CBT FOR ANXIETY DISORDERS

A Practitioner Book

EDITED BY
GREGORIS SIMOS
AND STEFAN G. HOFMANN

WILEY-BLACKWELL

Contents

Index

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List of Figures, Tables, and Boxes

Introduction

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Anxiety disorders are some of the most prevalent disorders, affecting three out of ten people in their lifetime (Kessler *et al.*, 2005). These disorders start early in life, negatively affect significant aspects of functioning, tend to be chronic and unremitting, and cause considerable psychological suffering and life impairments (Martin, 2003). In addition, they are highly comorbid with three out of four anxiety disorder patients experience at least one other mental disorder in their lifetime (Michael, Zetsche, and Margraf, 2007).

The efficacy of cognitive behavior therapy (CBT) for anxiety disorders in adults has been supported by multiple meta-analyses (Hofmann *et al.*, 2012). For example, CBT is superior to no treatment and control treatments (Norton and Price, 2007; Olatunji, Cisler, and Deacon, 2010), including placebo control (Hofmann and Smits, 2008). The latter meta-analysis shows that CBT is associated with medium to large effect sizes over placebo, suggesting that, although effective, there is still considerable room for further improvement (Hofmann and Smits, 2008).

Research on the cognitive model of anxiety and the development of disorder-specific cognitive treatment protocols for anxiety disorders is a continuously evolving process (e.g., Clark and Beck, 2010). For this reason, we invited some of the foremost experts on CBT for anxiety disorders to provide an update of the contemporary state of the art of treating anxiety disorders. All chapters include the

treatment rationale, concrete clinical case examples, therapist–patient dialogues and, where appropriate, subsections of techniques for dealing with treatment complications, comorbid disorders, and managing concurrent pharmacotherapy, and ethnicity.

The list of contributors and topics include “Panic Disorder and Agoraphobia” (Chapter 1) by Michelle G. Craske and Gregoris Simos; “Obsessive–Compulsive Spectrum Disorders: Diagnosis, Theory, and Treatment” (Chapter 2) by David A. Clark and Gregoris Simos; “Generalized Anxiety Disorder: Targeting Intolerance of Uncertainty” (Chapter 3) by Melisa Robichaud; “Social Anxiety Disorder: Treatment Targets and Strategies” (Chapter 4) by Stefan G. Hofmann, Jacqueline Bullis, and Cassidy Gutner; “Specific Phobias” (Chapter 5) by Lars-Göran Öst and Lena Reuterskiöld; “Health Anxiety” (Chapter 6) by Michel A. Thibodeau, Gordon J.G. Asmundson, and Steven Taylor; “Trauma-Focused Cognitive Behavior Therapy for Posttraumatic Stress Disorder and Acute Stress Disorder” (Chapter 7) by Anke Ehlers; “Culturally Appropriate CBT for the Anxiety Disorders” (Chapter 8) by Devon E. Hinton and Martin La Roche; and “Newer Generations of CBT for Anxiety Disorders” (Chapter 9) by Michael P. Twohig, Michelle R. Woidneck, and Jesse M. Crosby.

Our hope is that this text will provide the reader with up-to-date knowledge about the current state-of-the-art CBT approaches for anxiety disorders. We believe that it will be of interest to anyone who wants to help patients with anxiety disorders – practitioners in training, senior clinicians, researchers, residents, graduate psychology, and medical students.

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1

Panic Disorder and Agoraphobia

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Overview

The current diagnostic criteria for panic disorder, according to the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV, American Psychiatric Association, 1994), are comprised of recurrent unexpected panic attacks, and anxiety about future panic attacks or their consequences, or a significant behavioral change because of the panic attacks. The additional anxiety about panic, combined with catastrophic cognitions about panic sensations, contributes to the differentiation between the person with panic disorder and the person with occasional panic attacks (e.g., Telch, Lucas, and Nelson, 1989). Agoraphobia refers to avoidance or endurance with dread of situations from which escape might be difficult or help unavailable in the event of a panic attack, or in the event of developing symptoms that could be incapacitating and embarrassing, such as loss of bowel control or vomiting. Typical agoraphobic situations include shopping malls, waiting in line, movie theaters, traveling by car or bus, crowded restaurants, and being alone.

