship ends; and/or unrealistic fears of preoccupation with being left to take care of oneself. Prevalence in outpatient settings is estimated at 1.4% (Zimmerman et al., 2005) and in the general population between 0.5 and 1.5% (Grant et al., 2004; Torgersen et al., 2001).

It is important to note that cluster C PDs share many of the same psychometric limitations as other Axis II PDs due to the structure of *DSM-IV's* categorical classification system (Clark, 2007; Widiger & Trull, 2007). Criticisms of the system include excessive co-occurrence among Axis II disorders, extreme heterogeneity among patients with the same Axis II disorder, arbitrary diagnostic thresholds for the boundary between "normal" and "pathological" personality functioning, and inadequate coverage of personality pathology such that the diagnosis of PD not otherwise specified (NOS) is the most common PD diagnosis (Widiger & Trull). In addition, moderate reliability for cluster C PDs has been identified. McGlashan et al. (2000) reported reliabilities for AVPD and OCPD of 0.68 and 0.71, respectively. Blais and Norman (1997) reported a reliability of 0.67 for DPD. However, concerns about reliability continue to be a focus across *DSM-IV* Axis II disorders (Clark, 2007).

Nature of the Problem

Anxiety disorders are highly prevalent diagnoses and are associated with substantial life impairments (Boden, Fergusson, & Horwood, 2007; Bruce et al., 2005; Grant et al., 2005; Roy-Byrne & Cowley, 1994; Weisberg, 2009; Yates, 2009). These significant impairments are often complicated by the presence of a comorbid PD diagnosis which, as we will discuss below, has been found to increase clinical severity, decrease psychosocial functioning, reduce the probability of remission, and increase the likelihood for relapse.

Prevalence of co-occurrence. Investigators have documented the significant prevalence of co-occurring AVPD, OCPD, and DPD in individuals with anxiety disorders. Brown and Barlow (1992) reported high rates of comorbidity among anxiety disorders and PDs, especially for cluster C PDs. Oldham et al. (1995), in a study of 200 inpatients and outpatients, found that the odds of an anxiety disorder co-occurring with AVPD, OCPD, or DPD was more than five times greater than chance.

Within specific anxiety disorders, 40–70% of patients with panic disorder also met criteria for a comorbid PD (Otto & Gould, 1996) with the majority receiving diagnoses of AVPD, OCPD, or DPD (Mennin & Heimberg, 2000). In a review of AVPD, Alden, Laposa, Taylor, and Ryder (2002) reported that the frequency of comorbid generalized social phobia and AVPD ranges from 25 to 89%. Reich (2000) noted that when examining the co-occurrence of social phobia and PDs other than AVPD, DPD is the most frequent comorbid PD. Finally, recent studies on OCD have consistently found elevated rates of OCPD, with estimates ranging from 23 to 34% (Garyfallos et al., 2010; Lochner et al., 2011; Pinto, Mancebo, Eisen, Pagano, & Rasmussen, 2006; Samuels et al., 2000). This increased prevalence of cluster C PDs with anxiety disorders has significant impacts on severity, functioning, and course of anxiety disorders.

Increased symptom severity and decreased functioning. Prior research in individuals with anxiety disorders and co-occurring cluster C PDs indicate a clinical presentation associated with increased severity of psychopathology. For example, patients with panic disorder and a comorbid PD diagnosis were more likely than panic patients without a comorbid PD to have a history of depression, have a history of childhood anxiety, and exhibit more symptom severity prior to beginning treatment (Pollack, Otto, Rosenbaum, & Sachs, 1992). Individuals with social phobia and comorbid AVPD have consistently demonstrated more severe symptoms and poorer global functioning than those without AVPD suggesting that comorbid AVPD may be an indicator of greater severity (Brown, Heimberg, & Juster, 1995; Heimberg, 1996; Heimberg, Holt, Schneier, Spitzer, & Liebowitz, 1993; Hofmann, Newman, Becker, Taylor, & Roth, 1995; Hope, Herbert, & White, 1995; Van Velzen, Emmelkamp, & Scholing, 2000). In OCD, both Coles, Pinto, Mancebo, and Rasmussen (2008) and Garyfallos et al. (2010) found that individuals with OCD plus OCPD, when compared to individuals without OCPD, had a significantly younger age at onset of first obsessive–compulsive symptoms, as well as poorer psychosocial functioning, even though the groups did not differ in overall severity of OCD symptoms. In addition, they reported higher rates of hoarding and incompleteness-related symptoms (including symmetry obsessions and cleaning, ordering, repeating compulsions), as compared to OCD subjects without OCPD. It appears that the presence of a co-occurring PD increases the severity of the psychopathology across anxiety disorders.

Detrimental impact on course of anxiety disorders. There are relatively few empirical investigations of the prospective course of anxiety disorders. In a 5-year prospective study examining the natural course of anxiety disorders in 514 participants as part of the Harvard/Brown Anxiety Research Program (HARP), comorbid DSM-III-R PDs were associated with reduced remission rates for generalized anxiety disorder (GAD) and social phobia, but not panic disorder (Massion et al., 2002). Specifically, DPD and AVPD decreased remission rates for GAD and AVPD decreased remission rates for social phobia. However, this study had several limitations. Notably, only three anxiety disorders were assessed, and the rates of specific PDs in the overall sample was too low to allow the analysis of some PDs as course predictors (e.g., only 5–9% OCPD). In addition, their analyses examined remissions but did not look at relapse, chronicity, or new episodes of anxiety disorders. In a large study of the naturalistic course of OCD, those with comorbid OCPD were significantly less likely to partially remit from OCD after 2 years as compared to those without comorbid OCPD, controlling for the presence of other cluster C personality disorders (Pinto, 2009).

Shea et al. (2004) investigated longitudinal associations between PDs and Axis I disorders

over a 2-year follow-up period using a prospective design and continuous measures of course within the Collaborative Longitudinal Personality Disorders Study (CLPS), a prospective, naturalistic study designed to assess the course and outcome of patients with PDs. Specifically, they were interested in examining improvement in Axis I disorders as a predictor of remission from PDs as well as improvement in PDs as a predictor of remission from anxiety disorders. They found that AVPD demonstrated significant associations with social phobia and OCD in both directions such that decreased AVPD symptoms were associated with improvements in social phobia and OCD and that improvements in social phobia and OCD were associated with improvements in AVPD symptoms over 2 years. OCPD was not associated with changes in anxiety disorders over a 2-year course.

Ansell et al. (2010) extended the findings of Shea et al. (2004) in the CLPS sample by examining rates of remission, relapse, and new onset of anxiety disorders in the CLPS study groups over a 7-year follow-up period. Ansell and colleagues found that OCPD was associated with increased risk for new onset of OCD, GAD, and agoraphobic episodes; increased risk of GAD relapse; and decreased risk for PTSD relapse over and above other predictors. In addition, they found that AVPD was associated with decreased likelihood of social phobia remission, increased likelihood of social phobia and OCD onset, and greater chronicity in social phobia episodes. AVPD was also associated with decreased risk for relapse of panic disorder with agoraphobia (Ansell et al., 2010). The research suggests that, in general, individuals with cluster C PDs have a more negative natural course of anxiety disorder symptoms.

Detrimental impact on treatment of anxiety disorders. Consistent with the research on natural course, the majority of treatment outcome studies suggest that comorbid PDs are associated with negative outcomes in the treatment of anxiety disorders. For example, Chambless, Renneberg, Goldstein, and Gracely (1992) found that avoidant, dependent, histrionic, and paranoid PDs were associated with negative treatment outcome following CBT for panic disorder. Hoffart (1994) found that avoidant traits were significantly associated with poorer clinical outcome at 1-year post-CBT treatment for panic disorder. Feske, Perry, Chambless, Renneberg, and Goldstein (1996) found that individuals with comorbid social phobia and AVPD improve at a slower rate than those with social phobia alone. In a 6-year follow-up of 284 Norwegian outpatients using DSM-III-R diagnoses, OCPD predicted panic disorder at follow-up and AVPD predicted social phobia at follow-up (Alnæs & Torgersen, 1999). In studies of OCD, the presence of OCPD predicted poorer response to serotonin reuptake inhibitor treatment (Cavedini, Erzegovesi, Ronchi, & Bellodi, 1997) and exposure and ritual prevention (Pinto, Liebowitz, Foa, & Simpson, 2011).

It is important to note that research has not consistently identified negative outcomes in the treatment of anxiety disorders with comorbid cluster C PDs. For example, Dreessen, Arntz, Luttels, and Sallaerts (1994) examined the role of comorbid PDs in a sample of patients with panic disorder with or without agoraphobia. Thirty-one patients received CBT over a 12- to 15-week period and they found no differences on change from pre- to posttreatment for panic disorder patients with and without comorbid PDs. Similarly, a recent study found greater improvements in psychodynamic treatment of patients with panic disorder and cluster C PD comorbidity (Milrod, Leon, Barber, Markowitz, & Graf, 2007). In social phobia, Brown et al. (1995) found similar rates of response to group CBT among individuals with generalized social phobia with and without AVPD. Huppert, Strunk, Roth Ledley, Davidson, and Foa (2008) found that comorbid AVPD did not predict differential treatment response for social phobia. In fact, they found that individuals with AVPD improved more than those without AVPD early in treatment. In addition, as noted above, Ansell et al. (2010) found that AVPD was associated with *decreased* risk for relapse of panic disorder with agoraphobia and OCPD was associated with *decreased* risk for relapse of PTSD over and above other psychological predictors. Further research is needed to clarify how and for whom the treatment outcome of anxiety disorders is better with comorbid cluster C PDs. These findings may

reflect personality trait tendencies (e.g., avoidance and rigidity) that decrease experiences that may be associated with relapse.

Theoretical Models for How Personality Disorders Contribute to Treatment Complexity

Given the results investigating the impact of AVPD, OCPD, and DPD on the clinical course and treatment outcome of anxiety disorders, it is necessary to examine the features of personality that contribute to this treatment complexity. Several models have been proposed to address the mechanisms by which personality and psychopathology may influence the presentation or appearance of one another; may share a common, underlying etiology; and may contribute to the development or etiology of one another, thus leading to increased comorbidity and treatment complexity (Klein, Wonderlich, & Shea, 1993; Widiger & Smith, 2008; Widiger, Verheul, & van den Brink, 1999). In this chapter, we review two models, the spectrum model and the pathoplastic model, which help to clarify how cluster C personality features may impact the clinical presentation, clinical course, and treatment of specific anxiety disorders.

The spectrum model. The spectrum model argues that PDs and personality traits may represent characterological variants of Axis I mental disorders (Widiger & Smith, 2008) thus leading to high rates of comorbidity between the anxiety disorders and PDs. Brown and Barlow (1992) noted that the high co-occurrence of cluster C PDs among anxiety disorders may speak to the limitations inherent in the diagnostic criteria for these disorders and may point to the fact that Axis I anxiety disorders and Axis II cluster C disorders occur along a common dimension with differences primarily existing on chronicity and severity. For example, some argue that OCPD should be considered part of an OCD spectrum of disorders (Bartz, Kaplan, & Hollander, 2007), but underlying etiological similarities and differences have not been adequately studied to date (Pinto, Eisen, Mancebo, & Rasmussen, 2008).

The most common example of the spectrum model is the significant overlap between AVPD and generalized social phobia (Siever & Davis, 1991). Researchers have often noted a high degree of overlap between the generalized subtype of social phobia and AVPD (e.g., Heimberg, 1996; Hofmann, Heinrichs, & Moscovitch, 2004; Schneier, Spitzer, Gibbon, Fyer, & Liebowitz, 1991). This finding is not surprising given that six of seven diagnostic criteria for AVPD include the social/interaction component that is essential to the diagnosis of social phobia. However, this high degree of comorbidity has led researchers to question the utility of maintaining two diagnostic categories on two separate DSM axes. It has been suggested that it may be more clinically useful to consider these diagnoses as different points on a social phobia continuum of increasing severity: from non-generalized social phobia to generalized social phobia without AVPD to generalized social phobia with AVPD. This would allow for treatment interventions to be designed to target each point on the social phobia continuum thus improving clinical course and outcome for social phobia (Hummelen, Wilberg, Pederen, & Karterud, 2007; McNeil, 2001).

The pathoplastic model. Pathoplasticity is characterized by a mutually influencing non-etiological relationship between psychopathology and another psychological system, such as personality (Klein et al., 1993; Widiger & Smith, 2008; Widiger et al., 1999). In this way, psychopathology and personality influence the expression of each other, but neither exclusively causes the other, as is hypothesized to be the case in a spectrum relationship (Widiger et al.). Pathoplasticity recognizes that the expression of certain maladaptive behaviors, symptoms, and mental disorders all occur in the larger context of an individual's personality (Millon, 1996, 2005).

One example of a pathoplastic model uses interpersonal circumplex theory (IPC; Leary, 1957) to examine meaningful heterogeneity in social processes and traits within Axis I disorders. Interpersonal theory posits that adaptive and maladaptive interpersonal styles can be described using the two dimensions of the IPC: communion and agency. This model depicts an individual's interpersonal style by placing him or her in the

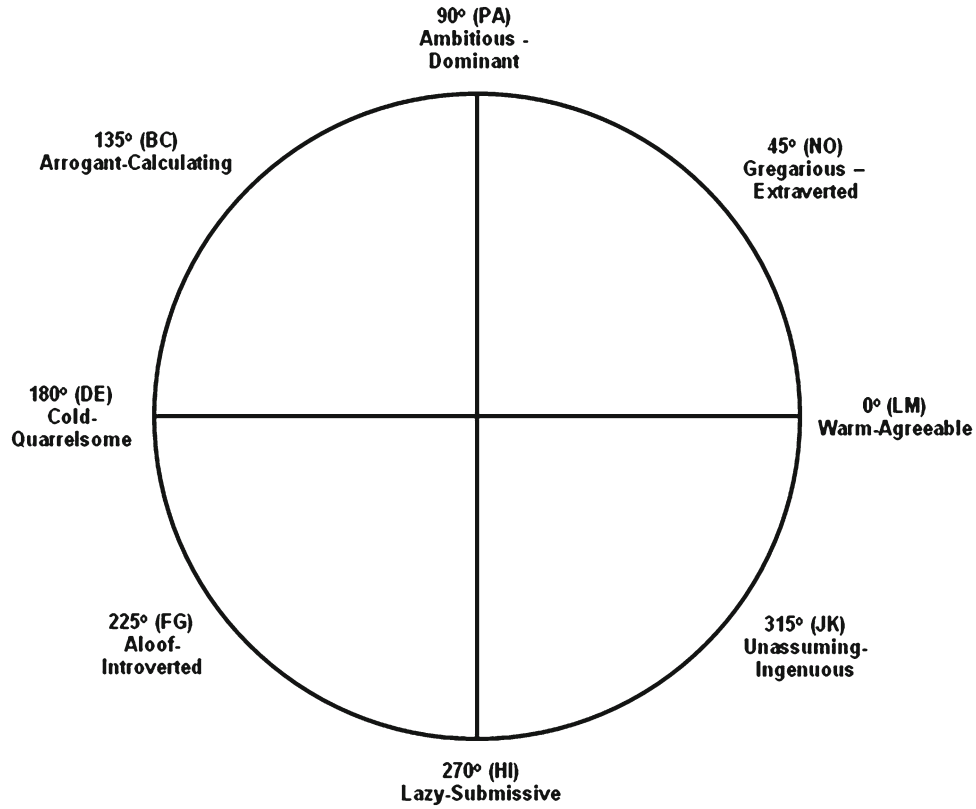

Fig. 22.1 Interpersonal circumplex. Note. An example of the eight octants found in the Interpersonal circumplex (IPC) adapted from Leary (1957). Octants are labeled with the alphabetical notation originally provided by Leary (1957)

two dimensional space created by the orthogonal dimensions of communion and agency (see Fig. 22.1 for an example of an IPC adapted from Leary, 1957). Circumplex octants offer useful summary descriptors of interpersonal behavior, marking the poles of the main dimensions but also representing blends of the underlying dimensions (i.e., hostile-dominance or friendly-submissiveness) (Pincus & Gurtman, 2006). Figure 22.1 illustrates a two-letter octant labeling scheme which has been used by convention to refer to the specific octants across measures with ease (e.g., PA, BC, DE). The IPC allows for the location of individual or group data within the circumplex. By computing scores on each axis, a set of Cartesian coordinates can be generated to define the location of the predominant interpersonal style reported by individuals or groups.

