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The Science of Cognitive Therapy

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Cognitive therapy (CT) refers to a family of interventions and a general scientific approach to psychological disorders. This family has evolved from a specific treatment model into a scientific approach that incorporates a wide variety of disorder-specific interventions and treatment techniques. The goal of this article is to describe the scientific approach of CT, review the efficacy and validity of the CT model, and exemplify important differences and commonalities of the CT approaches based on two specific disorders, posttraumatic stress disorder and health anxiety.

Keywords: cognitive therapy; acceptance and commitment therapy; mindfulness; cognitive science; neuroscience

COGNITIVE THERAPY (CT) is one of the big success stories of contemporary psychology. The most recent development in the United Kingdom is a very clear example of the enormous influence CT has gained. In October 2007, the UK health secretary announced that the United Kingdom will spend £300 million ($600 million) to initiate a 6-year program with the goal to provide people with psychological problems, such as anxiety disorders and depression, better access to empirically supported therapies, especially CT. The plan is to train a total of 8,000 new therapists in these evidence-based interventions for mood and anxiety disorders. This change in health care delivery was based on economic data showing that provision of CT for common mental disorders is more cost-efficient than pharmacotherapy or other interventions (for more information, see Rachman & Wilson, 2008). Similarly, the Australian government recommended in 1996 the provision of CT and introduced a plan to provide better access to these services.

Although impressive in magnitude, the recent development in the United Kingdom and other countries is not surprising to us. It appears to be a logical next step from the clear scientific evidence of CT efficacy and effectiveness (e.g., Butler, Chapman, Forman, & Beck, 2006; Hofmann & Smits, 2008) to its dissemination. Unfortunately, the United States, Canada, and other developed countries are still lagging behind other nations, such as the United Kingdom and Australia, in necessary dissemination efforts of CT. Therefore, wider dissemination efforts of CT in the United States and other countries should be a priority for future research.

Moreover, more research is necessary on the mechanisms of treatment change. Although recent studies have provided direct empirical support for some core assumptions of the CT model, a number of authors have criticized its validity. As we outlined

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in an earlier paper (Hofmann & Asmundson, 2008), this critique against CT is factually incorrect and/or based on an incorrect interpretation of the cognitive model. Instead of reiterating and elaborating on these criticisms, we will expand our discussion beyond these criticisms and clarify the scientific approach of CT, review a number of recent studies and exciting new research providing strong support for the CT model, and describe the general and disorder-specific CT models. More specifically, we (a) describe the philosophical roots of CT, (b) explain the interplay of cognition and emotion in CT, (c) specify the treatment goals of CT, (d) explain the hypothesized mechanisms of treatment change, (e) provide an overview of the relationship of cognitive models with medical models and associated diagnostic systems, (f) summarize emerging empirical findings that support postulates of the CT model and address key criticisms noted above, and (g) provide concrete illustrations of important general and disorder-specific considerations of CT. Our intent is to correct some of the misperceptions about modern CT and, importantly, to stimulate continuing research and collegial discussions that will ultimately serve to improve therapeutic tools and outcomes for those whose lives are impacted by psychopathology.

The Model of Scientific Development

CT is not specifically linked to a particular philosophical tradition. The philosophical foundation most closely associated with CT is critical rationalism, an epistemological philosophy (Popper, 1959) that shares its philosophical roots with the natural sciences. The core assumption of critical rationalism is that knowledge can only be gained by attempting to falsify hypotheses that are derived from scientific theories. Based on this philosophy, knowledge is objective and, thereby, shows properties and consequences that are not reducible to whatever one prefers the truth to be. Following the same philosophical principle, patients in CT are encouraged to generate hypotheses based on their beliefs (theories) about the world, themselves, and their future. This approach is combined with the Socratic method, in which a series of questions are posed to help a person determine his or her underlying beliefs. Through a process characterized as "collaborative empiricism," patients falsify hypotheses devised to test dysfunctional beliefs. They are then encouraged to revise their belief system, which results in reduced emotional distress.

The Interplay of Cognition and Emotion in CT

The core model of CT holds that cognitions causally influence emotions and behaviors and, in the case of dysfunctional thoughts and cognitive distortions, contribute to the maintenance of psychopathology. It should be noted that the relationship between emotions and cognitions is bidirectional because changes in emotions can also lead to changes in cognitions. The CT model simply builds on the fact that emotions are strongly, and causally, influenced by the perception of events or situations. This view is in stark contrast to other theorists who reject the notion that cognitions can cause emotions and behaviors (Wilson, 1997; Wilson, Hayes, & Gifford, 1997). For example, Wilson and colleagues have stated that "Cognition plays an important role in the regulation of other forms of behaviors . . . , but it is not a causal role" (p. 56). We agree with Wilson and colleagues in that this issue appears to be the most critical and possibly irreconcilable difference between behavior analysis and CT at a theoretical and philosophical level.

When using cognitive techniques, focus is placed upon the cognitive realm first and foremost, although the physiological, emotional, and behavioral components are clearly recognized for the role that they play. In short, from the CT perspective, it is the way one thinks about a situation or experience that influences the way he or she feels and behaves in the context of that situation or experience. Negative emotions and harmful behaviors are products of dysfunctional thoughts and cognitive distortions. An important aspect of CT is targeting all aspects of psychopathology, regulating emotional distress via interventions that directly target dysfunctional cognitions and related behaviors. A combination of cognitive restructuring techniques and behavioral experiments (i.e., in vivo hypothesis testing), as described above, can be used to modify maladaptive thoughts and cognitive distortions. This serves to increase perceived ability of coping, reduces perceptions of personal vulnerability, and reduces emotional distress.