In the general population, the 12-month prevalence estimate for panic disorder across the United States and several European countries is about 2% in adults and adolescents (Goodwin, Fergusson, and Horwood, 2005; Kessler *et al.*, 2005b). Lower estimates have been reported for some Asian, African, and Latin American countries, ranging from 0.1 to 0.8% (Lewis-Fernandez *et al.*, 2010). Across all studies, females are more frequently affected than males at a rate of approximately 2 : 1 (Kessler *et al.*, 2005b). Although panic attacks occur in children, the overall prevalence of panic disorder is low prior to 14 years of age (<0.4%) (Craske *et al.*, 2010). The rates of panic disorder show a gradual increase during adolescence, particularly in girls, and possibly following the onset of puberty (Craske *et al.*, 2010). The modal age of onset is late teenage years and early adulthood (Kessler *et al.*, 2005a), although treatment is usually sought at a much later age, around 34 years (e.g., Noyes *et al.*, 1986). The prevalence rates decline in older individuals, possibly reflecting diminishing severity to subclinical levels (Wolitzky-Taylor *et al.*, 2010). In general, differences in prevalence across gender, culture, and age groups may be due to a variety of factors, including the expression of the disorder, underlying physiology or biology, varying degrees of concern about the dangerousness of symptoms of autonomic arousal and mental symptoms of anxiety, and sensitivity of instrumentation for diagnosing panic disorder.

Panic disorder and agoraphobia tend to be chronic conditions, with severe financial and interpersonal costs; that is, only a minority (30%) of untreated individuals remit without subsequent relapse, although a similar number experience notable improvement, albeit with a waxing and waning course (35%) (Katschnig and Amering, 1998; Roy-Byrne and Cowley, 1995). Also, panic disorder is associated with high levels of social, occupational, and physical

disability, considerable economic costs, and the highest number of medical visits among the anxiety disorders, although the effects are strongest with the presence of agoraphobia (Wittchen *et al.*, 2010).

Rarely does the diagnosis of panic disorder, with or without agoraphobia, occur in isolation. Commonly co-occurring Axis I conditions include specific phobias, social phobia, dysthymia, generalized anxiety disorder, major depressive disorder, and substance abuse (e.g., Brown *et al.*, 2001; Kessler *et al.*, 2005b). Also, from 25 to 60% of persons with panic disorder also meet criteria for a personality disorder, mostly avoidant and dependent personality disorders (e.g., Chambless and Renneberg, 1988). However, the nature of the relationship between panic disorder/agoraphobia and personality disorders remains unclear, especially as some “personality disorders” remit after successful treatment of panic disorder/agoraphobia (e.g., Latas *et al.*, 2000; Marchesi *et al.*, 2005; Ozkan and Altindag, 2005).

Cognitive Behavioral Model

Several independent lines of research (Barlow, 1988; Clark, 1986; Ehlers and Margraf, 1989) converged in the 1980s on the same basic conceptualization of panic disorder as an acquired fear of bodily sensations, particularly sensations associated with autonomic arousal. Psychological (i.e., temperament, such as negative affectivity) and biological (i.e., genetic) predispositions are believed to enhance the vulnerability to acquire such fear. Fear conditioning, avoidant responding, and information processing biases are believed to perpetuate such fear. It is the perpetuating factors that are targeted in the cognitive behavioral treatment approach.