Interpersonal pathoplasticity can describe the observed heterogeneity in the phenotypic expression of psychopathology (e.g., Barrett & Barber, 2007), predict variability in response to psychotherapy within a disorder (e.g., Alden & Capreol, 1993; Borkovec, Newman, Pincus, & Lytle, 2002; Maling, Gurtman, & Howard, 1995), and account for a lack of uniformity in regulatory strategies displayed by those who otherwise are struggling with similar symptoms (e.g., Wright, Pincus, Conroy, & Elliot, 2009). Differences in interpersonal diagnosis will affect the manner in which patients express their distress and will influence the type of strategy needed to regulate their self, affect, and relationships (Pincus, Lukowitsky, & Wright, 2010).

IPC and cluster C. Several studies have examined cluster C PDs using the IPC. For example, Pincus and Wiggins (1990) reported that AVPD is associated with low agency and low communion on the IPC, which was replicated by Soldz,

Budman, Demby, and Merry (1993) in a sample of psychotherapy patients. More recently, Leising, Rehbein, and Eckhardt (2009) examined predictors of AVPD using octants of the IPC and found that problems with social inhibition were the best interpersonal predictor of AVPD diagnosis. A series of studies relating dependency to the IPC suggested that dependency is associated with the entire range of friendly, friendly-submissive, and submissive interpersonal functioning (Pincus, 2002; Pincus & Gurtman, 1995; Pincus & Wilson, 2001). Specifically, Pincus and Wilson (2001) noted that dependency might be expressed via passivity, helplessness, ingratiating deference, or a warm-loving approach.

Finally, in a recent investigation relating a measure of OCPD to the IPC, Cain (2011) found that the overall construct of OCPD was associated with hostile-dominant interpersonal functioning. However, the trait dimensions underlying OCPD were associated with a wide range of interpersonal functioning. In particular, difficulty with change was associated with submissive, nonassertive interpersonal problems; maladaptive perfectionism and reluctance to delegate were associated with exploitable and unassuming interpersonal problems; emotional restraint was associated with social inhibition; and rigidity was associated with hostile-dominant interpersonal problems (Cain, 2011).

IPC pathoplasticity with anxiety disorders. Numerous investigations have found that individual differences in interpersonal style exhibit pathoplastic relationships with anxiety disorders (e.g., Cain, Pincus, & Grosse Holtforth, 2010; Kachin, Newman, & Pincus, 2001; Kasoff & Pincus, 2002; Pincus & Borkovec, 1994; Salzer et al., 2008). For example, patients diagnosed with GAD can be discriminated based on distinct clusters of interpersonal problems (Kasoff & Pincus, 2002; Pincus & Borkovec, 1994; Przeworski et al., 2011). In these studies, Pincus and colleagues found four interpersonal subtypes of GAD patients reflecting predominantly cold, intrusive, exploitable, and nonassertive problems, respectively. These groups did not differ in symptom severity, comorbid psychopathology, or attachment style, but did exhibit differences in treatment response. In particular, Przeworski et al. (2011) reported that nonassertive and exploitable GAD patients exhibited higher end-state functioning immediately following CBT treatment and at 6-month follow-up than cold and intrusive GAD patients. Thus, the contrasting styles of interpersonal presentation within a diagnostic category have important implications for case formulation and treatment planning. Adding to the strength of these findings, these GAD interpersonal subtypes have also been replicated in a German clinical sample (Salzer et al., 2008).

Interpersonal pathoplasticity has also been examined in nonclinical (Kachin, Newman, & Pincus, 2001) and clinical (Cain et al., 2010) samples of socially phobic individuals. In their clinical sample, Cain et al. found two interpersonal subtypes of socially phobic patients: a friendly-submissive subtype and a cold-submissive subtype. The two subtypes did not differ on pretreatment symptom severity or diagnostic comorbidity, but did exhibit differential responses to outpatient psychotherapy. Overall, friendly-submissive social phobics had significantly lower scores on measures of social anxiety and significantly higher scores on measures of well-being and satisfaction at posttreatment than cold-submissive social phobics.

Taken together, the results of these studies investigating interpersonal pathoplasticity in anxiety disorders suggest that examining interpersonal traits may be key to understanding the influence of cluster C PDs on anxiety disorder course and treatment outcome. It may also be useful to begin developing and testing guidelines to more effectively treat patients who have a similar Axis I diagnosis but different interpersonal problems.

Treatment Approaches

Despite high rates of co-occurrence and poorer clinical course and treatment outcome, there is limited research examining specific treatment strategies and interventions that may be effective for treating anxiety disorders with comorbid cluster C PDs. Treatment of AVPD with comorbid social phobia has been the most widely investigated of

the cluster C PDs. For example, Huppert et al. (2008) examined treatment outcome in social phobia with comorbid AVPD in one of five treatment conditions: fluoxetine pharmacotherapy, comprehensive cognitive-behavior group therapy (CCBT), CCBT + fluoxetine pharmacotherapy, CCBT + pill placebo (PBO), and PBO only. All participants received 14 weeks of active treatment. Results suggested that all treatments were superior to PBO, but no significant differences among the active treatments emerged. As noted earlier, they found that comorbid AVPD did not predict differential treatment response for social phobia. However, Huppert et al. (2008) noted several qualitative differences between individuals with AVPD compared to those without AVPD. In particular, clinical impressions made during treatment suggested that patients with AVPD often avoid a range of situations that make them feel uncomfortable—not just anxiety-provoking social situations. In addition, those with AVPD often seem unable or unwilling to tolerate the anxiety associated with confronting any novel situation. Huppert et al. (2008) argued that perhaps those individuals with AVPD may need exposure to situations beyond those that are social to learn that novel experiences in general should not be avoided.

Borge et al. (2010) examined changes in AVPD and DPD dimensions in 77 socially phobic patients using a medication-free residential cognitive therapy (CT) or residential interpersonal therapy (IPT). They found that both treatments were associated with a decrease in avoidant and dependent personality dimensions; however, DPD dimensions decreased significantly more in CT than in IPT. Also, they found that changes in social phobia symptoms during treatment did not predict changes in AVPD or DPD dimensions. Their results suggest that socially phobic patients with comorbid DPD may benefit from CT rather than IPT. Borge et al. (2010) noted that it is important that AVPD and DPD be considered when assigning socially phobic individuals to specific treatments.

There is also limited research examining treatments for the *personality features* of cluster C PDs that impede progress in the treatment of anxiety disorders. For example, Alden and Capreol (1993) examined the extent to which the interpersonal problems of AVPD individuals predicted treatment response to behavioral treatments. Results showed that AVPD patients reported two distinct types of interpersonal problems on the IPC: exploitable problems and cold problems. Patients who reported problems related to being exploited by others (exploitable avoidants) benefited from both graduated exposure and skills training procedures, while AVPD patients with problems related to cold, distrustful, and angry behavior (cold avoidants) only benefited from graduated exposure. Alden and Capreol (1993) suggested that patients with AVPD differ in terms of their interpersonal problems and that these differences may influence response to behavior therapy for anxiety and avoidance.

Riley, Lee, Cooper, Fairburn, and Shafran (2007) examined CBT for clinical perfectionism. As described earlier, perfectionism is a core feature of OCPD. Riley et al. noted that perfectionism often complicates and impedes the progress of treatment of Axis I disorders particularly anxiety disorders. They conducted a randomized controlled trial of CBT for clinical perfectionism in twenty individuals. They defined clinical perfectionism as a dysfunctional type of self-focused perfectionism in which the individual pursues self-imposed, personally demanding standards despite adverse consequences (Riley et al.). Treatment consisted of ten sessions of CBT over 8 weeks. The treatment was manualized and consisted of four elements originally developed by Fairburn, Cooper, and Shafran (2003): (1) identifying perfectionism as a problem and the ways perfectionism is maintained (e.g., repeated performance checking or over training); (2) conducting behavioral experiments to learn more about the nature of their perfectionism and alternative ways of coping (e.g., the impact of checking repeatedly vs. checking only occasionally); (3) psychoeducation and cognitive restructuring (in combination with behavioral experiments) to modify personal standards, self-criticism, and cognitive biases such as selective attention to perceived failure; and (4) broadening the individual's capacity for self-evaluation, by identify-

ing and adopting alternative cognitions and behaviors. Riley et al. (2007) found that 75% of individuals (15/20) demonstrated clinically significant improvement and treatment gains were maintained at 8- and 16-week follow-ups. They recommended that this treatment could be used as an adjunct to CBT when clinical perfectionism is a treatment barrier.

Case Example: EX/RP for Patient with Comorbid OCD and OCPD

"George" is a highly intelligent, single male in his late 20s with severe OCD and comorbid OCPD. His major obsessions center on a need for exactness/certainty and a need for the "just right" feeling before completing an action. His major compulsions include checking, rereading, repeating, and handwashing. Beyond his OCD symptoms, George admits that he is rigid, stubborn, highly rule bound, and guided by a strict sense of right and wrong. He is precise and even exacting in his words and actions. He experiences extreme interference in functioning. He has been unemployed for the last 2 years, unable to return to his job in sales after taking a medical leave of absence due to OCD. He lives on his own in an apartment and currently supports himself with savings. George spends most of his time sleeping or watching television to avoid triggering symptoms. With the exception of buying fast food and infrequent social contacts, he is mostly homebound. He takes excessive amounts of time to complete even simple tasks (showering, laundry, preparing a meal, making phone calls, reading, handling money, and typing or using a computer) and will avoid these activities whenever possible since they are usually physically and mentally exhausting for him to complete. Because of his need for perfection and completeness, he is unable to manage or set limits with his time and will strongly object to any attempts by others to limit time spent on activities. For example, George took more than 8 h to complete clinic intake questionnaires that others can complete in less than 1 h.

George received exposure and response prevention (EX/RP) treatment for OCD (8 weeks; twice-weekly 90-min sessions) as part of a research study. Exposure sessions consisted of reviewing progress with between-session EX/RP procedures, confronting fears in session for prolonged periods of time without ritualizing, and assigning specific exposures to practice before the next session. George was instructed to stop rituals after the first exposure session and to record any rituals that occurred.

According to his therapist, George's personality style (e.g., precision about wording of therapist's instructions, rigidity with regard to how he completes his rituals, anger outbursts) significantly interfered in treatment progress. George was not compliant with treatment procedures, particularly assigned practice exposures and response prevention, and was frequently argumentative. He objected to the concept of response prevention, describing it as "unrealistic," and believed that it would be "wrong" not to do his rituals at all. One of his exposure assignments was to watch part of a TV program, rather than watching it from beginning to end (which was George's rule for TV viewing). George noted that after he completed the exposure assignment, he went back to watching TV "my way." For another exposure assignment, George was to spend 30 min using his computer imperfectly. He recalls getting so frustrated during this particular assignment that he punched his wall. George also adopted a narrow view of his assignments and had difficulty generalizing to related situations. After he skipped a session (which would not be made up) and arrived late for others, he deemed the treatment "imperfect." At the posttreatment assessment, he showed a mild reduction in symptoms. However, he was unable to maintain gains by the 6-month posttreatment assessment and OCD severity returned to baseline level.

Conclusions and Future Directions

This clinical case illustrates the detrimental impact that cognitions, behavior, and interpersonal problems associated with OCPD can have on the treatment course of OCD. As reviewed above, there is an extensive literature showing

that the presence of a comorbid cluster C PD diagnosis increases clinical severity, decreases psychosocial functioning, reduces the probability of remission, and/or increases the likelihood for relapse for anxiety disorders. However, despite the negative outcomes associated with having a comorbid cluster C PD, there is relatively little research examining treatment approaches to address this complexity. Future research is needed to begin developing and testing treatment interventions that will more effectively treat cluster C comorbidity for anxiety disorders. First, modifications to traditional CBT approaches may be necessary to target this complexity. For example, in their clinical impressions, Huppert et al. (2008) noted that patients with social phobia and AVPD avoid a wide range of situations and they are often unwilling to tolerate anxiety associated with novel stimuli. Huppert et al. suggested that modifications to CBT might be needed to expose those patients with AVPD to situations beyond those that are just social. Similarly, other researchers have suggested that lengthening brief CBT treatments and/or paying greater attention to maladaptive interpersonal patterns in CBT treatments may be necessary when treating patients with comorbid cluster C PDs (see Crits-Christoph & Barber, 2007).

Second, more research is needed to investigate possible adjunctive treatments for anxiety disorders that may be used to target comorbid cluster C PDs and their underlying facets. Riley et al. (2007) demonstrated clinically significant improvement in a study investigating CBT for clinical perfectionism, a facet of OCPD. They recommended that their treatment could be used as an adjunct to CBT when clinical perfectionism is a barrier to treatment. More research on adjunctive treatments for cluster C PDs and their underlying facets are needed.

Finally, another future direction would be to investigate matching patients to specific treatment modalities. For example, Borge et al. (2010) found that DPD dimensions improved more in CT for social phobia than in IPT. They suggested that patients with DPD be assigned to CT in order to maximize treatment outcome. Similarly, Alden and Capreol (1993) found differences in treatment response to behavioral interventions depending on the types of interpersonal problems reported by AVPD individuals. In particular, they noted that patients who reported problems related to being exploited by others benefited from graduated exposure and skills training procedures, while AVPD patients with problems related to cold, distrustful, and angry behavior only benefited from graduated exposure.

Given the substantial impairments and negative outcomes associated with anxiety disorders and co-occurring cluster C pathology, it is necessary to continue to explore the mechanisms by which personality and psychopathology may influence the presentation or appearance of one another; may share a common, underlying etiology; and may contribute to the development or etiology of one another, thus leading to increased comorbidity and treatment complexity. In addition, significant advances in treatment are necessary to adequately address this complexity and improve treatment outcome.

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Therapist Barriers to the Dissemination of Exposure Therapy

23

Brett J. Deacon and Nicholas R. Farrell

With hundreds of clinical trials and dozens of meta-analytic reviews attesting to its effectiveness (Deacon & Abramowitz, 2004; Olatunji, Cisler, & Deacon, 2010), exposure-based cognitive-behavioral therapy (CBT) is the most empirically supported psychological treatment for the anxiety disorders. Clinical practice guidelines published by the American Psychiatric Association (2011) and the National Institute for Clinical Excellence (2011) recommend exposure-based CBT approaches as first-line anxiety treatments. Relative to pharmacotherapy, exposure-based therapy typically produces similar short-term benefit and superior long-term maintenance of treatment gains (e.g., Barlow, Gorman, Shear, & Woods, 2000). Exposure therapy is also more cost-effective (Heuzenroeder et al., 2004) and more acceptable and preferable to clients and their caregivers (Brown, Deacon, Abramowitz, & Whiteside, 2007; Deacon & Abramowitz, 2005). Taken together, these observations support a strong case for exposure-based CBT as the treatment of choice for anxiety disorders. Indeed, this treatment may have more scientific support than any other psychotherapy of any kind, for any mental disorder.

Despite its demonstrated effectiveness, exposure therapy is rarely used by practicing clinicians. To illustrate, in a sample of over 800 licensed doctoral-level psychologists, Becker, Zayfert, and Anderson (2004) found that fewer than 20 % of respondents reported using exposure therapy to treat clients with posttraumatic stress disorder (PTSD). Indeed, exposure was not widely utilized even among trauma experts with specialized training in this approach. These findings were replicated in a more recent survey of more than 250 trauma experts by van Minnen, Hendriks, and Olff (2010). Imaginal exposure was the least used treatment for PTSD, and respondents preferred both eye movement reprocessing and desensitization and supportive counseling to exposure therapy despite the weaker scientific evidence for the efficacy of these approaches.