The techniques of CT are compatible with recent emotion research, as described by Gross's emotion-generative process model (Gross, 1998, 2002; Gross & John, 2003; Gross & Levenson, 1997). Gross's process model of emotions emphasizes the evaluation of external or internal emotional cues. Once these cues are processed, a set of physiological, behavioral, and experiential responses are activated, and these responses are influenced by emotion-regulation tendencies. Based on the time point at which a person engages in emotion regulation, the techniques can be divided into antecedent-focused and response-focused strategies. Antecedent-focused emotion-regulation strategies occur before the emotional response has been fully activated. Examples include cognitive reappraisal, situation modification, and attention deployment. In contrast, response-focused emotion-regulation strategies are attempts to alter

the expression or experience of an emotion after the response tendency has been initiated. Examples include strategies to suppress or tolerate the activated emotional response.

As we have described in detail elsewhere (Hofmann & Asmundson, 2008), a core element of CT is cognitive reappraisal, which is an example of an adaptive antecedent-focused emotion-regulation strategy. Other techniques, including acceptance and mindfulness-based practices, primarily target maladaptive response-focused strategies by discouraging emotional suppression. Many traditional CT protocols include response-focused strategies (e.g., relaxation techniques and other stress-reduction methods), and more recent CT protocols have incorporated mindfulness-based exercises (e.g., Roemer & Orsillo, 2002; Segal, Williams, & Teasdale, 2001).

The Treatment Goals

Emotional disorders are typically associated with negatively valenced emotional responses, such as fear, sadness, anger, and heightened level of distress. The goal of CT is not to eliminate or regulate these emotions in general. Instead, the goal is to foster the abilities of patients to provide for themselves more realistic and accurate appraisals of the situations that they face. Cognitive techniques do not ask patients to think positively but rather more realistically.

As described above, the underlying core model of CT is that cognitions profoundly and causally influence emotions and behaviors and, thereby, contribute to the maintenance of psychopathology. As we have discussed elsewhere (Hofmann & Asmundson, 2008), targeting the way one thinks about emotion-eliciting situations and experiences is different from approaches that attempt to regulate emotion through reduction of experiential avoidance (e.g., by embracing anxiety or pain). These latter strategies are characteristic of those employed by acceptance and commitment therapy (ACT) practitioners (e.g., Hayes, Luoma, Bond, Masuda, & Lillis, 2006); however, they are not incompatible with a CT approach. Indeed, based on evidence that individuals habitually employ either cognitive reappraisal or suppression to regulate emotion (Gross & John, 2003), and that flexibility in emotional expression is important to long-term emotional adjustment (Bonnanno, Papa, Lalande, Westphal, & Cifman, 2004), we strongly urge that future research efforts target the impact of flexibility in application of different emotion-regulation strategies on treatment outcome for various forms of psychopathology.

Hypothesized Mechanism of Treatment Change

In its simplest form, the CT model predicts that changes in cognitions causally lead to changes in behaviors and emotions. When CT was first developed, little empirical support existed for this basic model (Hollon & Beck, 1986). However, as statisticians developed strategies to test for treatment mediation, data has since accumulated to support the cognitive model for the treatment of panic disorder (Hofmann et al., 2007), social anxiety disorder (Hofmann, 2004; Smits, Rosenfield, Telch, & McDonald, 2006), obsessive-compulsive disorder (Moore & Abramowitz, 2007), depression (Kaysen, Scher, Mastnak, & Resick, 2005; Tang, DeRubeis, Beberman, & Pham, 2005), and pain (Price, 2000), to name only a few.

The statistical procedures for mediation have been well defined since the seminal paper by Baron and Kenny (1986); yet, mediation investigations of treatment change are still in their infancy. In contrast to the Baron and Kenny criteria, which outline mediation tests for cross-sectional data, mediation of treatment change requires significantly more complex methodologies. Recently, for example, investigators have proposed criteria to study mediation of change using regression discontinuation and interrupted time series for single-group study designs (Doss & Atkins, 2006), structural equation modeling procedures for longitudinal tests (Cole & Maxwell, 2003), multilevel models (Kenny, Korchmaros, & Bolger, 2003), and linear regression models for randomized controlled trials (RCTs; Kraemer, Wilson, Fairburn, & Agras, 2002).

Although the methodological recommendations are complex and have been published only recently, an impressive number of studies demonstrating cognitive mediation have already been published. For example, the Kraemer et al. (2002) criteria of mediation state that a meditational relationship exists if (a) the proposed mediator correlates with treatment assignment, (b) the mediator has either a main or interactive effect on outcome, and (c) changes in the mediator variable precede changes in the dependent variable. One of us (Hofmann, 2004) adopted these criteria to study cognitive mediation in CT for social anxiety disorder by comparing CT, exposure therapy without explicit cognitive techniques, and a wait-list control condition. In order to examine whether treatment changes in estimation of social cost (how bad it would be if the social situation did not go well) mediated changes in the dependent variable (social anxiety), patients were assessed at pretest, posttest, and follow-up. This design limits the mediation analyses because, in order to conduct an adequate test of mediation, assessments are necessary at time points when the changes in the proposed mediator are believed to causally affect the changes in the dependent variable. Due to design limitations, only pre- to postchanges in the proposed mediator

(estimated social cost) predict pre- to follow-up changes in the dependent variable (social anxiety) were examined. The results showed that short-term changes in estimated social cost mediated later treatment changes in both treatment conditions from pre- to posttest. Only participants who received CT showed continued improvement from posttest to 6-month follow-up. These long-term changes in social anxiety were associated with a reduction of estimated social cost from pre- to posttest. This suggests that cognitive intervention is mediated through changes in estimated social cost, especially among patients who received CT.