The temperament most associated with anxiety disorders, including panic disorder, is neuroticism (Eysenck, 1967; Gray, 1982) or proneness to experience negative emotions in response to stressors. A closely linked construct is "negative affect," or the tendency to experience a variety of negative emotions across a variety of situations, even in the absence of objective stressors (Watson and Clark, 1984). Neuroticism predicts the onset of panic attacks in adolescents (Hayward *et al.*, 2000; Schmidt, Lerew and Jackson, 1997, 1999), and "emotional reactivity" at age 3 was a significant variable in the classification of panic disorder in 18- to 21-year-old males (Craske *et al.*, 2001). Numerous multivariate genetic analyses of human twin samples consistently attribute approximately 30–50% of variance in neuroticism to additive genetic factors (Eley, 2001; Lake *et al.*, 2000). In addition, anxiety and depression appear to be variable expressions of the heritable tendency toward neuroticism (Kendler *et al.*, 1987). Symptoms of panic (i.e., breathlessness and heart pounding) may be additionally explained by a unique source of genetic variance that is differentiated from symptoms of depression and anxiety (Kendler *et al.*, 1987) and neuroticism (Martin *et al.*, 1988).

Another temperament is anxiety sensitivity, which refers to the trait of believing that anxiety and associated symptoms may cause deleterious physical, social, and psychological consequences that extend beyond any immediate physical discomfort during an episode of anxiety or panic (Reiss, 1980). Anxiety sensitivity is elevated across most anxiety disorders, but it is particularly elevated in panic disorder, especially the physical concerns subscale (Zinbarg, Barlow, and Brown, 1997). Anxiety sensitivity is believed to comprise a specific psychological vulnerability for panic disorder because it primes fear reactivity to bodily sensations. In support, several longitudinal studies indicate

that high scores on the anxiety sensitivity index predict the onset of panic attacks over 1- to 4-year intervals in adolescents (Hayward *et al.*, 2000), college students (Maller and Reiss, 1992), and community samples with specific phobias or no anxiety disorders (Ehlers, 1995). In addition, anxiety sensitivity index scores predicted spontaneous panic attacks, and worry about panic (and anxiety more generally), during an acute military stressor (i.e., 5 weeks of basic training), even after controlling for history of panic attacks and trait anxiety (Schmidt, Lerew and Jackson, 1999). Finally, panic attacks themselves elevate anxiety sensitivity over a 5-week period in adults (Schmidt Lerew, and Jackson, 1999), and over a 1-year period in adolescents, albeit to a lesser extent (Weems *et al.*, 2002).

However, Bouton, Mineka, and Barlow (2001) noted that the relationship between anxiety sensitivity and panic attacks in these studies was relatively small, not exclusive to panic, and was weaker than the relationship between panic and neuroticism. Furthermore, these studies have evaluated panic attacks and worry about panic but have not evaluated prediction of diagnosed panic disorder. Thus, the causal significance of anxiety sensitivity for panic disorder remains to be fully understood.

Acute “fear of fear” (or more accurately, anxiety focused on somatic sensations) that develops after initial panic attacks is attributed to two factors: catastrophic misappraisals of bodily sensations (i.e., misinterpretation of sensations as signs of imminent death, loss of control, and so on) (Clark, 1986); and interoceptive conditioning, or conditioned fear of internal cues, such as elevated heart rate, because of their association with intense fear, pain, or distress (Razran, 1961). Specifically, interoceptive conditioning refers to low-level bodily sensations of arousal coming to serve as conditional stimuli that trigger increased autonomic arousal and fear through Pavlovian conditioning

(Bouton, Mineka, and Barlow, 2001). Thus, small changes in physiological functioning lead to conditioned fear or panic as a result of prior pairings of these initial somatic sensations with full-blown panic attacks. An extensive experimental literature attests to the robustness of interoceptive conditioning (e.g., Dworkin and Dworkin, 1999), particularly with regard to early interoceptive drug onset cues becoming conditioned stimuli for larger drug effects (e.g., Sokolowska, Siegel, and Kim, 2002). In addition, interoceptive conditioned responses are not dependent on conscious awareness of triggering cues and thus have been observed under anesthesia (e.g., Block *et al.*, 1987). As such, interoceptive conditioning accounts for what appear to be “out of the blue” panic attacks.