The underutilization of exposure therapy is not specific to PTSD. A German study found that although almost all therapists requested coverage for exposure therapy from obsessive–compulsive disorder (OCD) clients' health insurers, over 80 % of their clients reported that no exposure component was used in their treatment (Böhm, Förstner, Külz, & Voderholzer, 2008). In addition, Becker et al. (2004) reported that fewer than 15 % of clinicians with expertise in PTSD reported using exposure therapy when treating other anxiety disorders due to a lack of training. Poor dissemination of CBT to mental health practitioners has resulted in a lack of client access to this treatment (Gunter & Whittal, 2010). The majority of adults with an anxiety disorder do not receive efficacious treatment (e.g., Stein et al., 2004; Young, Klap, Sherbourne, & Wells, 2001;

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Young, Klap, Shoai, & Wells, 2008), and when clients are able to access psychotherapy, it is rarely evidence-based (Goisman, Warshaw, & Keller, 1999; Taylor et al., 1989).

Dissemination failure alone cannot fully account for the underutilization of exposure therapy. A startling finding revealed by Becker et al. (2004) is that the majority of therapists who had received training in exposure therapy did not use this treatment. Why would therapists trained in this approach, and presumably aware of its well-established scientific efficacy, shun exposure for less substantiated therapies? We propose that *negative beliefs about exposure therapy* (*e.g.*, *that it is unethical*, *intolerable*, *and unsafe*) *impede the utilization of this treatment*, *even among therapists trained to administer it*.

Findings from therapist surveys reveal that even when exposure therapy is utilized, it is often implemented in a suboptimal manner. Freiheit, Vye, Swan, and Cady (2004) found that psychologists, nearly all of whom reported using "CBT" with their anxious clients, utilized techniques such as relaxation and breathing retraining more frequently than exposure in the treatment of anxious clients. Comparable findings were reported by Hipol and Deacon (in press) in a survey of Wyoming mental health practitioners. Therapist-assisted exposure was used by less than 30 % of clinicians in the treatment of clients with OCD, social phobia, panic disorder, and PTSD. Of interest, the majority of therapists who did utilize exposure with their clients also reported using breathing retraining, progressive muscle relaxation, meditation, and nondirective supportive therapy. Similar to the psychologists surveyed by Freiheit et al. (2004), Wyoming therapists utilized client self-directed exposure more than twice as often as therapist-assisted exposure (Hipol & Deacon, in press).

Surveys of practicing clinicians (Freiheit et al., 2004; Hipol & Deacon, in press) indicate that the implementation of exposure therapy in the community, when it occurs, is very different from its typical manner of delivery in treatment manuals studied in clinical trials (e.g., Kozak & Foa, 1997). The primary differences can be summarized as follows: (a) Community therapists emphasize client self-directed exposure rather than therapist-assisted exposure, and (b) community therapists typically combine exposure therapy with arousal-reduction strategies such as progressive muscle relaxation and breathing retraining, whereas treatment manuals typically omit such procedures and focus on the delivery of repeated, prolonged exposure tasks. The modal delivery of exposure by community therapists is concerning, as exposure appears less effective when implemented in a self-directed manner (e.g., Abramowitz, 1996), and arousal-reduction strategies are not evidence-based adjuncts and may even interfere with long-term improvement (e.g., Schmidt et al., 2000). Why do practitioners tend to implement exposure therapy in this manner? We hypothesize that *clinicians who use exposure therapy minimize the intensity of its delivery due to concerns about the adverse consequences of subjecting clients to high anxiety during exposure tasks*. This notion begs the question: How might minimizing the intensity of exposure therapy affect client outcomes?

Research has yet to directly address how therapist beliefs about exposure might influence the manner and effectiveness of its delivery. However, indirect evidence may be found in the Pediatric OCD Treatment Study (POTS Team, 2004), a large-scale, placebo-controlled clinical trial comparing exposure therapy, sertraline, and their combination in the treatment of children and adolescents with OCD. The study's primary finding was that combined treatment was superior to exposure therapy and sertraline alone, which did not differ from each other. However, this outcome was qualified by an extraordinarily large difference in the efficacy of exposure at two different study sites. Despite using procedures designed to standardize adherence with the exposure therapy treatment manual (e.g., direct supervision, case conferences, training meetings, review of videotaped sessions), exposure was more than four times as effective in reducing OCD symptoms at the University of Pennsylvania than at Duke University. At the Pennsylvania site, exposure alone was as effective as combination treatment; at Duke, augmentation with sertraline more than

doubled the efficacy of exposure therapy. How can such findings be explained? Franklin et al. (2004) reported significant variation between therapists in client outcomes and suggested that site differences were driven by "super-therapists" who may have set a more ambitious agenda with regard to exposure tasks and pushed their clients harder to pursue it (M. E. Franklin, September 10, 2010, personal communication). One (admittedly speculative) possibility raised by the POTS study is that *therapists who attain the best client outcomes deliver exposure therapy in a particularly intensive manner owing to their confidence in the safety, tolerability, and efficacy of this treatment*.

In summary, despite its status as the most effective psychological treatment for the anxiety disorders, exposure-based CBT is rarely utilized, even by clinicians trained in its delivery. Moreover, the minority of therapists who provide exposure therapy often do so in a less-than-ideal manner. A number of empirical findings suggest that therapists hold negative beliefs about exposure that may hinder its utilization and affect the manner in which it is delivered to anxious clients. These therapist-level barriers are reviewed below.

Barriers to the Dissemination of Evidence-Based Psychological Treatments

Undoubtedly, exposure therapy is affected by the same set of therapist barriers that obstruct the utilization of evidence-based psychotherapies more generally. These include a lack of training opportunities in CBT and an emphasis on training mental health professionals in practices not supported by scientific evidence. To illustrate, the majority of social work and professional clinical psychology (Psy.D.) graduate programs do not require a didactic and clinical supervision in *any* evidence-based treatment. Even when such training is provided (e.g., in psychiatry residency programs), it is often cursory and insufficient to instill adequate competency. At present, most required psychotherapy training in psychiatry, social work, and clinical psychology is not evidence-based (Weissman et al., 2006).

In the absence of scientifically grounded training, many mental health professionals are deeply ambivalent about the relevance of research to their clinical practice. In contrast to medicine in which there is near-unanimous agreement that practice should be guided by treatment guidelines derived from research evidence (Wolfe, Sharp, & Wang, 2004), mental health professionals often reject evidence-based treatments on the grounds that findings from clinical trials are invalid and irrelevant to real-world practice (e.g., Silberschatz, in Persons & Silberschatz, 1998). Indeed, the typical mental health practitioner is more likely to prize his or her intuition and experience over scientific evidence (Garb & Boyle, 2003). The notion that all psychotherapies are equivalent (aka, the "Dodo Bird" verdict), which remains popular despite clear evidence to the contrary (Hunsley & Di Giulio, 2002), provides little motivation for practitioners to seek additional training in evidence-based practices. The current tension between skeptical mental health practitioners and exasperated clinical scientists (Tavris, 2003) is reminiscent of the conflict that raged among physicians a century ago about whether the practice of medicine was an art or a science (Baker, McFall, & Shoham, 2009).

In 2006, the American Psychological Association published the organization's position statement on evidence-based practice in psychology. This report was the product of the Task Force on Evidence-Based Practice, a group composed of both ardent supporters and vehement opponents of the movement to identify and disseminate empirically supported treatments like exposure therapy. The Task Force defined evidence-based practice as "the integration of the best available research with clinical expertise in the context of client characteristics, culture, and preferences" (p. 273). This definition officially sanctions the notion that research evidence and clinical judgment are equally valid methods for selecting appropriate interventions. The report provides little guidance for resolving conflicts between the clinician's intuition and findings from empirical research, and therapists

thus appear free to consider their own practice "evidence-based" without regard to whether the treatments they use have passed muster in clinical trials. Indeed, a recent survey of clinical psychologists found that respondents, on average, characterized 73.1 % of their services as evidence-based according to the APA's definition (Berke, Rozell, Hogan, Norcross, & Karpiak, 2011). The modal response, provided by approximately one-third of psychologists, was 100 %. Thus, a striking incongruity exists between the low utilization of empirically supported treatments like exposure therapy and the high rate at which clinicians believe their practice is "evidence-based."

Negative therapist beliefs about the use of manualized treatments constitute another barrier to the dissemination of empirically supported treatments. Addis, Wade, and Hatgis (1999) identified several such beliefs about manuals, including the following: (a) The therapeutic relationship will be compromised, (b) treatment proceeds according to a one-size-fits all approach and cannot be adequately individualized to specific clients, and (c) therapist input and creativity will be stifled, thereby leading to job dissatisfaction. Although the accuracy of these perceptions is highly debatable (Addis et al.; Barlow, Levitt, & Bufka, 1999), they are commonly held by practicing clinicians and serve to dampen enthusiasm for the use of empirically supported treatments, including exposure therapy, that are often delivered using treatment manuals.

Dissemination efforts are also hampered by a host of economic and practical concerns. Learning a new psychotherapy is expensive, time-consuming, and requires a great deal of effort. Gray, Elhai, and Schmidt (2007) found that among a sample of trauma experts, the most endorsed barriers to use of empirically supported treatments included insufficient time to learn the treatment and attend training seminars, as well as the prohibitive expense associated with such training. Because experts in exposure-based CBT tend to be clustered in urban areas associated with major medical centers, many practitioners in rural settings lack convenient access to training opportunities.

Taken together, a large number of practical and ideological barriers contribute to the failure to adequately disseminate empirically supported treatments to mental health professionals. Principal among these include a lack of training in evidence-based interventions and the perception that science is only tangentially relevant to the practice of psychotherapy. In addition to these more general reservations about evidence-based treatments, exposure therapy is subject to a potent set of intervention-specific negative beliefs which we discuss below.

Therapist Barriers to the Dissemination of Exposure Therapy

Exposure therapy has a public relations problem with many in the field of psychotherapy (Olatunji, Deacon, & Abramowitz, 2009; Richard & Gloster, 2007). Prejudice against exposure often stems from the fact that this intervention evokes distress (albeit temporary), rather than soothes it, as one might intuitively expect a treatment for anxiety to do. More specific negative beliefs about exposure include the following: (a) It is unethical, (b) it poses an unacceptably high risk of harm to clients, and (c) it is stressful and potentially harmful to the therapist. In this section, we present a critical analysis of these concerns. Using case examples from our own clinical practice, we illustrate the manner in which endorsement of these beliefs might affect the manner in which clinicians implement exposure therapy.

Exposure therapy is unethical. The first principle in the American Psychological Association's *Ethical Principles of Psychologists and Code of Conduct* (2002) admonishes psychologists to "take care to do no harm" and "safeguard the welfare and rights" of their clients. Because exposure therapy entails deliberate provocation of anxiety and distress, some therapists believe its very nature violates accepted ethical standards. One therapist, quoted in a *New York Times* article (Slater, 2003), described exposure as "torture, plain and simple." Our experience suggests that this sentiment is commonplace among therapists across the mental health professions,

particularly those with psychodynamic and humanistic theoretical orientations, and is a primary reason why some practitioners do not provide exposure therapy—and would not do so, even if they were trained in this approach.

Some practicing exposure therapists likely harbor concerns about the ethicalness of this treatment. They may not consider exposure to be *inherently* unethical, but may tie its acceptability to the manner in which it is delivered. Exposure tasks that evoke very high levels of client anxiety, or that place the client in "extreme" situations beyond those encountered by most people on a daily basis (e.g., immersing one's hands in garbage), may be considered both unnecessary and ethically indefensible by well-meaning clinicians. Therapists who adopt this perspective may deliver exposure in an overly cautious and sympathetic manner in an attempt to safeguard their clients' rights and dignity. Consider the following case:

Mr. A is a 27-year-old Marine Corps veteran who served in operation Iraqi Freedom and currently suffers from combat-related PTSD. During his tour of duty he witnessed the deaths of numerous Iraqi civilians and members of his unit from gunfire and improvised explosive devices. He is bothered by intrusive recollections of these events and experiences distressing images of people around him being maimed and killed by explosions when he is in crowded public places.

A therapist overly concerned with upholding the ethical principles of beneficence and nonmaleficence (APA, 2002) might forego prolonged imaginal exposure with Mr. A, reasoning that asking him to revisit his painful memories would be inhumane. Alternatively, the therapist might implement imaginal exposure but allow the client to withhold the specific details of his traumatic experiences to minimize his distress. Rothbaum and Schwartz (2002) noted that overly sympathetic or cautious exposure therapists run the risk of unintentionally reinforcing their clients' fears. In the case of Mr. A, such an approach might also deprive him of the opportunity to emotionally process his traumatic memories, thereby preventing habituation to the full range of fear cues associated with his PTSD. The client might also fail to learn that particularly anxiety-provoking stimuli are not accompanied by catastrophic outcomes and that he possesses the ability to tolerate the distress they evoke.

Practitioners who believe exposure therapy to be unethical, either intrinsically or according to its manner of delivery, might benefit from considering the work of a physical therapist or physician. Often, their treatments involve exposing clients to temporary, manageable amounts of pain and distress for the sake of long-term recovery. Indeed, the experience of temporary discomfort is sometimes necessary to ensure the desired longer-term outcome. The process of exposure therapy requires that clients "invest anxiety now for a calmer future" (Abramowitz, Deacon, & Whiteside, 2010). Well-meaning therapists who minimize the anxiety invested by their clients for ethical and humanistic reasons are paradoxically depriving their clients of the optimally effective treatment they deserve.

Clinicians often assume that clients perceive exposure therapy as aversive and unethical and will instead prefer to undergo treatment that does not entail the distress associated with directly facing one's fears. Fortunately, exposure therapy appears to be held in generally high esteem by anxious clients and their caregivers. Compared to pharmacotherapy, exposure-based CBT is rated as more credible, acceptable, and likely to be effective in the long-term (Brown et al., 2007; Deacon & Abramowitz, 2005; Norton, Allen, & Hilton, 1983). Moreover, exposure therapy is rated as at least as acceptable, ethical, and effective as cognitive therapy and relationship-oriented psychotherapy by undergraduate students and agoraphobic clients (Norton et al.). The finding that therapist reservations about exposure therapy are not shared by clients who receive this treatment provides an important counterpoint to the notion that exposure therapy is inherently inhumane and unethical.

Exposure therapy is harmful to the client. Exposure is believed by some practitioners to place clients at an unacceptably high risk of harm in various ways. Most commonly, therapists worry that clients will be harmed by their own anxiety during exposure tasks. This concern

reflects a number of myths about the nature of anxiety itself. One such misconception is that the experience of prolonged, intense anxiety-related somatic symptoms may lead to a medical emergency, such as loss of consciousness or heart attack. A similar belief is that anxiety is literally intolerable in high doses. Some therapists believe their clients to be sufficiently fragile that the experience of high anxiety will cause them to decompensate, perhaps in the form of a psychotic episode or loss of control over their own behavior. A related belief is that trauma sufferers may be "revictimized" by the recollection of a painful memory. Other concerns associated with high anxiety during exposure tasks include the possibility of symptom exacerbation and/or treatment refusal and attrition. Common to these beliefs is the assumption that clients with anxiety disorders lack the resilience necessary to safely experience their own anxiety symptoms.

Exposure therapy is also sometimes assumed to pose a threat to clients in the form of dangerous stimuli used during exposure tasks. Examples include animals (e.g., dogs), potential contaminants (e.g., toilet seats), and external situations (e.g., driving). Some therapists believe that "extreme" exposure tasks, the likes of which appear at the top of many client fear hierarchies, are especially likely to be harmful.

How might such beliefs affect the delivery of exposure therapy? Consider the following three cases:

Mr. P, age 45, experiences daily, unexpected panic attacks. During his attacks, he has prominent symptoms of dizziness, shortness of breath, and heart palpitations which he fears will lead to a loss of consciousness. He avoids physical exercise and participation in any activities that evoke these sensations.

Mrs. G, age 28, gave birth to her first child two months ago. Since that time she has experienced intrusive, unwanted obsessions about stabbing her daughter with knives and drowning her in the bathtub. She has turned parenting duties over to her husband and avoids being alone in the house with her daughter.

Mrs. R is a 26-year-old married woman who is interested in having children. However, she is unwilling to become pregnant due to a severe phobia of vomiting. She believes that vomiting might cause her to choke and die and avoids stimuli that might cause her to become nauseous and/or ill.