Additional evidence for the cognitive mediation model comes from tightly controlled laboratory studies. A case in point is the cognitive model of social anxiety disorder (Clark & Wells, 1995; Hofmann, 2007a; Rapee & Heimberg, 1997). One of the concrete predictions of the model is that, when confronted with social threat, socially anxious individuals focus their attention inwardly onto negative self-focused cognitions, leading to heightened social anxiety and subsequent avoidance behaviors, resulting in the maintenance of the problem. Consistent with this model are correlational and mediation studies showing that successful treatment is associated with decreased self-focused attention (Hofmann, 2000; Wells & Papageorgiou, 1998; Woody, Chambless, & Glass, 1997), which is correlated with changes in social anxiety, especially among individuals who receive cognitive interventions (Hofmann, Moscovitch, Kim, & Taylor, 2004).

A recent laboratory study similarly demonstrated that anxiety in anticipation of a socially threatening situation is cognitively mediated (Schulz, Alpers, & Hofmann, 2008). This study assessed anxious responding during anticipation of public speaking. To examine the role of cognitions as a mediator, the study induced negative self-focused cognitions as compared to relaxation that encouraged participants to focus their attention away from negative cognitions. As predicted, negative self-focused cognitions fully mediated the effects of trait social anxiety on self-reported anxiety and heart rate variability during negative anticipation. Moreover, trait social anxiety predicted increased startle amplitudes.

In summary, a wealth of experimental evidence clearly supports the central assumptions of the cognitive model of anxiety and other disorders. This evidence comes from studies of mediation using both clinical treatment samples as well as samples participating in controlled laboratory investigations. It should be noted that an argument of some critics of CT is that the CT is invalid because treatment component analyses have not consistently demonstrated that the cognitive component is more effective than exposure without explicit cognitive intervention. This is not a valid criticism because a component analysis is neither a necessary nor a sufficient test for the cognitive model (Hofmann, 2008b).

A component analysis cannot answer the question of mediation for a number of methodological and logical reasons. First, the treatment length and the number of treatment components are two confounded variables in component studies. In other words, a 1-hour exposure treatment does not include the same exposure component as a 1-hour exposure therapy with added direct cognitive techniques, simply because the amount of exposure therapy is different in the two treatments. If the amount of exposure time is kept constant, then the treatment with more components will take more time than the treatment with fewer components; because therapist contact time is correlated with treatment success, it is impossible to directly compare the treatments that differ in the number of components. Second, cognitions can change and mediate treatment through a number of ways, not only through direct cognitive challenges. For example, a person with spider phobia who exposes him- or herself to spiders without experiencing any of the feared consequences will show a reduction in harm expectancy, even without explicit cognitive restructuring. Therefore, a trial showing that treatment (or treatment component) X is more efficacious than another treatment (or treatment component) Y simply shows that treatment X reduces symptoms more than treatment Y, but the mechanism of change in treatment X or Y remains unclear. Similarly, a null finding in the efficacy between two treatments does not rule out differences in mediation between the treatments. It is also possible that both or neither treatment components are mediated via variable Z. The crucial question remains—do changes in cognitions (e.g., harm expectancy) mediate changes in symptoms (e.g., fear of spiders)? Accordingly, the critical tests for the cognitive model are mediation studies, not component analyses. This call for mediation analyses is consistent with many other authors (e.g., Hayes et al., 2006; Zettle & Hayes, 1986).

Relationship to the Medical Model and Its Diagnostic System

Many contemporary cognitive models of psychopathology are aligned with medical classification systems of mental disorders, such as that provided by the DSM-IV-TR (American Psychiatric Association, 2000) and the International Classification of

Diseases-10 (ICD-10; World Health Organization, 1992, 1993). Cognitive-behavioral researchers and clinicians have taken advantage of the semblance of order that these atheoretical classification systems offer to the field of psychopathology and, from that order, have developed models for a wide range of conditions. These models most often focus on understanding and explaining psychopathology reflected by specific DSM/ICD diagnostic categories (e.g., social anxiety disorder, generalized anxiety disorder, major depressive disorder), although there are some exceptions. Cognitive models of health anxiety diverge from a criteria-based categorical conceptualization, viewing the cognitive features of conditions characterized by severe health anxiety as occurring along a continuum ranging from adaptive to maladaptive. Likewise, cognitive models of chronic pain behavior are neither rooted in the traditional biomedical model of pain nor DSM-defined pain disorder. They are, instead, integrative models that account for complex interplay between biological, psychological, and sociocultural mechanisms (e.g., Asmundson & Wright, 2004). All cognitive models of psychopathology—whether based on DSM/ICD diagnostic categories, dimensional conceptualization, mechanistic conceptualization, or some other system—serve to guide, and are informed by, research and development of treatment techniques.

General alignment of cognitive models with medical classification systems of mental disorders is not without controversy (e.g., Andersson & Ghaderi, 2006). A number of specific issues are at the heart of the controversy but, in essence, it revolves around whether classifying people into criteria-based psychiatric diagnostic categories is compatible with behaviorism and the practice of behavioral analysis. There are also numerous other issues being discussed and debated as the process of revising the DSM-IV-TR and ICD-10 takes flight (e.g., Andrews, Anderson, Slade, & Sunderland, 2008). Despite the issues of current discussion and debate, the evidence is clear that the development and refinement of cognitive models for various DSM-IV-TR/ICD-10 diagnoses has permitted application of specific treatment techniques across a diverse range of psychopathologies for which outcomes have been favorable (see below). Moreover, another benefit of this alignment is evidenced in the development of novel ways for combining CT with short-term pharmacotherapy in exposure treatment (Hofmann, 2007b; Hofmann, Pollack, & Otto, 2006). Approaches such as these have substantial potential for further increasing the effectiveness of the treatments available for various forms of psychopathology.