Evidence for extreme fear and anxiety of somatic sensations is robust across a variety of paradigms. Persons with panic disorder endorse strong beliefs that bodily sensations associated with panic attacks cause physical or mental harm (e.g., McNally and Lorenz, 1987). They are more likely to interpret bodily sensations in a catastrophic fashion (Clark *et al.*, 1988), and to allocate more attentional resources to words that represent physical threat such as “disease” and “fatality” (e.g., Hope *et al.*, 1990), catastrophe words, such as “death” and “insane” (e.g., Maidenberg *et al.*, 1996), and heartbeat stimuli (Kroeze and van den Hout, 2000). Also, individuals with panic disorder show enhanced brain potentials to panic-related words (Pauli *et al.*, 2005). In addition, they are more likely to become anxious in procedures that elicit bodily sensations similar to the ones experienced during panic attacks, including benign cardiovascular, respiratory, and audiovestibular exercises (Antony *et al.*, 2006), as well as more invasive procedures such as carbon dioxide inhalations, compared to clients with other anxiety disorders (e.g., Perna *et al.*, 1995; Rapee *et al.*, 1992) or healthy

controls (e.g., Gorman *et al.*, 1994). The findings are not fully consistent, however, as clients with panic disorder did not differ from clients with social phobia in response to an epinephrine challenge (Veltman *et al.*, 1996). Nonetheless, individuals with panic disorder also fear signals that ostensibly reflect heightened arousal and false physiological feedback (Craske *et al.*, 2002; Ehlers *et al.*, 1988).

Such anxiety about bodily sensations plays a central role in the perpetuation of panic disorder. First, once the bodily sensations are noticed, they elicit fear in an individual with panic disorder. This fear serves to intensify the sensations, causing an increase in fear, which further enhances the bodily sensations in a self-perpetuating cycle of fear and bodily sensations that typically results in a panic attack. Second, because bodily sensations that trigger panic attacks are not always immediately obvious, they may generate the perception of unexpected or "out of the blue" panic attacks that generates even further distress (Craske, Glover, and DeCola, 1995). Third, the perceived uncontrollability, or inability to escape from, or terminate bodily sensations again is likely to generate heightened anxiety (e.g., Maier, Laudenslager, and Ryan, 1985). Unpredictability and uncontrollability, then, are seen as enhancing general levels of anxiety about "when is it going to happen again" and "what do I do when it happens," thereby contributing to high levels of chronic anxious apprehension. In turn, anxious apprehension increases the likelihood of panic, by directly increasing the availability of sensations that have become conditioned cues for panic and/or by increasing attentional vigilance for these bodily cues. Thus, a maintaining cycle of panic and anxious apprehension develops.

Individuals with panic disorder often engage in safety behaviors that they believe enable them to escape or avoid the feared outcome. For example, if individuals believe that

they will pass out during a panic attack, they might sit down or hold on to an object for support. Engaging in safety behaviors prevents disconfirmation of cognitive misappraisals, thus contributing to the maintenance of panic disorder (Salkovskis, Clark, and Gelder, 1996). Individuals may also engage in safety behaviors designed to prevent panic, or its feared consequences, such as carrying around anxiolytic medication or traveling with a companion who makes them feel safe. Another panic-maintaining behavioral response is overt avoidance of particular places or situations where panic attacks are anticipated to occur. Avoidance prevents disconfirmation of catastrophic misappraisals, and reinforces the fear that those particular situations are dangerous, increasing the likelihood of panicking in those situations in the future.

Components of Cognitive Behavioral Therapy

As outlined in more detail elsewhere (Simos, 2002), the treatment begins with education about the nature of panic, the causes of panic and anxiety, and the way in which panic and anxiety are perpetuated by feedback loops among physical, cognitive, and behavioral response systems. In addition, specific descriptions of the psychophysiology of the fight–flight response are provided, as well as an explanation of the adaptive value of the various physiological changes that occur during panic and anxiety. The purpose of this education is to correct common myths and misconceptions about panic symptoms (i.e., beliefs about going crazy, dying, or losing control).