Mr. P's exposure therapy would be expected to emphasize interoceptive tasks such as hyperventilation and breathing through a straw. A therapist who believes that the anxiety-related body sensations evoked by these exercises are potentially dangerous might employ concurrent arousal-reduction strategies such as relaxation and breathing retraining. Similarly, the therapist might encourage the client to perform the exercises using a small number of brief trials, each separated by a long rest period to allow his symptoms to subside. In this manner, the client would be spared from experiencing anxiety symptoms that the therapist fears could escalate to potentially dangerous levels. Unfortunately, the client would not be able to learn that the experience of prolonged and intense anxiety-related physical sensations, such as those experienced during his panic attacks, do not lead to catastrophic outcomes.

Mrs. G is extremely distressed by her obsessions and is ashamed of their content. A therapist concerned about the harmful effects of high anxiety is likely to be especially cautious in the use of exposure with this client. Concerned that Mrs. G would be unable to tolerate the distress associated with imaginal exposure to obsessions involving the violent death of her beloved daughter at her own hands, the therapist may forego this technique altogether. Alternatively, the therapist might allow the client to conduct imaginal exposure in a self-directed manner in order to avoid the heightened anxiety associated with sharing the details of her obsessional fears with the therapist. Concerned that the client might decompensate due to intolerably high anxiety during situational exposures (e.g., giving her daughter a bath) and act on her harming obsessions, the therapist might refrain from implementing in vivo exposure, or require the husband to be present as a safety measure. Exposure therapy conducted in this manner runs the risk of reinforcing the client's catastrophic beliefs about being crazy for

having such obsessions and posing a threat to her daughter's safety.

Mrs. R's emetophobia is driven primarily by the belief that she may choke and die during the act of vomiting. Despite the obvious therapeutic value of having the client vomit during exposure, the cautious therapist might elect to forego such an "extreme" task in order to avoid subjecting the client to intolerably high anxiety and the possibility, however remote, that vomiting may actually prove harmful. An exposure therapist concerned about the client's safety may proceed with tasks such as viewing video clips of individuals vomiting and asking the client to engage in activities with the potential to induce mild stomach discomfort (e.g., moderate exercise immediately following consumption of a large meal). Although such exposure tasks may be useful, they would not provide sufficient corrective information regarding the client's principal feared outcome. Accordingly, the client would fail to learn that the act of vomiting itself is acceptably safe and tolerable (albeit unpleasant) and might continue to postpone her plans for starting a family.

Exposure therapists can take heart in the realization that, by definition, individuals with anxiety disorders are *already* experiencing significant anxiety symptoms in their daily lives. As such, the experience of high anxiety during exposure tasks is not novel and in most cases is likely to be no more intolerable or dangerous than the anxiety symptoms clients are used to dealing with from time to time. It is also useful for therapists to remember that despite its distressing and sometimes dramatic nature, anxiety is an adaptive response that is designed to protect us from harm. It is rather absurd to suppose that evolution equipped humans with an alarm system for dealing with threats to our survival that is, itself, dangerous.

A very large body of research attests to the tolerability, safety, and efficacy of exposure therapy. This treatment is not reliably associated with increased risk of client attrition relative to other psychotherapies, and symptom exacerbation is rare, temporary if it occurs at all, and unrelated to prognosis (Olatunji et al., 2009). These observations aside, it is undeniable that the unique requirements of exposure therapy sometimes place clients at greater emotional and/or physical risks than many traditional forms of verbal psychotherapy. For example, exposure can involve the remote but real potential for harm when clients handle snakes or touch "contaminated" objects such as garbage cans. Although when conducted properly these exercises involve acceptably low levels of risk, exposure therapists must carefully consider the client's safety when designing and implementing exposure practices. Strategies for minimizing risk such as negotiating informed consent, determining acceptable levels of safety during exposure tasks, and dealing with negative outcomes are reviewed by Olatunji et al. (2009).

Exposure therapy is harmful to the therapist. This treatment is often viewed as posing a number of risks to the therapist. Concerns about one's ability to tolerate the client's negative affect represent a significant therapist barrier to the dissemination of exposure therapy (Litz, 2002). This concern may be especially likely to arise in the context of imaginal exposure for PTSD, during which the therapist listens to detailed accounts of often horrifying trauma narratives. Some therapists believe that such experiences can be "vicariously traumatizing" and produce persistent, negative psychological effects. Other practitioners may question their ability to tolerate their own negative affect during particularly intense exposure sessions.

Clinicians who believe exposure to be inhumane, intolerably aversive, or potentially dangerous may also worry about the legal risks associated with the use of this treatment. Boundary crossings associated with exposure sessions conducted outside the office might be viewed as paving the way for an inappropriate dual relationship. Therapists may believe that especially anxiety-provoking exposure tasks increase the risk of malpractice lawsuits from clients who may decompensate and/or experience harm in other ways from the treatment.

The following case examples help to illustrate the manner in which negative therapist beliefs about exposure may affect its delivery:

Mr. L, age 50, was repeatedly sexually abused in his early teens by a 16-year-old boy. He is ashamed of his failure to fight off the perpetrator and frequently bursts into tears when discussing his sexual abuse history. He attempts to suppress memories of the abuse and avoids external cues associated with the trauma.

Ms. W, age 23, experiences frequent, unexpected panic attacks during which she fears that she will suffocate and die. She requires the presence of a trusted friend or family member when leaving home and avoids traveling more than a few miles from a local hospital next to her home where she frequents the emergency room.

The use of imaginal exposure would doubtless evoke substantial distress for Mr. L., and a therapist concerned about his or her own ability to tolerate the client's anxiety might elect not to use this procedure. Alternatively, the therapist might attempt to minimize the client's anxiety by implementing imaginal exposure in a client self-directed manner or by allowing the client to refrain from elaborating on the most distressing elements of the trauma narratives during therapy sessions. Therapists who attempt to protect themselves from emotional distress during exposure run the risk of depriving clients from fully overcoming their pathological anxiety. In the case of Mr. L., failure to conduct prolonged imaginal exposure might prevent him from emotionally processing the full range of memories associated with his history of sexual abuse. The client's failure to habituate to particularly distressing traumatic memories would likely maintain his avoidance and belief that he is unable to tolerate the distress associated with recalling memories of his trauma history.

In vivo exposure for Ms. W. might involve traveling increasingly further outside her "safe zone" around the hospital. An obvious exposure task would be for her to drive outside of town to a rural area where immediate help would be unavailable in the event of a panic attack. A therapist who is overly concerned with the ethical "slippery slope" of conducting an out-of-the-office exposure with a client of the opposite sex might assign this task as homework rather than risking the appearance of impropriety by accompanying the client. Given that the client is currently unable to perform this task on her own, the failure to conduct this exposure in a therapist-assisted manner increases the risk that the exposure would result in a negative outcome, such as the client prematurely terminating the task due to high anxiety. Such an outcome might decrease the client's self-efficacy and foster the perception that she will not be able to fully benefit from exposure therapy.

Practitioners who lack the ability to tolerate their own distress during exposure therapy sessions are ill equipped to provide this treatment in a competent fashion. We agree with Gunter and Whittal's (2010) contention that "trust in the intervention, comfort in administering it, and confidence in one's ability to address client reactions to exposure treatment are all vital prerequisites to the use of exposure in clinical practice" (p. 196). Exposure therapists must strike a balance between empathy for their client's distress and maintaining a professional distance that allows for therapeutic, professional responses (Foa & Rothbaum, 1998). This balance is difficult to maintain in some instances, as when trauma victims recount particularly terrible experiences during imaginal exposure. However, even the most compassionate therapist must remember that it is his or her job to assist the client in recovery from clinical anxiety, and losing emotional control or withholding exposure therapy is incompatible with this goal. Indeed, clients draw strength from the therapist's outward expressions of confidence in their ability to tolerate the distress associated with particularly difficult exposures. An important aspect of one's development as an exposure therapist involves learning to cope with and accept the emotional distress clients exhibit during particularly challenging therapeutic tasks. From time to time, unburdening oneself by talking to colleagues, or seeking distraction in the form of other professional or personal activities, is useful to cope with the unique demands of exposure therapy.

Therapists who believe that exposure therapy poses a risk management problem would benefit from the knowledge that the anxiety evoked during exposure sessions is generally tolerable,

harmless, and no different from what clients are already experiencing. Reassuringly, there is no evidence to suggest that exposure is associated with an increased risk of litigation. Richard and Gloster (2007) searched the legal record for court cases involving exposure therapy. Their exhaustive search criteria did not reveal a single instance of litigation related to this treatment. Similarly, none of the 84 members of the Anxiety Disorders Association of America surveyed by Richard and Gloster reported knowledge of any legal action or ethics complaints regarding exposure. This survey approach, however, cannot rule out the possibility that relevant complaints have been filed but dismissed or settled out of court. Lastly, we note that malpractice insurance carriers appear unconcerned with the use of exposure. Malpractice rates are much lower for psychotherapy than for many other healthcare providers, and we are not aware of any insurance companies that charge higher premiums for therapists who provide exposure therapy. In summary, the available evidence suggests that exposure is acceptably safe and tolerable and that it carries little risk of actively harming clients (or their therapists).

Conclusions and Future Directions

Exposure-based CBT is the most evidence-based psychological treatment for pathological anxiety. Unfortunately, clients suffering from anxiety disorders are often unable to access this intervention owing to the widespread failure to disseminate it to practitioners. This chapter reviews the numerous and formidable barriers that prevent mental health practitioners from utilizing exposure therapy. However, the poor utilization of exposure is only part of the story, as a host of additional barriers may serve to reduce the efficacy of exposure therapy even when it is delivered by trained therapists.

Exposure therapy is a uniquely difficult treatment to disseminate. Strong, negative beliefs about this intervention are pervasive among mental health professionals. Despite its well-established efficacy, exposure is widely considered to be unethical, harmful, and intolerable for clients and therapists alike. Because of such beliefs, efforts to disseminate exposure therapy to practitioners likely require more than simple instruction in the nuts and bolts of the application of exposure techniques.

Clinical scientists continue to strive to improve the efficacy (e.g., Rapee, Gaston, & Abbott, 2009) and acceptability (e.g., Rachman, Radomsky, & Shafran, 2008) of exposure therapy and will doubtless do so for the foreseeable future. However, the evidence base for existing exposure-based cognitive-behavioral therapies is now sufficiently well developed that efforts at dissemination are proceeding in earnest (McHugh & Barlow, 2010). In the United States, the most prominent example is the widespread effort within the Veteran's Health Administration to train therapists in evidence-based psychotherapies for PTSD, including prolonged exposure therapy (Foa, Hembree, & Rothbaum, 2007). The Improving Access to Psychological Therapies (IAPT) program in the United Kingdom is the most extensive dissemination effort in the world. In 2010, the Department of Health invested approximately £300 million (approximately $435 million U.S. dollars) to train healthcare professionals in evidence-based treatments for depression and anxiety, and early clinical outcomes are impressive (Clark et al., 2009).

Empirical research on the nature and modification of therapist barriers to exposure has the potential to improve efforts to disseminate this treatment to mental health professionals. Future studies might address the following questions: (a) What are the negative beliefs about exposure therapy held by therapists? (b) How do such beliefs affect whether or not, and how, therapists utilize exposure techniques in their practice? (c) How do negative therapist beliefs about exposure affect client outcomes? (d) What training strategies are most effective in modifying negative beliefs about exposure? (e) To what extent is the success of efforts to train practitioners in the competent delivery of exposure therapy contingent upon modification of negative beliefs about this treatment? Efforts to develop measurement tools for assessing therapist beliefs about exposure are under way, and researchers are beginning

to tackle these questions in a systematic manner (e.g., Harned, Dimeff, Woodcock, & Skutch, 2011). Despite a host of practical and ideological barriers, substantial progress is being made in the dissemination of exposure-based treatments for anxiety disorders. We hope that the information presented in this chapter will encourage additional progress in the important effort to increase the availability of exposure-based CBT to clients with anxiety disorders.

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24 Harnessing the Web: Internet and Self-Help Therapy for People with Obsessive–Compulsive Disorder and Posttraumatic Stress Disorder

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People with an acute eye infection, a broken leg, or abdominal pain will seek help from a physician usually without much hesitation. In contrast, the threshold to consult a treatment provider for disorders labeled as psychiatric or psychological is much higher. Often, many years pass until professional help is sought and for some patients traditional mental health care specialists (i.e., a psychologist and/or psychiatrist) are not even the first choice. Before turning to the primary focus of this chapter—self-help and Internet therapy for obsessive-compulsive disorder (OCD) and posttraumatic stress disorder (PTSD)—we will briefly summarize why and to what extent people with mental disorders refrain from or are deprived of efficacious therapy. This section is meant to highlight the necessity for alternative approaches to help to "treat the untreated." We will begin with a review of more general reservations of many patients against the health care system and then consider specific illness-related reasons for treatment abstinence. Many of the treatment barriers discussed have been identified across different cultures and nations, and as we hope to demonstrate, Internet and self-help therapy may provide an effective and timely solution to some of the current challenges faced by the psychiatric-psychological help system.

Reasons for Not Seeking Psychological or Psychiatric Help

Most individuals with psychiatric disorders do not receive psychological or psychiatric treatment because treatment is either not available, denied, or not competently delivered. Moreover, as we will discuss in greater detail below, a large subgroup of patients choose not to pursue treatment options. Further, the conventional healthcare system is increasingly challenged by alternative medicine (AM). A recent German study reported that 37% of psychiatric patients had visited a *healing* or *alternative practitioner* ("Heilpraktiker") before their hospital stay (Demling, Neubauer, Luderer, & Worthmuller, 2002). In the United States, 21% of the people with mental disorders had sought alternative or complementary medicine during the last 12 months (Unützer et al., 2000), and up to 50% of the general population in English-speaking countries consult therapists specialized in AM (Silenzio, 2002). Many patients do this in secrecy, fearing to be judged as "traitors" by

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their physicians (White, 2000). Importantly, large proportions of patients with mental illness as well as the general public are very critical of pharmacotherapy, which is often (mis-)perceived as the standard, first-line intervention of the psychiatric/psychological health care system (Angermeyer & Matschinger, 1996; Lauber, Nordt, & Rossler, 2005; Moritz, Peters, Karow, Deljkovic, & Naber, 2009). Approximately 60% of the psychiatric population shares the opinion that conventional (chemical) medication should only be taken when herbal remedies are not effective (Demling et al., 2002).

Mind the (Treatment) Gap!

Perhaps the most common reason why psychiatric disorders are not treated is lack of money and/or poor availability of appropriate interventions. In fact, a large proportion of people are deprived of any medical help, regardless if it is conventional or alternative in nature. Around 70% of the world population has access to less than 1 psychiatrist per 100,000 inhabitants. In some African regions, the ratio drops to 1 per 5,000,000 in contrast to 0.5–1 per 10,000 in European countries (Klecha, Barke, & Gureje, 2004). In many developing countries and those struck by war, no psychiatric system exists at all. Many of these countries lack the most basic medical care. Even in countries that have established a working health care system, individuals living in remote areas may not have access to treatment providers (Wootton & Titov, 2010).

The World Health Organization (WHO) has estimated that only 40% of individuals with OCD actually receive professional care (Kohn, Saxena, Levav, & Saraceno, 2004). The corresponding rates for PTSD are varying across different populations (combat veterans, victims of sexual violence, crime victims) and range between 25 and 40% (e.g., Elhai, North, & Frueh, 2005; Hoge, Auchterlonie, & Milliken, 2006). For these populations, shame and stigma are predominant motives for treatment abstinence. In a recent survey, a substantial subgroup of OCD patients (Hauschildt, Jelinek, Randjbar, Hottenrott, & Moritz, 2010) disclosed that they fear becoming "mad" or "dangerous," and two-thirds were ashamed of their mental illness. Accordingly, a number of patients are afraid to be questioned by the police or may lose their children if they disclose aggressive obsessions. A related study in the United States (Marques et al., 2010) examined the extent of treatment utilization and barriers to treatment in a sample of 175 individuals recruited over the Internet with (self-reported) OCD. While the rates of treatment utilization were a bit higher than those reported by the WHO, only 60% of the participants were currently receiving treatment for their OCD symptoms. The following barriers to treatment were identified: cost of treatment, lack of insurance coverage, shame, and doubt that treatment would be effective. Importantly, many participants received treatments other than the gold-standard interventions. This was particularly true for psychotherapy; the majority of participants received mere "talk therapy" rather than the empirically validated cognitive behavioral therapy.