Efficacy of CT

A review of the efficacy of CT for mental disorders would easily fill a textbook. A recent review of 16 meta-analytic studies found large controlled effect sizes for CT for unipolar depression, generalized anxiety disorder, panic disorder with or without agoraphobia, social phobia (i.e., social anxiety disorder), PTSD, and childhood depressive and anxiety disorders, and medium controlled effect sizes for CT of chronic pain, childhood somatic disorders, marital distress, and anger (Butler et al., 2006). Furthermore, large uncontrolled effect sizes were found for bulimia nervosa and schizophrenia. Similarly, a recent review of methodologically rigorous, randomized placebo-controlled studies of anxiety disorders indicated that CT yielded medium to large effect sizes over placebo (Hofmann & Smits, 2008). Large effect sizes were observed for obsessive-compulsive disorder and acute stress disorder. Moreover, the various CT treatment protocols showed clear disorder specificity, because depression changed to a significantly lesser degree than the targeted anxiety disorder. In sum, CT is clearly an effective treatment for a range of psychopathology. The effects of CT have been replicated many times in well-controlled studies. As a result, many CT protocols have been classified as empirically supported treatments as defined by the APA Division 12 Task Force in 1995 (Chambless et al., 1998).

In contrast, much less is known about the efficacy of ACT. A recent review by Öst (2008) examined 13 RCTs in ACT and dialectic behavior therapy (DT), 1 in cognitive behavioral analysis system of psychotherapy, and 2 in integrative behavioral couple therapy. The results showed that the RCTs used a significantly less stringent research methodology. The mean effect size of the available studies was moderate for both ACT and DT. None of these therapies fulfilled the criteria for empirically supported treatments as defined by the Division 12 Task Force.

The Biological Correlates of Cognitions and CT

The cognitive and affective neuroscience literature convincingly shows that changes in cognitions due to CT are associated with changes in brain activity. The literature on the effects of cognitions on brain activity is enormous, and we can provide only a glimpse into this exciting literature.

EFFECTS OF COGNITIONS ON BRAIN ACTIVITY

A number of studies from the field of neuroscience support the notion that changes in cognitions and conscious self-regulation of emotions directly influence the electrochemical dynamics in the brain (e.g., Beauregard, Levesque, & Bourgouin, 2001; Blair

et al., 2007; Bunge, Ochsner, Desmond, Glover, & Gabrieli, 2001; LeDoux, 1996; Ochsner, Bungem, Gross, & Gabrieli, 2002; Ochsner & Gross, 2005; Ochsner et al., 2004). For example, Ochsner and colleagues (2002) observed a causal relationship between reappraisal of negative stimuli, improvement in mood, and brain activity patterns. In this study, negatively valenced and neutral pictures were presented to 15 healthy female participants. For each trial, participants were instructed to view the picture and fully experience any emotional response it might elicit. The picture remained on the screen for an additional period of time with the instructions either to attend or to reappraise the stimulus. On the attend trials, participants were asked to be aware of the picture, but not to try to modify any feelings induced by the picture. As part of the reappraisal instructions, participants were asked to reinterpret the negative picture so that it no longer generated the negative emotional response. Reappraisal of the negative pictures lessened negative affect and was associated with increased activity in the dorsal and ventral regions of the left lateral prefrontal cortex (LPFC) and the dorsal medial prefrontal cortex (MPFC), but decreased activity in the amygdala and orbitofrontal cortex. The medial orbitofrontal cortex (MOFC) showed greater activation for most negative pictures on attend than on reappraise trials. Furthermore, increased activation in the ventrolateral prefrontal cortex (VLPFC) was correlated with decreased activation in the amygdala, suggesting that the VLPFC may play an important role in conscious and voluntary regulation of emotional processes.

These findings seem to suggest that reappraisal instructions modulate the emotion processes implemented in the amygdala and MOFC and are involved in the evaluation of the emotional significance and contextual relevance of the stimulus. The finding that ventral LPFC activation was inversely associated with activation of the amygdala and MOFC suggests that the VLPFC may play an important role in conscious and voluntary regulation of emotional processes. The data further suggest that limbic and ventral prefrontal structures generate negative affect in response to certain stimuli, whereas dorsal prefrontal regions may be engaged through reappraisal techniques to dampen this outflow from more ventral structures.

In a later study by the same research group (Ochsner et al., 2004), 24 healthy female participants were instructed to up or down regulate negative emotion either by focusing internally on the self-relevance of aversive scenes or by focusing externally on alternative meanings of picture actions and their situational contexts. The functional magnetic resonance imaging (fMRI) data showed that amygdala activation was modulated up or down in accordance with the regulatory goal. Whereas up regulation recruited the anterior cingulate cortex, MPFC, left LPFC, and the left amygdala, down regulation recruited the left LPFC and the left orbitofrontal cortex. Moreover, self-focused regulation was associated with MPFC regions, whereas situation-focused regulation recruited lateral prefrontal regions. These findings indicate that there are both common and distinct neural systems that support various forms of reappraisal. The specific prefrontal systems that modulate the amygdala appear to be contingent on the regulatory goal and strategy that is used. In sum, these findings demonstrate that cognitive reappraisal selectively alters the way the brain processes and reacts to emotional stimuli (see Beauregard, 2007, for a review).