Self-monitoring is introduced in the first treatment session and is continued throughout the entire treatment. Self-monitoring functions in two ways: to provide ongoing

assessment of change in panic, anxiety, and avoidance; and as a therapeutic tool to encourage objective self-awareness and increase accuracy in self-observation. Clients are asked to keep at least two types of self-monitoring records. The first is a panic attack record, to be completed as soon as possible after each panic attack; this record provides a description of cues, maximal distress, symptoms, thoughts, and behaviors. The second is a daily mood record, completed at the end of each day, to keep record of overall or average levels of anxiety, depression, and so on. Additionally, clients may keep a daily record of activities or situations avoided.

Breathing retraining is a commonly used somatic coping skill, given evidence for respiratory abnormalities in panic disorder possibly due to hypersensitive medullary carbon dioxide (CO_2) detectors, resulting in hypocapnia (i.e., lower than normal levels of pCO_2) (e.g., Caldirola *et al.*, 2004). Traditional breathing retraining involves slow, abdominal breathing exercises. However, its value has been questioned in terms of the degree to which it actually corrects hypocapnic breathing or rather serves as a distraction (Garssen, de Ruiter, and van Dyck, 1992). In contrast to traditional breathing retraining, capnometry-assisted respiratory training (CART) (Meuret *et al.*, 2008) uses immediate feedback of end-tidal pCO_2 to teach clients how to raise their subnormal levels of pCO_2 (hyperventilation) and thereby gain control over dysfunctional respiratory patterns and associated panic symptoms (e.g., shortness of breath and dizziness). CART has been shown to improve panic symptoms, in part through reducing hypocapnic breathing (Meuret *et al.*, 2010). Another somatic coping skill is progressive muscle relaxation, in which clients are trained over a number of weeks in 16-muscle groups, 8-muscle groups, 4-muscle groups, and finally cue-control relaxation, at which point relaxation is used as a coping skill for

practicing exposure to items from a hierarchy of anxiety-provoking tasks.

In the cognitive restructuring component of cognitive behavioral therapy (CBT), detailed self-monitoring of emotions and associated cognitions is used to identify specific beliefs, appraisals, and assumptions. Relevant cognitions are categorized into types of errors, such as overestimations of risk of negative events, or catastrophizing the meaning of events. In labeling the type of cognitive distortion, the client is encouraged to use an empirical approach to examine the validity of thoughts by considering all of the available evidence. Therapists use Socratic questioning to help clients make guided discoveries and question their anxious thoughts. Next, alternative hypotheses are generated that are more evidence-based. In addition to surface-level appraisals (such as “my heart is racing dangerously too fast”), core-level beliefs or schemata (such as “I am too weak to withstand distress”) are questioned in the same way.

In vivo exposure refers to repeated and systematic real-life exposure to agoraphobic situations. Most often, *in vivo* exposure is conducted in a graduated manner, proceeding from the least to the most anxiety-provoking situations on an avoidance hierarchy, although there is some evidence to suggest that intensive or ungraduated exposure may be effective (e.g., Feigenbaum, 1988). Critical to *in vivo* exposure is the removal of safety signals and safety behaviors, such as other people, empty or full medication bottles, seeking reassurance, or checking for exits. Reliance on safety signals and safety behaviors attenuates distress in the short term but is believed to maintain excessive anxiety in the long term. They are replaced by effective use of cognitive restructuring and somatic coping skills, with care to ensure that the coping skills themselves do not become alternative safety behaviors. *In vivo* exposure can be

conducted with the therapist's guidance, followed by self-directed exposures between sessions (to enhance generalization of learning and to limit the safety signal value of the therapist). Recent data support the value of therapist-directed exposure (Gloster *et al.*, 2011).

In interoceptive exposure, the goal is to deliberately induce feared physical sensations a sufficient number of times and for long enough each time so that misappraisals about the sensations are disconfirmed and conditioned anxiety responding extinguishes. A standard list of exercises, such as hyperventilating and spinning, are used to establish a hierarchy of interoceptive exposures. Clients are encouraged to endure the sensations beyond the point at which they are first noticed because early termination interferes with new learning. Interoceptive exposure is usually first conducted in-session with the therapist's guidance, followed by self-directed practice between sessions. Interoceptive exposure extends to naturalistic activities that inherently induce somatic sensations (e.g., caffeine consumption and exercise programs). Eventually, *in vivo* exposure is combined with interoceptive exposure, by deliberately inducing feared sensations in feared situations.