The Use of the Internet and Self-Help Books for (Self-)Treatment

There is growing evidence that many people not actively engaged in face-to-face treatment look for health/treatment information on the Internet, engage in Internet networks devoted to their problems, or turn to self-help books. A Norwegian study estimates that in 2010, 84% of the Norwegian population has been using the Internet for health purposes (Wangberg, Andreassen, Kummervold, Wynn, & Sørensen, 2009), which is roughly equivalent with data obtained in Germany (Otto & Eichenberg, 2010). Of 2,411 German people from the general population, approximately 90% appraised the Internet as potentially useful for seeking health information and around 40% would consider communicating with people with similar problems in an Internet forum (Eichenberg, Blokus, & Brähler, 2010). According to the German survey by Otto and Eichenberg (2010), almost every fourth patient

informs her- or himself on the Internet before seeing a clinician (22.95%), and 89% of the physicians have experience with patients who reference Internet-based health information during treatment (Masters, 2008). As the sections below will demonstrate, the Internet is a vital tool not only for the assessment but also the delivery of treatment via email or online tools.

In addition to the Internet, a more conventional way to obtain health/treatment information is by the means of books. A total of 75% of the participants with OCD affirmed that they had read at least one self-help book on OCD (Moritz, Wess, Treszl & Jelinek, 2011). Bibliotherapy is not only popular with patients but is also endorsed by 85% of clinicians as an adjunct to face-to-face treatment. A similar number of clinicians (82%) recommends self-help groups (Harwood & L'Abate, 2009). The development and use of self-help bibliotherapy is not without problems. Unlike new clinical interventions, which necessitate approval by ethical review boards and are in many cases only employed following large-scale empirical support and their inclusion into national guidelines, self-help books can be published by anyone. A conventional publisher is not even necessary as self- or electronic publishing facilities are widely available. The advertisement of these books is also not rigorously regulated. Most online bookstores have established a para-academic peer-review system where readers or lay people provide reviews. These endorsements are often prone to biases and may perhaps even blatantly mislead the reader. Harwood and L'Abate (2009) write "a problem with commercially available self-help materials is that systematic evaluation of their effectiveness is not easy to obtain" (p. 63). A recent evaluation raised grave concerns even against self-help "best sellers." The assessment by Shaked (2005) on ten contemporary and popular personal self-help books published between 1997 and 2002 arrived at the conclusion that most of these books lack empirical support to a moderate extent. Another study asked expert psychologists to assess 50 top-selling self-help books for anxiety, depressive, and trauma-related disorders (Redding, Herbert, Forman, & Gaudiano, 2008). Criteria for evaluation included overall usefulness, grounding in psychological science, the extent to which it offers reasonable expectations, and whether it offers specific guidance for implementing the self-help techniques or offers potentially harmful advice. Results demonstrated that five OCD books were identified as "high quality" and were among the "top ten," whereas books on PTSD received mostly lower marks and one was even identified as potentially harmful.

Some therapists have actively responded to this new trend of patients using self-help bibliotherapy materials and searching the Internet to acquire knowledge. As cited above, more than four out of five clinicians recommend bibliotherapy or the participation in self-help groups for their patients (cited after Harwood & L'Abate, 2009). According to a German survey (Eichenberg et al., 2010), most therapists offer email communication to their patients (92.3%), for example, for the purpose of crisis intervention or information exchange. Given the ubiquitous nature of email, it may be surprising that in reality a factual exchange takes place only in every 15th patient. It should also be noted that most survey respondents regarded email usage merely as an adjunct to direct intervention (i.e., not as a stand-alone intervention). There also remains a substantial minority of psychotherapists who entirely reject online communication in a counseling (22%) or therapeutic (45%) context, largely due to problems associated with data privacy and safety.

Studies Conducted over the Internet: Pros and Cons

Internet surveys are an innovative mechanism that allows the research community to examine the effectiveness of self-help books and Internet treatments and also provide a means by which a currently underserved population can be reached. Numerous sophisticated online tools exist that enable researchers to administer questionnaires and collect data over the Internet (e.g., http://www.unipark.info/ or http://www.limesurvey.org/). The section below on "Bibliotherapy in OCD" provides several examples for this kind of

Table 24.1 Arguments for and against online studies to evaluate Internet therapy and self-help books

Pro	Con
Provides help at a low threshold: especially valuable for individuals currently on a waitlist for treatment or who are at present unwilling or unable to obtain treatment (no therapies offered because of war, third world country, political/cultural paradigm [e.g., psychological disorder erroneously considered as religious sin], no health insurance)	Diagnostic status is hard to verify: expert ratings are generally favored over self-help instruments
Economic: far less costly than standard studies	Psychotherapy by a psychotherapist is likely more helpful than an Internet therapy (for exceptions see Carlbring et al., 2005; Kiropoulos et al., 2008)
Effective option to assess the efficacy of self-help manuals	Crisis intervention is hard to implement
Reliability and completion rates are satisfactory when certain precautions are taken (e.g., incentives, reminders)	Completion rates can be very low; psychometric properties of many paper-and-pencil tests are largely unknown for Internet administration
	Multiple log-ons cannot entirely be prevented in the event that *cookies* are deleted

research. Table 24.1 contrasts a number of pros and cons for conducting this type of research. A strong argument in favor of Internet studies is that they are economic, from both a monetary and a time perspective. Internet studies are particularly useful when assessing the validity of alternative medical approaches that would unlikely receive external funding by prominent research organizations such as the National Institute of Mental Health, the German Research Foundation, or the Wellcome Institute. Internet studies can also provide an informative basis for which book or self-help technique should be recommended to patients after discharge or patients on a waitlist. It often takes 10 years until OCD patients seek professional help for their problems, and there can be an additional lag of 6 or more years until the diagnosis is correctly determined and appropriate treatment is initiated (Blanco et al., 2006; Hollander et al., 1996; Pinto, Mancebo, Eisen, Pagano, & Rasmussen, 2006). From this perspective, self-help literature represents a useful mechanism to potentially bridge the treatment gap and may even enhance motivation for face-to-face treatment.

The most poignant problems and challenges to conducting Internet studies lie in inherent methodological issues. Internet studies assessing the efficacy of psychological interventions (e.g., email, Internet-based therapy, self-help bibliotherapy, guided self-help) often have low completion rates (e.g., Meyer et al., 2009). There are also concerns about the validity of diagnoses and the appropriate measures that should be adopted in case of emergencies/crisis. Another concern about the online evaluation of symptoms and treatment responses relates to the psychometric properties of self-report instruments used in Internet studies. Traditionally, clinician ratings are regarded as the gold standard; such direct interviews are often not feasible in Internet studies. A growing number of studies, however, assert satisfactory to very good psychometric properties of scales administered over the Internet (e.g., Moritz, Jelinek, Hauschildt, & Naber, 2010). For example, in one of our Internet studies, which will be reviewed in greater detail below, a self-report version of the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) (Goodman et al., 1989), the major outcome instrument in OCD research, yielded a 4-week retest reliability of $r = 0.8$ and was highly correlated ($r = 0.6$) with the Obsessive-Compulsive Inventory-Revised (OCI-R)

(Foa et al., 2002), another OCD scale (Moritz, Jelinek, et al., 2010). A study by Coles, Cook, and Blake (2007) directly compared paper vs. Internet administration of the Obsessive-Compulsive Inventory (OCI) (Foa, Kozak, Salkovskis, Coles, & Amir, 1998). Results assert that the two formats were virtually equivalent.

Empirical Studies on Internet and Self-Help Therapy for OCD and PTSD

As mentioned above, a large proportion of patients in need of treatment never sees the inside of a psychiatric hospital or specialist's office. In OCD, this population has been estimated at roughly 60% (Kohn et al., 2004). In other words, only 40% of those with clinically impairing OCD symptoms actually seek treatment, and of the minority who do, most wait an average of 10 years or more to ultimately seek a treatment provider (Marques et al., 2010). In PTSD the extent of the problem is comparable (Hoge et al., 2006) and so are the reasons, particularly perceived stigma and poor treatment availability (Pietrzak, Johnson, Goldstein, Malley, & Southwick, 2009). In the subsequent subsections, the available research on self-help and Internet therapy for OCD and PTSD is summarized. The closing section will provide a summary and some recommendations for future research.

Obsessive-Compulsive Disorder

The literature on Internet therapy and self-help interventions for OCD prior to 2006 was first summarized by Mataix-Cols and Marks (2006). In their review considering case studies, open and randomized controlled trials (RCTs) of bibliotherapy, self-help groups, telecare, and computer-aided self-help for OCD, they expressed guarded optimism for this novel (nondirective) approach and recommended a stepped care model. Whereas for less complex cases self-help or bibliotherapy with brief help-line live advice may suffice, for more severely disturbed patients, intensive face-to-face guidance was deemed indispensable. Harwood and L'Abate (2009) provided a more critical appraisal: "In general, across a variety of self-help approaches for the treatment of OCD, good outcomes appear to occur in less than 50% of patients" (p. 65). However, it should be stressed that clinical studies often struggle with similarly low response rates when dropout is included. Moreover, as Harwood and L'Abate (2009) recognized, low outcome may not relate to self-help per se but rather to the specific technique or intervention employed.

Most studies summarized below reported symptom relief under self-help or Internet therapy, although the magnitude of symptom reduction tended to be lower than that expected by standard face-to-face therapy. However, Mataix-Cols and Marks (2006) noted that "making effective self-treatment guidance available may increase the number of patients being helped" (p. 75)—even if no optimal level of symptom reduction is achieved. At the same time, a methodological caveat lies in the fact that virtually all self-help studies conducted so far included an element of therapeutic guidance. Pre-post assessments in many studies summarized below were made in a clinical environment which likely scared off some potential participants reluctant to seek treatment because of anxiety (fear to be treated against one's will because of severe psychopathology and "public danger"), shame (e.g., to be judged as "pervert" due to aggressive and sexual obsessions), and avoidance (e.g., fear of an in-depth confrontation with traumatic memories). Since the review by Mataix-Cols and Marks (2006), new studies have been conducted. A growing number did not include direct therapeutic support and thus represent a more unconfounded assessment for the effectiveness of self-help.

Computer- and Phone-Assisted Therapy in OCD

The implementation of computer-aided therapy for OCD dates back to the 1980s, when Baer and colleagues developed a computer program (OC-CHECK) to enhance patients' compliance

with behavioral therapy (Baer, Minichiello, & Jenike, 1987). Each of the two patients who participated in the study was provided with two portable computers: a laptop that was used at home familiarizing the patients with the procedure and a smaller, calculator-size computer to carry with them outside of the home. The program was designed to help patients refrain from ritualizing, by asking them to resist the urge for 3 min. Furthermore, OC-CHECK stored information about the date, time, intensity, and frequency of urges and checking rituals per day. Both patients reduced their checking rituals significantly when the computers were used in conjunction with standard behavioral therapy, and in turn, rituals increased when participants stopped using the computers. Still, two additional study patients declined to use this approach. One clear limitation consists in the very small sample size questioning the validity of the results. These promising findings should be therefore interpreted with caution.

Kirkby et al. (2000) tested the efficacy of a human–computer interaction (HCI; see also Clark, Kirkby, Daniels, & Marks, 1998). Thirteen subjects with OCD (seven mainly washers and six mainly checkers) completed three weekly 45-min computer-administered treatment sessions consisting of an exposure treatment program, whereby the principles of exposure therapy and ritual prevention were conveyed by tracking an interactive person with contamination obsessions and washing rituals. The participants' task was to direct the figure through a dirt exposure with ritual prevention (ERP) to reduce the figure's anxiety level and its urge to wash. Participants were instructed to imagine they were the person on the computer screen doing the ERPs. During the execution of the program, HCIs were recorded to describe the participants' behavior. None of the subjects had attended behavioral treatment for OCD prior to the study. Across the three sessions, participants increased their vicarious exposure behavior and decreased their washing behavior. The clinical improvement was greater for subjects who performed more enactments of hand washing in the first session. Scores from the OCD subjects fell modestly but significantly on the Padua Inventory (PI) (Sanavio, 1988) and the Beck Depression Inventory (BDI), but improvement on the Y-BOCS was nonsignificant.

A recent study tested the effectiveness of an existing online-group treatment for compulsive hoarding[1] (Muroff, Steketee, Himle, & Frost, 2010). The program requires members to take active steps to reduce compulsive hoarding and to post their activities on a regular basis. The web training is based on CBT methods. Members have electronic access to mental health information, educational resources on hoarding, and to a chat group. The sample consisted of forum members and a natural wait-list group. Data were collected via the Internet at five time points over the course of 1 year (every 3 months). Results indicated that recent- as well as long-term members improved significantly over 6 months. Long-term members reported fewer hoarding symptoms than recent members possibly suggesting benefits from membership over time. In contrast, wait-list members improved somewhat but not significantly on most measures. Differences between recent- and wait-list groups did not reach significance. Less posting activity was associated with greater hoarding severity.

Most studies to date have been conducted with BT Steps (now named OCFighter), whereby BT stands for behavior therapy. The BT Steps system is a fully interactive computer program accessed remotely via touch-tone telephone using (phone-) interactive-voice-response (IVR) technology. Patients obtain a manual, an ID number, a personal password, and an IVR access at home. It guides the patient though an individually tailored self-help for OCD. BT Steps contained nine steps, whereby the first four steps are devoted to self-assessment.

An Anglo-American research group (Marks et al., 1998) was the first to test BT Steps. In total, 63 subjects participated: 40 from the UK and the USA in study 1 and 23 from the UK in study 2. At baseline, Y-BOCS, the Hamilton Depression

[1] Hoarding will not be classified as an obsessive-compulsive disorder in the DSM-V suggesting that hoarding might differ substantially in form, aetiology, and treatment from other presentations of OCD.

Rating Scale (HDRS) as well as the Work/Social Adjustment Scale (WSAS) were assessed. Additionally, in study 2, patients rated understanding of exposure therapy and their motivation to use BT Steps. After completion of BT Steps, all participants were asked to complete the same questionnaires as before. Symptom change was rated using a single-item version of the Patient Global Improvement (PGI). The two studies revealed similar outcomes: 84% of the 63 subjects completed the self-assessment component and 43% (study 1) to 48% (study 2) of the participants proceeded to the self-treatment part. Improvement only occurred when participants went on to perform self-exposure. If participants completed more than one ERP task, they improved significantly on Y-BOCS and WSAS scores. 71% of the participants rated themselves as responders (PGI). Performing more ERPs was associated with greater gains. In study 2, high motivation at baseline and rapid completion of the self-assessment significantly predicted lower symptoms at posttreatment.

In a second study on BT Steps (Bachofen et al., 1999), 21 patients initially participated, whereby 16 completed self-assessment over a mean of 21 days and rated themselves at baseline and at the end of BT Steps on the Y-BOCS, HDRS, and on the WSAS. At the end of BT Steps, the 1-item PGI scale was completed along with scales about motivation. Patients who rated themselves as more motivated at baseline improved significantly more in the course of the intervention on the Y-BOCS total, Y-BOCS obsessions, and WSAS social leisure item scores. Baseline motivation was also higher in patients who went on to do two or more ERP sessions than in those who did not. The outcomes across the two open studies are similar, but the patients in the present study progressed more rapidly.

Kenwright, Marks, Graham, Franses, and Mataix-Cols (2005) studied the impact of phone support from a clinician (scheduled support vs. requested support) for the compliance and outcome of BT Steps. A total of 44 therapy resistant OCD patients from around the UK used the program over 17 weeks. All participants received the rationale of BT Steps. Phone support from a clinician could include either nine scheduled, therapist-initiated phone calls ($n=22$) or took place only on request from the patients ($n=22$). The latter group was instructed to call the clinic if they had problems working through BT Steps. At baseline, patients reported chronic OCD symptoms (mean duration = 16 years; mean Y-BOCS = 26) and moderate depressive symptoms. After the intervention, both groups improved significantly in OCD symptoms and disability (WSAS). However, significantly fewer scheduled-support patients dropped out (2 vs. 9) and for this group improvement was significantly greater on the Y-BOCS and the WSAS total score. Furthermore, scheduled-support patients completed more homework sessions of self-exposure and ritual prevention (95% vs. 57%). Total support time was 76 min per patient for the scheduled patients and 16 min for the on-demand group. In summary, patients' compliance and improvement in BT Steps was enhanced by providing proactive phone calls from a clinician.