EFFECTS OF CT ON BRAIN ACTIVITY

A number of studies have examined changes in brain activity from before to after CT. In fact, enough data have accumulated for no fewer than three systematic review articles on the effects of CT on changes in brain functioning (Frewen, Dozois, & Lanius, 2008; Linden, 2006; Roffman, Marci, Glick, Dougherty, & Rauch, 2005). Studies included in these meta-analyses examined CT for obsessive-compulsive disorder (Baxter et al., 1992; Nakao et al., 2005; Nakatani et al., 2003; Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996), social anxiety disorder (Furmark et al., 2002), spider phobia (Paquette et al., 2003; Straube, Glauer, Dilger, Mentzel, & Miltner, 2006), panic disorder (Prasko et al., 2004), PTSD (Felmingham et al., 2007), and major depressive disorder (Goldapple et al., 2004). In addition, two studies examined neural changes as a result of interpersonal therapy for depression (Brody et al., 1998; Martin, Martin, Rai, Richardson, & Royall, 2001).

When interpreting these results, it should be taken into consideration that all of the studies were based on a relatively small number of participants and used different techniques to study changes in brain activation, ranging from glucose metabolism, blood flow, and task-induced blood oxygenation (i.e., the BOLD signal of fMRI). These physiologic parameters are governed by different regulatory systems and are, therefore, prone to influences from different confounding variables. However, despite the differences in methodology and the preliminary nature of these studies, a number of consistent findings emerged. The studies collectively support the notion that response to CT for mood and anxiety disorders is associated with changes in brain regions that are consistent with neural models

of affective regulation and self-regulation (Davidson, 2000; Lieberman, 2007; Ochsner & Gross, 2005), and suggest that CT alters brain functioning associated with problem solving, affect regulation, and self-referential and relational processing. For example, CT is associated with changes in anterior and posterior cortical midline structures that are related to self-referential processing (Northoff et al., 2006). CT was further associated with changes in regions that are involved in the regulation of negative affect (Ochsner & Gross, 2005), including the ventral and dorsal anterior cingulate cortex, MPFC, and the right ventrolateral and inferior frontal cortex. Moreover, activity within the left dorsolateral prefrontal cortex was modulated by CT for mood and anxiety disorders, suggesting that CT leads to increased executive functioning by enhancing deployment of working memory in problem-solving solutions to difficult emotional and stressful situations (e.g., Frewen et al., 2008). For a review of the recent literature on the neurobiological correlates of cognitions and CT, see Hofmann, Ellard and Siegle (in press)

The Role of Cognitions in Extinction and Conditioning Processes

Even basic learning is moderated and possibly mediated via cognitions. In the case of fear learning, it has been shown that fear can be acquired without directly experiencing the conditioned stimulus (CS) and unconditioned stimulus (US). For example, Rhesus monkeys learn quickly to acquire a fear of snakes simply by observing another monkey respond fearfully to them. Similarly, observing another monkey responding nonfearfully can effectively prevent the acquisition of this fear following later exposure to models behaving fearfully (e.g., Mineka & Zinbarg, 2006). Therefore, fear can be acquired by observing two events contiguous in time—the snake and the fear response to the snake exhibited by another monkey. This points to the involvement of higher-order cognitive processes.

Similarly, extinction learning involves cognitive processes. Evidence against the notion that extinction is merely due to an unlearning or erasure of previously acquired fear include the observations that the original fear response returns after the passage of time (spontaneous recovery; e.g., Robbins, 1990), after a change of context from the extinction context (renewal; e.g., Bouton & Bolles, 1979; Rodriguez, Craske, Mineka, & Hladeck, 1999), or after unsignaled presentations of the US that occur within the context of the retention test (reinstatement; e.g., Bouton & Swartzentruber, 1991; Dirikx, Hermans, Vansteenwegen, Baeyens, & Eelen, 2004; Hermans et al., 2005; Rescorla & Heth, 1975).

Modern learning theories of extinction assume that conditioning happens as participants form representations of the relevant cues (CS and US) and situational contexts, and as they acquire information about the association between these cues and the situations (see Myers & Davis, 2002, for a review). These associations can be either excitatory (i.e., activation of one representation activates another) or inhibitory (i.e., activation of one representation inhibits activation of another). Acquisition of conditioned responses is explained by the formation of an excitatory association between representations of the CS and the US. The US representation is activated indirectly through its association with the CS representation that, in turn, triggers the conditioned response. Extinction is assumed to proceed through multiple mechanisms (Myers & Davis, 2002) that also include new learning that inhibits the excitatory association between the CS and the US (e.g., Bouton, 1993; Myers, Ressler, & Davis, 2006). As part of this new form of learning, the participant changes the CS–US contingency in such a way that the CS no longer signals an aversive event and thereby inhibits the expression of the fear response (e.g., Bouton, 1993; Myers & Davis, 2002).

This brief review of the animal literature suggests that fear acquisition, extinction learning, and exposure therapy involve higher-order cognitive processes. Thus, it can be concluded that a reduction in US expectancy mediates extinction learning and exposure therapy, as well as CT (Hofmann, 2008a). This view is also consistent with a number of earlier psychotherapy models that emphasize the prediction of harm during exposure procedures (Foa & Kozak, 1986).