A final step of CBT is relapse prevention, in which clients are informed that recurrences of panic, anxiety or avoidance behavior are likely to occur in the future. They are encouraged to view such recurrences as lapses rather than failure, and to reapply their coping skills and reinstitute their practice of interoceptive and *in vivo* exposure.

Science of Exposure Therapy

Exposure therapy has developed over time, originating with graduated imaginal exposure combined with counterconditioning through relaxation (i.e., systematic desensitization) developed by Wolpe (1959). Emotional

processing theory emphasized habituation of fear responding within an exposure trial as a necessary precursor to habituation across treatment sessions, with the aim of long-term corrective learning (Foa and Kozak, 1986; Foa and McNally, 1996). Most recently, we have emphasized optimizing inhibitory learning and its retrieval in ways that are not necessarily dependent on reductions in fear throughout trials of exposure (Craske *et al.*, 2008); we discuss this approach below.

Emotional processing theory emphasizes mechanisms of habituation as precursors to cognitive correction. Specifically, emotional processing theory purports that the effects of exposure therapy derive from activation of a "fear structure" and integration of information that is incompatible with it, resulting in the development of a nonfear structure that replaces or competes with the original one. Incompatible information derives first from within-session habituation, or reduction in fear responding with prolonged exposure to the fear stimulus. Within-session habituation is seen as a prerequisite for the second piece of incompatible information, which derives from between-session habituation over repeated occasions of exposure. Between-session habituation is purported to form the basis for long-term learning and to be mediated by changes in "meaning," or lowered probability of harm (i.e., risk) and lessened negativity (i.e., valence) of the stimulus. Emotional processing theory guides clinicians to focus on the initial elevation of fear followed by within- and between-session reductions in fear as signs of treatment success. Although enticing in its face validity, support for the theory has been inconsistent at best (Craske *et al.*, 2008, 2012). Rather, the evidence suggests that the amount by which fear habituates from the beginning to the end of an exposure practice is not a good predictor of overall outcomes, and

that evidence for between-session habituation is mixed (Craske *et al.*, 2008, 2012).

A return to the science of fear learning and extinction may help to explain the effects of exposure therapy and thereby optimize its implementation. It is now thought that inhibitory learning is central to extinction (Bouton, 1993). Inhibitory pathways are also recognized in the neurobiology of fear extinction (see Sotres-Bayon, Cain, and LeDoux, 2006). Within a Pavlovian conditioning approach, inhibitory learning means that the original association between the conditional stimulus (i.e., the neutral stimulus that is paired with an innately aversive stimulus) and the unconditional stimulus (the innately aversive stimulus) learned during fear conditioning is not erased during extinction, but rather is left intact as a new, secondary learning (i.e., the conditional stimulus no longer predicts the unconditional stimulus) develops (Bouton, 1993). The degree to which inhibitory associations shape fear responding at retest (the index of strength and stability of new “learning”) is independent of fear levels expressed throughout extinction and instead is dependent on factors such as context and time.

Based on the inhibitory retrieval model of extinction, outcomes may be enhanced by strategies that do not rely on fear reduction within a trial of exposure (Craske *et al.*, 2008, 2012). Indeed, fear reduction may become a safety behavior for persons with panic disorder (since fear reduction eradicates the very thing that is feared), such that a more appropriate goal may be to maintain high levels of fear and anxiety in order to disconfirm the expectancy of negative consequences. One translational possibility is “deepened extinction” (Rescorla, 2006), where multiple fear conditional stimuli are first extinguished separately before being combined during extinction, and in animal studies, decreases spontaneous recovery and reinstatement of fear. Indeed, this is what is essentially done when interoceptive