In another study, Greist et al. (2002) assessed the efficacy of BT Steps. The participants consisted of 218 OCD patients meeting DSM-IV-TR criteria who were randomly assigned to one of the following treatment options: (1) BT Steps, (2) clinician-guided behavior therapy, and (3) systematic progressive muscle relaxation (PMR; manual-guided). All subjects went through an assessment on which different measurements were administered. Data was collected on the Y-BOCS, Patient and Clinical Global Impressions scales (PGI & CGI), WSAS, and the HDRS. The treatment duration for all groups took 10 weeks. Treatment outcome revealed that systematic relaxation therapy was ineffective to help patients with OCD (see also Moritz et al., 2010). OCD subjects obtaining a computer-guided therapy showed significant improvement on the Y-BOCS, the CGI, and PGI scales, although clinician-guided treatment was more effective. The efficacy of the computer-guided treatment increased with greater use of the computer and higher frequency of instructed self-exposure.

Marks et al. (2003) investigated the efficacy of four different self-help programs for depression (Cope), phobia/panic (FearFighter), general

anxiety (Balance), and OCD (BT Steps). Different self-ratings were collected at pre- and posttreatment. Dependent on the patient's diagnosis, they either received the Fear Questionnaire (for phobia/panic), the BDI (for depression), the Beck Anxiety Inventory (for generalized anxiety disorder), or the Y-BOCS (OCD). Out of 355 referrals, 210 were screened and identified as eligible, and 108 eligibles completed the computer-aided CBT. The findings showed a statistically significant improvement in three of four systems (Fear Fighter, Cope, and BT Steps). The completers needed a per-patient overall mean support of only about 1 h over 12 weeks by a clinician (CBT: at least 8 h per clinician). In addition, the patients were satisfied with their computer-aided CBT. The authors conclude that computer-aided self-help could be a "clinician extender" shortening the patient time per clinician and thus reducing the costs for CBT.

With the exception of the final study, the literature on BT Steps has been reviewed by Tumur, Kaltenthaler, Ferriter, Beverley, and Parry (2007). They conclude that BT Steps may broaden the access to CBT and potentially save therapist's time which in turn may shorten waiting periods for OCD patients. However, they note potential problems with the current empirical basis in that a publication bias cannot be ruled out and only two investigations were planned as RCTs with adequate quality. The pilot studies are also plagued by high dropout rates. Overall, we agree with the evaluation of Tumur et al. "that BT Steps is an important treatment strategy that could have an important role in the future of psychological treatment" (p. 201).

A yet unpublished wait-list controlled randomized trial (Storch et al., 2011) tested the efficacy of a webcam-delivered CBT program (W-CBT) for children and adolescents with OCD. Following pre-assessment, the 31 children and adolescents (7–16 years, 12 female) met with their therapist once in order to build rapport. Participants assigned to the W-CBT group ($n = 16$) received fourteen 60–90-min sessions of family-based CBT over 12 weeks. Treatment was individualized to symptom profile and developmental level. Reassessment took place within 1 week posttreatment and at 3-month follow-up. The wait-list control group ($n = 15$) was reassessed after 4 weeks (no follow-up). Outcome was assessed with the Clinical Global Impression Scales (CGI-Severity/CGI-Improvement), the CY-BOCS, the ADIS-IV-C/P, the Child Obsessive-Compulsive Impact Scale Child and Parent (COIS-C/P), the Multidimensional Anxiety Scale for Children (MASC), the Children's Depression Inventory (CDI), Family Accommodation, and a Satisfaction with Services Scale. When controlling for baseline group differences, W-CBT was superior on all primary outcome measures (CY-BOCS, CGI, remission status) with very large effect sizes ($d \geq 1.36$). Average CY-BOCS reduction was 56.1% for W-CBT (waitlist—12.9%). Eighty-one percent of the W-CBT participants were considered treatment responders (waitlist—13%) and 56% met remission criteria (waitlist—13%). Despite a slight increase of symptom severity over time, gains were generally maintained in the follow-up. Participants improved significantly in the COIS-C and family accommodation. Improvements in the MASC and the CDI did not reach significance.

Bibliotherapy in OCD

This section is devoted to the evaluation of bibliotherapy for OCD, whereby most studies have been conducted over the Internet. The first study of this kind was by Fritzler, Hecker, and Losee (1997) who explored the efficacy of self-directed treatment in OCD. Of 12 patients who had initially participated, 6 males and 3 females with primary checking, cleaning, cleaning/checking, and hoarding problems eventually completed the treatment and met with therapists for five therapy sessions over a 12-week period. Participants were provided a self-help book (*When once is not enough*; Steketee & White, 1990) on which the treatment was founded. Among other topics, it explained how to implement self-directed exposure with response prevention. The self-report version of the Y-BOCS was chosen as the primary outcome measure. Results demonstrated statistically

significant improvement for nine patients—three met criteria for clinical significance of improvement. The authors concluded that bibliotherapy with brief therapist intervention may be the first choice of intervention for people with OCD but also hypothesized that therapists' professional experience may be related to outcome. Furthermore, they speculated that this intervention may be less successful for severely impaired patients and presumably also for hoarders.

A study conducted by Tolin et al. (2007) directly compared self-administered (guided by the manual Stop Obsessing!) and therapist-administered ERP (guided by an experienced doctoral-level psychologist) in a RCT with 41 OCD patients. Patients had a history of at least one current or previous adequate psychopharmacological trial. Overall, the groups were comparable according to baseline characteristics, although the ERP group showed somewhat more comorbid diagnoses and was a little older (40 vs. 34 years). Clinical assessments were made at pretreatment and posttreatment, and three additional follow-up assessments were conducted 1, 3, and 6 months later. Whereas therapist-administered ERP was superior to self-help (35% vs. 17% improvement from pre- to posttreatment according to ITT), self-help also exerted significant gains over time. From pretreatment to the 6-month follow-up, the therapist-administered ERP group showed an improvement of 8.19 points on the Y-BOCS total score. In the self-administered group the score fell 3.3 points during this time. The authors conclude that although improvement seen under self-administered ERP was lower than that of conventionally guided ERP, self-administered ERP represents a (cost-)effective intervention for a subgroup of patients.

A study published by one of the authors (Moritz, Jelinek, Klinge, & Naber, 2007) investigated the efficacy of association splitting, a technique aimed at the reduction of obsessive thoughts (Moritz & Jelinek, 2007). The technique, which is available in different languages at no cost at www.uke.de/assoziationsspaltung, teaches patients to generate and associate novel cognitions to fear-related OCD cognitions. New associations should either be neutral or positive and in no overt semantic relationship with OCD-related concerns. The concept draws upon a cognitive phenomenon termed the fan effect: The more associations exist for a given cognition, the less the weight of each single association. For example, a patient who is preoccupied with the number "13"—which for him or her solely means or predicts disaster—should learn that "13" has alternative neutral meanings. For example, that the 13th element of the periodic system is aluminum, the USA was initially formed with 13 states and that some prominent sports players have the number 13 on their jerseys. Novel associations may not extinguish the linkage between a certain cognition with OCD symptoms (i.e., worries, obsessive thoughts, compulsions) but may reduce the strength of the connection and thereby empower the subject to withstand or ignore obsessive urges. A core assumption of the model predicting that individuals with OCD process ambiguous words (e.g., homographs such as cancer) preferably in the context of the OC meaning (i.e., illness) and connect them to a lesser degree to other (neutral or positive) cognitions (e.g., animal) has been recently confirmed (Jelinek, Hottenrott, & Moritz, 2009).

For the evaluation study, a total of 38 people with a likely diagnosis of OCD were recruited over the Internet via online OCD self-help forums. Four weeks after the email dispatch of the association splitting manual, a reassessment was conducted. Pre- and post-assessments included the Maudsley Obsessive-Compulsive Inventory (MOCI; Hodgson & Rachman, 1977), the Y-BOCS, and the BDI. A retrospective rating showed that at least one-third of the subjects felt that the technique had decreased their symptoms. A more rigorous pre-post comparison asserted this for the Y-BOCS score. Depending whether a per protocol or intention to treat analysis was adopted, 33–42% of the participants fulfilled response criteria. As no follow-up was conducted and no comparison group was recruited, this result should be interpreted with caution. However, an experimental study, also conducted over the Internet, confirmed that patients familiar with the association splitting generated the least OC-related and negative associations to core

OCD words (Jelinek et al., 2009). A recent (Moritz & Jelinek, 2011) study in 46 participants with a likely diagnosis of OCD who were randomly allocated to either association splitting or a wait-list control also showed that our technique reduces OCD symptoms, especially obsessions, as well as depression in the range of a medium to large effect size.

We have learned several lessons from this pilot study. Our subsequent Internet studies now all employ either wait-list or active control participants. In another study (Moritz et al., 2011) the attention training technique (ATT; Wells, White, & Carter, 1997) was administered and tested against a wait-list condition. The ATT is aimed at intrusive thoughts and usually conveyed by a therapist. However, as it is simple to learn and patients can easily perform the technique on their own, we reformulated the original instructions as a self-help technique. Preferably, two sessions, each lasting 15 min, had to be performed each day (see also Fisher & Wells, 2009, pp. 97–100; Wells & Papageorgiou, 2004, pp. 266–267). In the first step, participants had to detect several distinct noises inside and outside a room. In step 2, attention should be focused for approximately 1 min on one of these noises only, before attention is switched to another noise while ignoring all others. In step 3, once a sound has captured full attention, an attention switch to another noise should be undertaken, whereby attention should switch from noises inside to noises outside the room back and forth. In the fourth and final step, the patient should contemplate all noises at the same time and count these. The ATT is an intuitive method for the treatment of OCD in view of neuropsychological findings (Greisberg & McKay, 2003; Külz, Hohagen, & Voderholzer, 2004) linking OCD to enhanced rigidity, perseveration, and poor executive functioning (however see Basso, Bornstein, Carona, & Morton, 2001; Moritz et al., 2001). For the study, an invitation was posted on OCD help forums and communicated via the web site of the German and Swiss OCD Societies. A total of 80 participants with OCD were recruited and either assigned to the ATT or a wait-list condition. Assessments were made at baseline and 4 weeks later. Groups performed similar at both time points on the self-report version of the Y-BOCS and the OCI-R. The lack of effect was mirrored by patients' retrospective ratings. The results speak against the efficacy of ATT as a stand-alone bibliotherapy approach for OCD, even for those who performed the technique regularly according to self-report. The present study demonstrates another potential advantage of Internet research over clinical studies. Clinical trials usually apply a "cocktail" of different approaches (ranging from psychopharmacological treatment to occupational therapy) making treatment effects "messy" and hard to attribute to single factors. Internet investigations can keep such confounds low.

Meridian tapping (MT) is a body-oriented alternative medical technique which among other psychological problems claims to *cure* anxiety disorders. It is aggressively promoted as an alternative treatment for all kinds of problems and disturbances. Some of its advocates report that at least 70 or even 97% of the patients are cured (Craig, 2003). Solid empirical evidence for its efficacy is scarce, and some studies that were seen as proof for its success by its propagators can in fact be interpreted differently (see Moritz, Aravena, et al., 2010). As the theoretical foundations of MT are refuted by many scientists (e.g., Gaudiano & Herbert, 2000), chances for public funding for a large-scale trial are limited. For the present study (Moritz, Aravena, et al., 2010), we therefore tested the efficacy of a published MT self-help approach for OCD (Raubart & Seebeck, 2008) against PMR via the Internet. After a baseline assessment using standard outcome scales (Y-BOCS, OCI-R, BDI short form), 70 participants likely suffering from OCD were randomly allocated to either MT or to PMR. Four weeks after the dispatch of the self-help manuals (including video demonstrations of the technique), participants were asked to take part in a post assessment involving the same instruments as before and a retrospective questionnaire. In retrospect, MT was deemed more helpful than PMR (39% vs. 19%). However, the more rigorous pre-post assessment yielded no evidence for a stronger decline of OCD

symptoms under MT on any of the psychometric measures. Importantly, the Y-BOCS scores did not even change substantially across time for both interventions. The present study thus stands in strong opposition to bold claims about the efficacy of MT.

Recently, our research group has developed an eclectic self-help manual entitled "My Metacognitive Training for OCD (myMCT)" (Moritz, Jelinek, et al., 2010). The myMCT aims at raising patients' awareness about cognitive biases that are broadly regarded as risk and maintenance factors of OCD. Among these are the six cognitive biases and beliefs proposed by the OCD working group (Obsessive Compulsive Cognitions Working Group, 1997; e.g., inflated responsibility, over-estimation of threat, perfectionism; 2001; 2003; 2005). In addition, the myMCT comprises self-developed techniques like association splitting (see above) or attention splitting. The myMCT also touches latent aggression which is frequently found in OCD patients in combination with over-moral attitudes (Moritz, Wahl, et al. 2009). The training was primarily intended for patients currently unable or unwilling to attend standard therapy. Via the recruitment channels sought for the prior studies, 86 individuals with a likely diagnosis of OCD were recruited over the Internet. Half of the participants were immediately sent the myMCT manual; the other half was allocated to a wait-list group. After 4 weeks, a reassessment was scheduled. The myMCT group showed significantly greater improvement for OCD symptoms according to the Y-BOCS total score compared with the wait-list group ($d = 0.63$), particularly for obsessions ($d = 0.69$). Medium to strong differences emerged for the OCI-R ($d = 0.70$). A significant but smaller effect was observed for the short form of the BDI ($d = 0.50$). Since this pilot study, the manual has been expanded and contains novel exercises on response prevention and self-esteem and is thus hoped to yield even stronger effects on compulsions and depression than the first version. The manual has been translated into English and is also available in Russian, Portuguese and German (Moritz, 2010).

Finally, we evaluated competitive memory training (COMET) which has shown some effectiveness in people with low self-esteem (e.g., Korrelboom, de Jong, Huijbrechts, & Daansen, 2009) but also severe psychiatric disorders, for example, OCD (Korrelboom, van der Gaag, Hendriks, Huijbrechts, & Berretty, 2008). In brief, the subject is instructed to blend obsessive thoughts with competing memories of a different modality. For example, if a subject is afraid that he could harm his own child, he learns to defuse the obsessive thoughts with real memories that stand in strong opposition (e.g., being gentle to one's child, reading a birthday card to the "best dad in the world"). The thoughts should also be attenuated by taking an incompatible (e.g., proud) posture. A total of 65 people with a likely diagnosis of OCD were recruited and randomly allocated to either the COMET group (39-page manual) or wait-list control. For the primary outcome, the Y-BOCS, no effects emerged neither for group nor time nor the interaction. For the BDI and the OCI-R, unspecific improvements occurred in both groups. While most subjects (80%) found the technique comprehensible, our study might not have been a fair test of the technique as the manual was rather long so that it cannot be excluded that subjects did not adopt the approach as intended. We are thus reluctant to draw firm conclusions as the method is quite complicated and originally conveyed by a therapist.

Posttraumatic Stress Disorder

Internet resources and interventions for PTSD have dramatically increased in the last decade. Web sites dedicated to information for trauma survivors are particularly prevalent and address a broad range of traumatic experiences, including sexual violence, fatal diseases, and natural disasters. Some of these informational web sites can be a valuable resource for trauma survivors. However, there are also examples that present biased and inaccurate information. One of the problems is that web sites lack consensually

defined criteria or universal certificates of approval, making it difficult for consumers to identify if a web site is run by a professional or a trustworthy organization vs. a lay person/organization conveying false information. In an analysis of 80 sites targeting trauma survivors, Bremner, Quinn, Quinn, and Veledar (2006) found that 42% of the web sites provided inaccurate or even harmful information. Only 18% cited scientific references for the information they provided, and 50% of the web sites were not authorized by mental health professionals.