Recognizing the importance of cognitive processes in fear acquisition, extinction, and exposure therapy offers a new possibility for intervention research, namely, to improve the effects of exposure therapy with pharmacological intervention that are believed to act as cognitive enhancers. Animal research has shown that fear and extinction learning are both blocked by antagonists at the glutamatergic N-methyl-D-aspartate (NMDA) receptor, which is critically involved in learning and memory. For example, intra-amygdala infusions of an NMDA receptor antagonist shortly before extinction training dose-dependently blocks extinction (Falls, Miserendino, & Davis, 1992). Moreover, d-cycloserine (DCS), a partial NMDA agonist dose-dependently enhances extinction in rats (Ledgerwood, Richardson, & Cranney, 2003, 2004; Walker, Ressler, Lu, & Davis, 2002). Similarly, DCS has been shown to enhance exposure therapy of height phobia (Ressler et al., 2004), social anxiety disorder

(Guastella et al., 2008; Hofmann, Meuret, et al., 2006), and obsessive-compulsive disorder (Kushner et al., 2007; Wilhelm et al., 2008).

The Family of CTs

Psychology is most commonly defined in contemporary textbooks as the scientific study of behavior and mental processes. CT, which is rooted in behavioral and cognitive sciences, targets psychotherapy as the object of scientific study. Since its initial development, CT has undergone extensive scientific scrutiny through comparisons in RCTs, component analyses, and mediation analyses (as discussed above). Predictions of aspects of the intervention have been tested in laboratory experiments, and knowledge gained about the psychopathology of a disorder has been integrated into existing CT protocols for specific disorders. Therefore, the evolution of CT reflects the progress of psychology as a science.

CT is not a single-treatment protocol, and it is inappropriate to talk about *the* cognitive therapy or *the* cognitive model. The specific model and treatment techniques depend on the symptoms that are targeted. Therefore, as researchers and practitioners, we are forced to select a specific CT model for specific harmful dysfunctions to exemplify the link between the theory and the specific treatment techniques. More recently, some authors have begun to develop unified CT protocols that cut across diagnostic categories that are focused on specific dysfunctional emotion-regulation strategies (e.g., Barlow, Allen, & Choate, 2004). Thus, CT describes a family of interventions that share the same basic elements of the CT model that focus on the importance of cognitive processes for emotion regulation (for a recent review, see Hofmann, 2011).

We do not consider this to be a weakness of CT. Instead, it is a sign that CT is a maturing scientific discipline rather than an assembly of specific treatment techniques. The reason for this is the strong commitment to the scientific enterprise and openness to translate and integrate new empirical findings of the psychopathology of a disorder into a working CT model of the disorder. This is an ongoing and iterative process; for example, CT for a specific anxiety disorder 10 years ago looks very different from CT for the same disorder today. Although the core assumption of CT remains the same—changes in cognitions causally predict changes in negative emotions and maladaptive behavior—the specific treatment techniques have certainly changed and will continue to change as basic research on psychopathology progresses.

To illustrate the link between the underlying CT model and the specific models that are informed by empirical research and specific treatment techniques, we have chosen to highlight PTSD and health anxiety. We have selected these conditions for several reasons. The CT model of PTSD provides an excellent illustration of how cognitions add explanatory value to simple conditioning learning. The CT model of health anxiety clearly illustrates that it is cognitive restructuring, not symptom reduction or elimination, that is important in reducing psychopathology. Both examples further illustrate two crucial points that are often questioned by critics: First, there is a direct causal link between cognitions and emotions, and second, changing maladaptive cognitions leads to improvements in psychopathology.

Examples of CT Models and Treatments

CT, as with every advanced science, has many subdisciplines. All CT approaches are connected by the same basic model (i.e., cognitions causally affect emotions and behaviors), but the various techniques and models show a number of unique features, depending on the targeted disorder.

Although traditional CT protocols are more effective than placebo treatments, there is clearly still room for improvement. For example, the average effect size of CT for anxiety disorders is 0.73 (Hofmann & Smits, 2008). Since CT was first formulated, knowledge from the experimental literature has been translated into improved therapeutic techniques that have since been incorporated into improved treatment protocols to specifically target the maintenance factors of a disorder. A comprehensive review of these modifications is outside the scope of this paper. Below are two recent examples.

THE COGNITIVE MODEL OF PTSD

PTSD is only one of many examples that illustrate how cognitive theory provides an alternative and more heuristically useful model for a disorder that is more in line with empirical data than the behavioristic models of fear acquisition and extinction (Mineka & Zinbarg, 2006). Pavlovian fear conditioning is an obvious animal model for the study of PTSD in humans (e.g., Charney, 2004; Pitman, 1997). After repeated pairing of a CS with a US, the CS comes to elicit conditioned fear responses. Similarly, a soldier in combat may associate the sound of a helicopter (CS) with a severe traumatic event (US), leading to a conditioned fear response when exposed to stimuli that resemble the original CS. This model assumes that, as with fear extinction training in animals, the association between a CS and a US can be weakened if the CS no longer predicts the US (e.g., Milad, Rauch, Pitman, & Quirk, 2006). However, such a simple animal model does not explain other prominent features of PTSD in humans, ranging from depression and feelings of guilt to social

isolation and emotional numbing. Therefore, cognitive models of PTSD have been formulated to account for these and other issues (e.g., Ehlers & Clark, 2000; Resick & Schnicke, 1993).

The most recent version of the cognitive model of PTSD has been formulated by Ehlers and Clark (2000). This model posits that individuals develop persistent PTSD if they process the traumatic event and/or its sequelae in a way that produces a sense of a serious current threat. This sense of current threat is thought to be the result of (a) excessively negative appraisals of the trauma and/or its sequelae, and (b) a disturbance of the memory processing of the trauma due to poor elaboration and appraisal. Once these processes are activated, the perception of current threat is then accompanied by arousal, anxiety, other emotional responses, intrusions, and reexperiencing symptoms. Persistent reexperiencing symptoms are assumed to be caused by deficits in the trauma memory. The model further assumes that the perceived threat motivates a series of behavioral and cognitive responses that are intended to reduce perceived threat and distress in the short term. However, these maladaptive attempts do not resolve the problems but, rather, prevent cognitive change and thereby contribute to the maintenance of the problem.

COGNITIVE THERAPY OF PTSD

This model has concrete treatment implications (Ehlers, Clark, Hackmann, McManus, & Fennell, 2005). Based on this model, effective treatment of PTSD will have to identify the relevant appraisals, memory characteristics and triggers, and the cognitive and behavioral variables that maintain PTSD. The specific treatment techniques to achieve this objective include strategies to (a) modify excessively negative appraisals of the trauma and/or its sequelae by examining the meaning of the moments of greatest distress in the trauma memory (the *hot spots*), (b) elaborate the trauma memories and discriminate the triggers that lead to the reexperiencing of the trauma, and (c) eliminate dysfunctional cognitive factors that lead to the maintenance of PTSD.

A cognitive treatment protocol with these cognitive strategies shows impressive results for treating chronic PTSD (Ehlers et al., 2005). As compared to a wait-list control group, the treatment led to large reductions in PTSD symptoms, disability, depression, and anxiety at posttest and 6-month follow-up. The authors conducted a consecutive case series analysis with 20 participants with PTSD and also an RCT comparing 14 participants with PTSD who underwent cognitive therapy and 14 individuals with PTSD assigned to a wait-list control group. The treatment and the waiting period lasted for 3 months. The treatment consisted of 12 weekly treatment sessions (the first session lasted 90 minutes, and remaining sessions lasted 60 minutes). The assessment instruments included standard self-report and independent evaluator assessments, including the Post-traumatic Diagnostic Scale (Foa, Cashman, Jaycox, & Perry, 1997) and the clinician-administered PTSD scale for DSM-IV (Blake et al., 1995). The intent-to-treat effect sizes for the change in PTSD symptoms from pre- to posttreatment ranged between 2.07 and 2.82. The controlled effect sizes for the treatment versus the wait-list group at posttreatment ranged between 2.18 and 2.25. Treatment outcome was positively associated with changes in dysfunctional posttraumatic cognitions (Ehlers et al., 2005). A more recent study further showed that cognitive changes predict disorder-specific PTSD symptoms above and beyond what could be predicted by the initial symptom levels or other commonly found predictors of PTSD (Ehring, Ehlers, & Glucksman, 2008).

THE COGNITIVE MODEL OF HEALTH ANXIETY

Health anxiety refers to anxiety stemming from the belief that bodily "noise" (i.e., bodily sensations and/or changes) are indicative of disease. The extent to which one experiences health anxiety varies considerably from person to person. Like other forms of anxiety, health anxiety can serve an adaptive function in that it sometimes motivates us to seek medical care when such care is needed. Potentially maladaptive expressions of health anxiety, including full and abridged forms of hypochondriasis, typically occur when the anxiety experienced is out of proportion with the objective degree of medical risk. Health anxiety often arises when a person is under stress, seriously ill, or recovering from a serious illness, or has suffered the loss of a family member (Barsky & Klerman, 1983).

The cognitive model of health anxiety is supported by a good deal of empirical research (see Taylor & Asmundson, 2004, for a review) and, importantly, has led to effective treatment (Salkovskis, Warwick, & Deale, 2003; Taylor & Asmundson, 2004). Health anxiety is posited to arise from dysfunctional beliefs about sickness, health, and health care; in particular, people with severe health anxiety are convinced that the bodily "noise" that they experience is due to some sort of serious physical malady rather than somatic sensations arising from benign bodily perturbations (e.g., trembling associated with muscle tension, a blemish on the skin), symptoms of minor disease (e.g., chest pain associated with dyspepsia), and autonomic nervous system arousal (Taylor & Asmundson, 2004). As a consequence of disease conviction, people with severe health anxiety become preoccupied with, and fearful of, the

possibility of having to suffer the agony of having a serious disease. Recurring images of disease and death, often intrusive in nature, are not uncommon in people with severe health anxiety (Warwick & Salkovskis, 1989).

Along with disease conviction and preoccupation, other dysfunctional beliefs (e.g., viewing one's self as weak and vulnerable; a need for absolute certainty regarding medical test results) motivate maladaptive coping behaviors (e.g., reassurance seeking, recurrent checking of the body or medical information, avoidance). The cognitions and behavioral responses associated with severe health anxiety are intended to reduce perceived threat and distress; however, while effective in the short term, these responses typically prevent cognitive change and thereby serve to perpetuate and often exacerbate anxiety.

COGNITIVE THERAPY OF HEALTH ANXIETY

The cognitive model of health anxiety has a number of specific treatment implications. In essence, CT for severe health anxiety begins with identification of dysfunctional beliefs, including those regarding the meaning of bodily "noise" and related issues (e.g., vulnerability to disease), triggers of these beliefs and associated anxiety, and the behaviors that maintain health anxiety. Once these factors have been identified, the therapist works collaboratively with the individual to discover noncatastrophic (benign) alternative explanations for bodily noise, and then applies behavioral exercises (e.g., exposure, response prevention) as a means of facilitating belief change (e.g., Taylor & Asmundson, 2004). It is important to note that the goal of CT for severe health anxiety is not to reduce bodily noise but, rather, to find alternative and adaptive non-disease-related explanations for the bodily noise.