Still, the Internet offers several characteristics that might be beneficial for trauma patients. One such characteristic is the anonymity with which individuals can participate in chat rooms, support groups, or even online interventions. Traumatic events are often associated with degrading and shameful experiences (Budden, 2009), which can give rise to guilt and self-blame (Kubany et al., 1996). These feelings may in turn be associated with a reduced readiness to seek therapeutic help in a conventional face-to-face setting. The Internet and its (visual) anonymity therefore may provide a comparably safe environment where patients can regulate and control the degree of intimacy they want to share, without the fear of real-life judgment, rejection, or devaluation. This mode of communication may reduce (feared) social risks and promotes the disclosure of painful and shameful thoughts.

A second helpful characteristic of the Internet is the ease of portability of information. From a public health perspective, technological interventions via the Internet facilitate mental health recovery. This aspect is particularly relevant following natural disasters or mass catastrophes (e.g., after the Tsunami, 2006), when immediate care for a large number of individuals is critically needed, yet incredibly difficult to deliver via traditional interventions. Internet-based interventions represent a mode of care that is inexpensive, highly transportable, easily standardized, administered, and updated, as well as easily tailored to the needs of specific individuals.

All currently available evidence-based Internet-treatment programs for PTSD are exclusively cognitive behavioral oriented and translate traditional, empirically supported approaches into a Web-based interface (Amstadter, Broman-Fulks, Zinzow, Ruggiero, & Cercone, 2009). However they vary distinctively according to degree of human support, ranging along a continuum extending from completely self-help or stand-alone programs to primarily therapist-administered treatment using a Web-based program to augment the intervention.

Internet-Based Self-Help for PTSD

Ruggiero et al. (2006) investigated the feasibility of a stand-alone, online-based intervention to provide mental health resources to trauma victims of disaster and terrorist attacks (survivors of the 9/11 terrorist attacks). The aim of the program was to provide information and educational resources covering a broad range of relevant clinical issues (in total seven modules—PTSD/panic, depression, worry, alcohol, marijuana, other drugs) and to promote effective coping strategies. Based on the user's clinical symptom profile and predefined clinical thresholds for relevant symptom levels, the relevant self-help modules were automatically identified and administered. Module screeners asked about past-year symptoms and were designed to be brief, highly sensitive, and moderately specific.

The PTSD/panic module screener asked, for example: "In the past year, have you (a) had panic or anxiety attacks?, (b) avoided people, places, situations, or conversations that remind you about something very bad that happened to you?. (c) felt anxious or very upset when in the presence of people, places, or things that remind you about something very bad that happened to you?" Upon completion of each module, the level of distress was assessed and subsequent modules were adapted accordingly. To prevent early dropout and improve compliance, the authors employed a stage-of-change approach (i.e., precontemplative, contemplative, preparation, action, and maintenance stages) through individualized feedback and a motivational language.

Two years after the 9/11 terrorist attacks, 1,035 New Yorker inhabitants who initially took part in an epidemiological study received an invitation to take part in the treatment investigation. In total, 28% ($n=285$) of the original sample were included. The intervention was rated as feasible by the participants; however, completion rates for the individual modules were modest (63.5%, depression; 63.4%, tobacco use; 57.7%, marijuana; 56.1%, PTSD; 42.6%, alcohol; 36.4%, anxiety; and 36.4%, drugs). The time spent per module varied considerably from 4.4 min for the alcohol module to 20.3 min for the depression module. Participants acknowledged an increase of knowledge. One caveat for this investigation was that standard efficacy assessment measures (changes in symptoms/clinically relevant behaviors) were not included. Therefore, although the program seemed generally acceptable to participants, the impact of this approach is difficult to ascertain.

Hirai and Clum (2005) tested the feasibility and efficacy of an 8-week Internet-based self-help program with interactive behavioral techniques for traumatic event-related consequences (SHTC) with undergraduate students and adults from a community-based setting. Participants were recruited from ads in the print media, online, and a student subject pool. Diagnostic screening was completed via the telephone. To be included in the study, applicants had to report a significant traumatic event and meet the reexperiencing and avoidance criteria from the PTSD diagnosis. The treatment consisted of psychoeducation, relaxation training, cognitive restructuring, and written exposure modules. The program also included skills practice in combination with mastery tests and automatic feedback. Therapist involvement during the program was made only to prompt participants to undergo assessments or mastery tests or to provide information about the timeline toward completion of the program and in case of need of technical assistance. In total, 27 applicants were found to be eligible for participation and were randomly assigned to the active treatment group or a wait-list control group. The majority of participants were female students. Participants assigned to the intervention group compared to the wait-list controls showed significant reductions in depressive symptoms and anxiety as well as less avoidance behaviors and intrusions with effect sizes ranging from $d=0.59$ to $d=2.08$. However, treatment adherence, log-in time, or completion rates were not reported. Furthermore, the generalizability of these promising results is somewhat restricted by the small sample size and the homogenous sample (primarily female students).

Interestingly, previous well-designed and methodologically sound studies on the efficacy of conventional (i.e., non-Internet-based) self-help for PTSD failed to produce significant reductions in symptomatology (Ehlers et al., 2003; Turpin, Downs, & Mason, 2005) or to prevent the development of PTSD (Bugg, Turpin, Mason, & Scholes, 2009). One reason for the effects found by Hirai and Clum (2005) might be due to the adaptability of computer-supported self-help programs to the specific patient and their needs. Based on the symptom profile, the patient's input and progress through the modules, computerized self-help programs select treatment modules, generate feedback, and adapt didactic presentations, reinforcement, and future assignments which might promote the efficacy of these approaches.

Despite the initially promising support for Internet-based self-help programs for PTSD, the limited empirical data and methodological limitations of this research indicate that findings should be regarded with caution. Two recent meta-analyses revealed that low rates of treatment initiation and high rates of dropout are two problems that emerge in programs that do not involve human contact (Barak, Hen, Boniel-Nissim, & Shapira, 2008; Spek et al., 2007). The missing therapist contact might have contributed to a higher probability of treatment disengagement. Also, despite sophisticated programming, fully automated programs are always based on a limited number of scenarios and response options. This implies that specific concerns of the patient may not be addressed and could also lessen adherence or limit the use of the program.

Web-Enhanced Therapist-Driven Interventions for PTSD

One of the first research groups to explore the potential of Internet-based interventions for PTSD was Alfred Lange et al. (2000) at the University of Amsterdam. In the 1990s, they developed a therapist-supported, Internet-based cognitive behavioral treatment for posttraumatic stress subsequent to a traumatic event (Interapy). The theoretical base of Interapy emerged from experimental research regarding the efficacy of structured writing therapies on mental and physical health. The treatment consists of structured writing assignments facilitated through a database implemented on the Internet. Communication between therapist and patient is exclusively text-based and asynchronous. The writing protocol comprises three treatment phases: (a) self-confrontation, (b) cognitive reappraisal, and (c) social sharing. Potential patients log in and complete the screening questionnaires (Impact of Event Scale (IES) Horowitz, Wilner, & Alvarez, 1979; Symptom Checklist-90 (SCL-90), anxiety, depression, somatization, and sleeping problems subscales; Somatoform Dissociation Questionnaire (SDQ-5); Nijenhuis, Spinhoven, Van Dyck, Van der Hart, & Vanderlinden, 1997; online). Patients are assigned to two weekly 45-min writing assignments over a 5-week period (10 essays in total). After every second essay, patients receive feedback and further instructions from the therapist (within 24 h). At the beginning of each phase of treatment, patients receive psychoeducation on the principles of the treatment module.

Uncontrolled (Lange et al., 2000) and as well as RCT trials (Lange, van de Ven, Schrieken, & Emmelkamp, 2001) have been conducted to evaluate Interapy for the treatment of PTSD in the Netherlands. The first study included 20 undergraduate students who had experienced traumatic life events and showed symptoms of PTSD. At posttreatment, participants showed significant improvements on posttraumatic stress symptoms and psychological functioning (IES, SCL-90). The first randomized control trial included 30 traumatized undergraduate students. At posttreatment assessment, participants in the treatment condition showed a strong reduction of PTSD symptoms (IES) with large effect sizes ($d = 1.50$ on avoidance and $d = 1.99$ on intrusions). General psychopathology also decreased significantly and yielded large effect sizes for anxiety, depression, and somatization (SCL-90) ($d = 1.23$, $d = 1.28$, and $d = 1.25$, respectively). The second randomized control trial included a clinical sample of 101 patients which replicated prior results of the preceding studies (Lange et al., 2003). Significant improvement on all health-related measures such as depression, anxiety, and physical health was detected. In addition, trauma-related symptoms, such as intrusions and avoidance, were significantly reduced. Effect sizes ranged from $d = 1.28$ for intrusions to $d = 1.39$ for avoidance. The dropout rate was fairly high (41%). In a separate investigation, Lange et al. (2000) found that prior experience with computers was not a prerequisite for a successful treatment response. The improvement levels of participants with little or no experience with the Internet were comparable to the improvement of participants who had extensive experience with the Internet. The Interapy treatment approach was cross-culturally examined in a RCT with 96 patients from a German-speaking sample with posttraumatic stress reactions (Knaevelsrud & Maercker, 2007, 2010). Intention to treat analyses produced similar effect sizes as in the Dutch study (Impact of Event Scale revised, IES-R) with $d = 1.40$ for intrusions, $d = 0.98$ for avoidance, and $d = 1.41$ for hyperarousal. However, a lower dropout rate (16%) was reported.

The Interapy program was tested (using a culturally adapted version called "Ilajnafsy" meaning "psychological help" in Arabic) in Iraq with a sample of Arabic-speaking, war-traumatized civilians with PTSD reactions (Knaevelsrud & Maercker, 2007; Wagner, Schulz, & Knaevelsrud, in press). Ilajnafsy is provided by native Arabic-speaking psychotherapists or psychiatrists living in relatively safe areas in Iraq or neighboring countries (e.g., Palestine, Syria, the Emirates) or in Europe. The therapists were trained in the Interapy approach in 7-day workshops. Just as Interapy, the Ilajnafsy treatment is highly standardized. Text blocks are used for psychoeducation and instruc-

tions, but feedback is tailored to the individual case. All therapists participate in weekly supervision sessions and contribute to an online supervision forum implemented on the web site.

Participants were mainly recruited by means of radio, TV, and print media. Further information was posted on Arabic health-related web sites and made available on a Facebook information page and in a YouTube film clip. An initial pilot study examined the feasibility and applicability of this treatment program in the Iraqi context. Of 212 persons who completed the online screening questionnaires, 40 patients were found to be eligible for participation. Of those, only 15 completed the course of treatment. The majority of patients were female ($n=13$; 86.7%) and had experienced an average of five different traumas, including the kidnapping or killing of a family member/close relative, torture, sexual violence related to war, and threat to their own life. The effect sizes of symptom reduction ranged from $d=1.23$–1.44 for PTSD. The effect size for depression was $d=1.51$ and $d=1.50$ for anxiety. Considering the fact that no psychological intervention for PTSD has previously been evaluated in the Arab context, a key finding is that participants seem to benefit from the Internet-based cognitive behavioral intervention to the same extent as patients in a non-conflict Western context. However, the attrition rate in this pilot study was relatively high (62%), which may partly reflect the ongoing instable and insecure living conditions in Iraq.

Litz, Williams, Wang, Bryant, and Engel (2004) designed a therapist-assisted Internet self-help program for traumatic stress (DE-STRESS). They included a modified version of stress inoculation training (8 weeks in total). The program focuses more on improving coping skills than on trauma processing. The first 6 weeks are dedicated to the improvement of coping skills and management of dysfunctional thinking. Week 7 and 8 comprise trauma processing and relapse prevention receptively. The intervention involves teaching individuals strategies to help cope and manage reactions to trauma cues and comorbid problems. These strategies are reinforced through daily homework assignments. Participants make initial face-to-face contact with a therapist in the context of a clinical interview/assessment session (PTSD Symptom Scale—interview version, PCL; BDI) and are provided with additional email and/or telephone contact as necessary. In addition to the face-to-face evaluation, participants receive an introduction and orientation on how to use the program and how to apply simple relaxation techniques. Patients are encouraged to contact their therapist when needed and are assured that their therapist will monitor their progress. In a RCT, DE-STRESS was compared to Internet-based supportive counseling for PTSD in a sample of survivors of the 2001 Pentagon attacks and Iraq/Afghanistan combat veterans (Litz, Engel, Bryant, & Papa, 2007). A total of 45 patients were included, 33 patients completed treatment (30% dropout rate). Both groups (DE-STRESS and Internet-based supportive counseling) showed a significant reduction for total PTSD severity (PCL), avoidance behaviors, and hyperarousal levels. However, no treatment effects could be detected with regard to reexperiencing symptoms. Although depression (BDI) scores did not differ between groups at posttreatment, differences between groups emerged at the 6-month follow-up assessment. Specifically, the CBT group showed lower depression, anxiety, and total PTSD symptoms (effect sizes comparing the two groups ranged from $d=0.95$–1.03).

Most recently, Klein and colleagues published results from an open trial of a therapist-assisted cognitive behavior therapy Internet intervention for patients with PTSD (PTSD-Online; Klein et al., 2010). This was the first trial where the diagnosis was based on a telephone-based structured clinical interview according to DSM-IV criteria. A total of 134 adults were recruited through mental health web sites, as well as local and national media; however, only 22 individuals were included in the study. The treatment consisted of a 10-week, interactive, cognitive behavioral program that included the following elements: psychoeducation on anxiety, stress, and trauma (module 1); anxiety management including instructions, video/audio instructions on breathing exercises, and PMR (modules 2 and 3); management of

dysfunctional thinking (modules 4–6); individually tailored instruction (audio files and written) on how to expose oneself to the images of the trauma (writing about the trauma) and/or in vivo (modules 7–9); and relapse prevention including information on other anxiety disorders, mood, substance abuse/use, stress, and sleep management (module 10). Each participant was allocated a username and log-in password and had to work through one module per week. The participant and the therapist communicated via encrypted email and therapists answered within 48 h to participants' email. The dropout rate was 27%. At postassessment, 69% of the sample showed clinically significant improvement of PTSD symptoms and 77% of the sample at follow-up assessment. However no significant improvement on general psychological measures was found. The average total therapist time required was 194.5 min.

The results of Web-enhanced therapist-driven interventions for PTSD suggest that online interventions are feasible and that therapist-assisted Internet program for trauma survivors can effectively reduce PTSD symptom severity. However, sample selections (e.g., gender, education, and racial/ethnicity); uptake rates (how many of those targeted actually use this approach), just as user profiles (e.g., age, social economic status); and indication ranges (e.g., symptom severity, comorbidity, specific symptom pattern) are yet to be critically investigated.

Humanitarian Aid Online: Interapy in Iraq (Ilajnafsy)

Subsequently, a case illustration of Ilajnafsy, as introduced above, is provided. Each treatment phase includes an example of the therapist's writing instruction and an excerpt of the patient's text. The patient Ms. I. is a 41-year-old female Iraqi who survived two kidnapping attempts and a severe physical assault. At her initial assessment, she reported severe PTSD symptoms and a very restricted level of functioning (inability to work, social withdrawal). The text below is taken from the actual transcripts of the treatment.

First Phase: Self-Confrontation

First, patients are instructed to write two essays on the circumstances of the traumatic event. They are asked to express all their fears and thoughts about the event and to focus on sensory perceptions in as much detail as possible. Participants are asked to write their essays in the present tense, in the first person, and without worrying about grammar, style, or the logical chronology of events.

Therapist instruction: "*In the following texts, I would like to ask you to choose one moment from your traumatic event. One moment that you can hardly bear to think about, but that keeps intruding on your thoughts. Write down the most painful memories and emotions you have when you think about it and describe everything that you experience every feeling, every thought and physical reaction.*"

Before Ms. I started with her text she commented: "*This is the first message I write to you Dr. X. As a matter of fact my head bursts from all the bad memories. In the last days since I decided to do this therapy I was almost flooded with pictures from the past – it is almost impossible to choose one single incident. Our daily life is still so violent and the feeling of being vitally threatened has become natural not just to me – but for every Iraqi.*"

Example of the patient's text: "*The men in the car are still tracking me – every time I switch lanes – they do too, I try to speed up now – but they do the same. I force myself to focus; I keep on looking in the rear mirror, trying to memorize their face. I have never seen these men before; they are not Iraqis (I can tell it from the way they wear their Eqal (headband)). What do they want from me? What do they want from my family? They must have followed us from the hospital. I feel hot. I don't know where to go. If I go home they know where I live and I will put my whole family at risk? Where can I go? Where should I go????? My mind is almost blacking out. My head is aching, I cannot think – it feels like a stone. What can I do? Pictures of dead bodies on a pickup truck from last week are coming in my head again. I need help, please!!! I am sweating*

and my heart is racing. My head is about to explode. My eyes are tearing. I am scared and angry, my brain is of no use. Why is no one helping me? I feel so lost, so weak. Why is this done to me? What have I done wrong to deserve this? God, tell me what to do! The car is pulling over and cuts off my way – I have to stop! Three men are stepping out of the car."

Thank god –writing time is up…I am still shaking!!

Ms. I got robbed and assaulted but survived as the perpetrators got disturbed by a local police force, which was alarmed by witnesses. The subjectively worst moment was the situation described above when she was afraid to be raped and/or killed. She felt ashamed of having been unable to defend herself and of having been unable to prevent this assault from happening. Especially in the first phase, she needed repeated encouragement to continue the exposure.

Second Phase: Cognitive Reappraisal

In this cognitive restructuring phase, patients are instructed to write a supportive and encouraging letter to a hypothetical friend. They are asked to imagine that this friend had also experienced the same kind of traumatic experience and was now facing the same difficulties. The letter should reflect on guilt feelings, challenge dysfunctional automatic thinking and behavior patterns, and correct unrealistic assumptions. The aim is to foster the development of new perspectives on the traumatic event and its circumstances. An example instruction of the therapist for the first two essays in this phase is as follows:

Therapist instruction: *"Imagine you are writing a supportive letter to your friend, who experienced the same situation as you. Could she have foreseen what happened? Do you think she was responsible for this?"*

***Example of the patient's text*:**

"My friend, I am writing you, hoping my words will make a difference, that they help to ease your pain…We both know that I can't do much more than to remind you of some facts in life that you may have forgotten or lost faith in after what you have been through…

First of all, you have managed to escape the attempted kidnappings and did not get kidnapped and this is an achievement by itself, you managed to run and save your family. You are safe now and that is very important.

You are a strong person who fought to save her family from serious dangers. You have done what you could at the moment and you were brave. I know it is difficult but we may need to learn to accept experiences that we can't change and to be smart enough to learn the wisdom in each hard lesson we take. Maybe you can think of a way to redirect those negative feelings into something positive. Express your anger when you feel angry; if you feel like talking talk to someone that you trust or feel comfortable with. Don't be ashamed. And don't waste your energy on feelings like hate and revenge. These feelings hurt you more than they hurt the people who harmed you.

You are a good person who is loved and cherished by all the people who know you, by your family, your friends this is the greatest strength any human can have, this is the real treasure in life."

Third Phase: Social Sharing and Farewell Ritual

During the third phase, patients receive psychoeducation about the positive effects of social sharing. In a final letter, they then take symbolic leave of the traumatic event. Patients can address the letter either to themselves, to a close friend, or another significant person involved in the traumatic event. The letter does not ultimately have to be sent.

Therapist instruction: "*You wrote that you would like to write the letter to your friend. First, I would like to ask you to describe the circumstances that happened. Which moments were so important that you would like to tell yourself about them? It is important to give the past, the present and the future the same weight in this letter.*"

Example of the patient's text:

"*Dear X*,

I write this letter to you, to tell you about some experiences you don't know about. Things I was not courageous enough to share with you. But I want you to know now as I believe it may help you to understand me more and at the same time help me to get over some of the difficult events I have experienced…

For a very long time I thought that all the nightmares and the flashbacks and my panic reactions were a "normal" thing – I just got so used to it. I did not realize that these were "symptoms" and that they were expanding. Like the exaggerated reactions when someone tried to wake me up when I was asleep, or when hearing fireworks or other simple everyday events. I did not even notice my increased solitude and increased loneliness and introverted behavior that all my friends and beloved ones had noticed….

The two kidnapping attempts, the awful scenes of the dead people and threats I have been through, radically changed my view of the world. I could not trust anyone or more precisely any men. I could not trust life anymore….

Now I look back on these horrible events. I have more trust in myself as I see myself as a better person. I believe now that my reactions were the best I could do at that time and that given the circumstances I could not have reacted differently or in a better way. I have managed to save myself and the people with me. And I should be proud of that. And I am!…"

Discussion

The use of the Internet and self-help therapy is gaining momentum. While it is, and will presumably remain a less potent alternative to face-to-face psychotherapy, it comes with many advantages and for some settings it may represent the only option for providing some form of empirically validated treatment to sufferers (e.g., currently treatment-reluctant patients due to shame or stigma; patients living in countries with no proper psychological-medical help system; people in remote areas). As the current review demonstrates, Internet therapy and self-help is an umbrella term comprising very different approaches (e.g., bibliotherapy evaluated via Internet studies, computer-assisted therapy). A common denominator is that no face-to-face therapy is conducted: the therapist is either absent (bibliotherapy), available only upon request (some forms of computer-assisted therapy), or is involved in an asynchronous (email therapy) or synchronous non-face-to-face fashion. As the reviewed investigations employed very different programs in different settings, it is not surprising that no consistent picture emerges with regard to the efficacy of (guided) self-help. Clearly, no bold claims can be made about self-help per se. The success of self-help largely depends on the methods adopted.

While interventions delivered or supported by a health care specialist is probably superior to the same intervention practiced via self-help (e.g., Tolin et al., 2007) according to the few studies comparing both approaches directly (see however Carlbring et al., 2005; Kiropoulos et al., 2008), self-help is not only an important alternative where proper therapy cannot be delivered but may act as a complement to raise the quality of regular therapy. Self-help manuals and Internet devices could complement the arsenal of psychotherapists and shorten treatment times (Marks et al., 2003). For example, patients undergoing short-term intervention may be encouraged to read special chapters and exercise techniques for which there is no sufficient time during therapy (Mataix-Cols & Marks, 2006). Studies indicate that the effect sizes and success rates of interventions conducted in RCTs with well-trained, experienced, and ardent psychotherapeutic staff do not fully translate into clinical practice even if the same label is used (e.g., CBT). Approximately 50% of the therapists trained in CBT do not practice exposure and response prevention, and some newer evidence-based techniques (e.g., mindfulness) may not be known to some therapists, dismissed or forgotten/neglected (Böhm, Förstner, Külz, & Voderholzer, 2008; Külz et al., 2009).

Table 24.2 Recommendations for online studies evaluating Internet and self-help approaches

Basic requirements
Cookies should be enabled (in order to prevent multiple log-ons)
Large sample ($N > 50$)
Recruitment over specialized self-help forums or a contact list of previously treated and adequately diagnosed in- or outpatients to ensure that the target population is reached
Lie scales (e.g., openness); minimum performance duration of 15 min to fill out the entire survey in order to recruit individuals with high treatment motivation
Availability of a specialist in case of technical or psychological problems
Approval by the local ethic committee
Advanced features
Randomized controlled trial (experimental vs. waitlist or active control)
Completion >70% (multiple reminders are recommended to raise completion rates)
Retest reliability of primary outcome measure ($r > 0.7$) as assessed by Internet administration of the measure
Verification of diagnoses via email exchange and preferably telephone, Skype, or doctor-in-charge (downside: may scare off some potential participants)
Follow-up study (may decrease completion rate)

Currently, many Internet and self-help approaches are evaluated in a clinical setting. A problem with this approach—apart from funding—is that it only reaches the subgroup (and perhaps minority) of patients who are willing to see a health care professional. Shame, stigma, and unfounded fears especially in OCD patients to be incarcerated against one's will, e.g., because of aggressive obsessions may scare off many patients to participate in such studies which are thus not representative. Indeed, it has been found that help-seeking and non-help-seeking patients differ on many aspects, most importantly quality of life and illness insight (Besiroglu, Cilli, & Askin, 2004). In our view, Internet studies come with many advantages and are especially valuable for feasibility (i.e., proof-of-concept) studies. Table 24.2 lists a number of criteria and precautions that should be taken into account when planning such a study. Importantly, Internet studies like clinical trials need approval by an ethics committee. In view of only peripheral contact (mainly email) researchers are advised to provide participants with telephone numbers and (email) addresses in case of adverse events.

To conclude, we hope that self-help and Internet approaches gain more attention within the scientific community and are no longer judged as ineffective or perhaps even harmful substitutes to more traditional treatments. As discussed in our review, these alternative approaches could be instrumental in providing treatment to individuals who would otherwise not have access to treatment providers. Furthermore, both self-help and Internet interventions may foster treatment motivation in patients who, for example, are skeptical about the benefits of face-to-face therapy and who would be more amenable to starting with such an alternative treatment format. The overarching hope in continuing to develop this type of intervention is to better disseminate efficacious treatments to the many millions of individuals worldwide who are not receiving the treatments they need.

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25 Where Do We Go from Here? How Addressing Clinical Complexities Will Result in Improved Therapeutic Outcomes

Eric A. Storch and Dean McKay

The past several decades have been characterized by significant advancements in the understanding and treatment of psychiatric diagnoses. Well-conceptualized and empirically supported interventions/approaches are in place for virtually all of the disorders covered in the book, and new studies are rapidly coming out that provide further insight into mental health treatments that work. Although encouraging and a marked step forward, straightforward, uncomplicated presentations are often the exception rather than the rule in terms of the individuals included in these trials. Yet, in applied practice, clinical presentations characterized by varied complexities are common and can markedly impact treatment course and outcome without appropriately consideration. The purpose of this book is to advance the literature beyond the understanding that a particular treatment, on average, works for the average person with the corresponding disorder. Rather, it is our hope that this two-series volume increases the application of personalized care in the mental health treatment of adults and children who present as clinically complex.

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The movement to determine best practices for common complexities associated with psychopathology has been with us for some time. As Gordon Paul famously intoned, the goal of psychotherapy research is to identify the conditions, clients, and circumstances for which any treatment is ideally suited (Paul, 1969). This implies that clinicians have at their disposal a menu of therapies from which to select when treating clients with various complex presentations. Interestingly, many signs and symptoms that imply different psychopathological states have been observed and go by names that would be considered antiquated in the current nosology. Many problems considered complications in the treatment of psychopathology have been described, and at the same time, practitioners traditionally fail to adequately understand or account for these problems (Meehl, 1973). These accounts, however, predate the movement to promote scientifically informed principles of treatment delivery. It therefore seems that the time has arrived to fully address the complexities associated with core diagnostic problems. In this vein, the anxiety disorders have been subject to extensive study, many commonly observed complexities have been systematically examined, and modifications to the protocols developed for "uncomplicated" cases have been described and in some cases tested.

The empirically supported treatment movement has shown that numerous therapies could, indeed, become established as well-validated approaches for treating psychopathology (reviewed in Chambless & Ollendick, 2001). Concurrent with the movement to identify core principles and

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practices that would reliably alleviate symptoms, it was considered best to also examine potential comorbid conditions as complicating factors (i.e., Rachman, 1991). However, comorbidity implies two conditions that are prohibitive in understanding complexities. First, comorbidity implies that specific diagnostic thresholds are passed resulting in more than one diagnosis being assigned to a case. While this does in fact occur with some regularity, it also poses a problem for the many more cases that are subthreshold for the diagnosis, and yet the comorbid associated problem nonetheless interferes with treatment delivery. And, second, comorbidity implies that the potential diagnostic problem is alongside the condition for which a target treatment is applied. While this too is frequently the case (i.e., the common complication in obsessive–compulsive disorder (OCD) of comorbid depression; see Keeley, Storch, Merlo, & Geffken, 2008), it does not account for the many times when a condition is instead secondary to the target problem (such as depression that results from OCD). These observed limitations in considering comorbidity as a pure model for understanding complexities have led instead to consideration given to dimensional features of ancillary psychopathology that interferes with therapy. This is the primary thrust of this volume.

As noted in each chapter, there are fairly robust empirical data supporting cognitive behaviorally oriented interventions for a range of disorders and problem behaviors. However, also as shown across the varied chapters, multiple confounding factors can impact treatment delivery and outcome, which require adjustments to established approaches. Given this, treatments must be tailored to the clinical presentation of each individual to maximize efficacy, as well as intervention acceptability. For example, a topic that has received attention among anxious youth is the presence of disruptive behavior (Storch et al., 2008). Application of existing therapies without accounting for the manner in which disruptiveness might impact treatment course would likely yield attenuated outcome. Similarly, comorbid psychopathology must be considered in the individual's clinical presentation and may require adaptations in how the case is conceptualized and treated. Given this, it is critical to move beyond efficacy studies that demonstrate that treatment X works for Y condition, by examining moderators of treatment response with an eye for developing interventions that account for these issues. Stated differently, we need to know who is not benefiting from extant interventions, as well as why this may be the case (i.e., outcome mediators). With that information, the intervention in question can then be tailored to account for these variables. Along these lines, we highlight the role of empirically supported practice (Treat, Bootzin, & Baker, 2007) that involves the application of established treatment approaches within the context of specific symptom presentations that fosters the use of treatment plans that are formulated for specific diagnoses, not necessarily specific individuals who happen to suffer from the diagnosis.

Beyond the issue of how to effectively intervene with individuals who present with varied complexities are issues with treatment dissemination. We perceive the field to be at a critical juncture in this regard as there are numerous providers in the community, yet many do not provide evidence-based interventions, and, arguably, others provide forms of intervention that do not benefit the affected person and weaken confidence in the field of psychology as a health profession. While this has been recognized and served as a motivating force in federally funded research and the development of alternative service delivery platforms (e.g., telemedicine), progress has been slow at best, and problematically, there are increasing numbers of programs that train well-intentioned providers to provide nonempirically established or evaluated services. Inconsistency among psychological providers in the community—who trusts that the provider has their best interest at heart—in the type of psychotherapy provided conveys the inaccurate notion that psychotherapy is ineffective. And, valuable resources are drained (e.g., insurance, family savings) at the risk of continued impairment and encouraging a sense of hopelessness in those who have had limited response to non-evidence-based treatments. Indeed, while ineffective treatment may be potentially benign in the eyes of some providers, it has been cited recently as a specific harmful effect of therapy (Dimidjian & Hollon, 2010). For these reasons, dissemination

efforts that encourage the use of evidence-based interventions in clinical settings are critically highlighted as an area for additional attention. Although it is clear that dissemination efforts are required to move the path forward, barriers exist that need to be considered. Efforts to bring training of effective interventions into training programs that provide personnel who work on the "front lines" are required. At a grassroots level, this means reaching out to colleagues across disciplines to provide interdisciplinary training in therapies that work. A recent task force has developed guidelines to inform training programs in best practices in ensuring practitioners have the necessary skills to evaluate and implement empirically supported practices, particularly cognitive behavior therapy (Klepac et al., in press). This mirrors a growing international movement to increase the delivery of empirically supported treatments (i.e., the National Institute of Clinical Excellence; Silk, 2010). With these efforts, and working with funding sources to prioritize how resources are allocated for the provision of treatments that have demonstrated efficacy, the likelihood that clients will receive the proper care will increase. However, it is not simply enough to disseminate effective interventions. Rather, consideration for how these interventions must be tailored to address clinical factors—many of which were detailed in this book—is a necessity and will direct the next way of clinical dissemination efforts. Indeed, we are seeing some of this with the focus on modularized interventions in their application to childhood problems (Weisz et al., 2012).

Conclusion

When we crafted the book, our goal was to convey the multiple intricacies and complexities for working with individuals with anxiety. It is our hope that the book helps the reader conceptualize factors that may contribute to clinical complexity and treatment challenges, with the goal of formulating interventions that are tailored to individual patient characteristics and yield improved outcomes. Although mental health providers are improving at recognizing and integrating varied clinical factors into their case conceptualization and intervention, there is more to be done to disseminate information about best practices for handling such factors with consistency and replicability. With this in mind, we hope that the present volume provides a starting point in this regard to (1) improve clinical outcome and (2) guide researchers for evaluating the efficacy of varied approaches to dealing with diverse patient factors.

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