Many uncontrolled trials have suggested that CT can effectively reduce severe health anxiety (e.g., Martinez & Botella, 2005; Stern & Fernandez, 1991). Trials comparing CT to wait-list controls, other treatment conditions, and medical treatment as usual have also produced results indicative of the superiority of CT (e.g., Barsky & Ahern, 2004; Clark et al., 1998; Warwick, Clark, Cobb, & Salkovskis, 1996). In a recent meta-analytic review of 25 treatment trials of full-blown or abridged hypochondriasis (Taylor, Asmundson, & Coons, 2005), we found that effect sizes were larger for all psychosocial interventions (e.g., psychoeducation, exposure and response prevention, cognitive therapy, CT, behavioral stress management) than for wait-lists. In fact, pre- to posttreatment effect sizes from trials of patients with full hypochondriasis were larger for CT (i.e., a combination of cognitive and behavioral interventions; effect size = 2.05) compared to either cognitive or behavioral intervention alone (cognitive therapy effect size = .83; exposure and response prevention effect size = 1.00) or pharmacotherapy (effect size range = 1.07 for nefazadone to 1.92 for fluoxetine). This suggests that the combination of cognitive intervention strategies and behavioral experiments, the latter of which serve as a means of hypothesis testing to facilitate revision of maladaptive beliefs, is more effective than cognitive or behavioral intervention alone. The meta-analytic findings also indicated that, for the psychosocial interventions, gains were most likely to be maintained at 12-month follow-up for those who received CT (mean pre- to follow-up effect size = 1.74).

Discussion

CT is a general scientific approach to psychological disorders that has been the foundation of a wide variety of psychological treatments. The overarching principle of these interventions is that cognitions causally influence emotional experiences and behaviors. We reviewed two prominent CT models to illustrate the significant variations in the basic assumptions of the maintaining factors of the disorders and the differences in the disorder-specific treatment techniques. The Ehlers and Clark (2000) CT model of PTSD assumes that dysfunctional cognitive processing of a past traumatic event is causally linked to the current emotional state associated with this event. As a result, the CT techniques focus on the reappraisal and the memory of the trauma. In contrast, the CT model of health anxiety assumes that it is the dysfunctional cognitive processes of a multitude of current or future experiences, rather than the dysfunctional processing of an identifiable event in the past, that is associated with emotional distress and maladaptive behaviors. This model further assumes that many different harmless physical sensations or minor and benign unexplained pains or aches (bodily "noise") are consistently interpreted as being indicative of a serious physical disease in the present or future. Characteristic features of health anxiety include disease conviction and preoccupation, as well as other dysfunctional beliefs that motivate maladaptive coping behaviors. Similar to PTSD, the cognitions and behavioral responses associated with severe health anxiety are intended to reduce perceived threat and distress; however, although effective in the short term, these responses typically prevent cognitive change and thereby serve to perpetuate and often exacerbate anxiety. Despite the differences in the cognitive conceptualization of, and approach to treating, PTSD and health anxiety, they are firmly rooted within the basic CT approach—namely, that dysfunctional cognitions are causally

linked to emotional distress, and that correcting dysfunctional cognitions results in improvement of emotional distress and maladaptive behaviors.

CT has had an enormous influence on contemporary psychology. Aaron T. Beck received the Lasker Award in 2006, the most prestigious medical prize that is often bestowed to individuals who later win the Nobel Prize. Joseph L. Goldstein, the chairman of the Lasker jury noted that "cognitive therapy is one of the most important advances—if not the most important advance—in the treatment of mental diseases in the last 50 years" (Altman, 2006). We fully agree with the chairman's conclusion. Although there is still room for improvement, CT is efficacious for treating virtually all forms of psychopathology. Moreover, recent mediation studies, experimental studies, and neuroimaging studies in affective neuroscience clearly support the basic model of CT. Still, proponents of the "new wave" movement have criticized and continue to criticize CT on both theoretical and empirical grounds. As noted at the outset of this paper, we had several intents. First, we wanted to demonstrate that the "new wave" criticisms are based on misinterpretations and misunderstandings of CT; in our opinion, the data speak clearly in support of the CT model. Second, we wanted to add our voice to collegial discussions that serve to stimulate continuing research toward improving available evidence-based/empirically supported interventions, and outcomes, for the many people whose lives are impacted by psychopathology; now, we look forward to a positive outcome in this regard.

References

Altman, L. K. (2006, September 17). Psychiatrist is among five chosen for medical award. *The New York Times*. Retrieved from http://www.nytimes.com/2006/09/17/health/17lasker.html

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Andersson, G., & Ghaderi, A. (2006). Overview and analysis of the behaviourist criticism of the diagnostic and statistical manual of mental disorders (DSM). *Clinical Psychologist*, *10*, 67–77.

Andrews, G., Anderson, T. M., Slade, T., & Sunderland, M. (2008). Classification of anxiety and depressive disorders: Problems and solutions. *Depression and Anxiety*, *25*, 274–281.

Asmundson, G. J. G., & Wright, K. D. (2004). The biopsychosocial model of pain. In T. Hadjistavropoulos, & K. D. Craig (Eds.), *Pain: Psychological perspectives* (pp. 35–57). Hillsboro, NJ: Erlbaum.

Barlow, D. H., Allen, L. B., & Choate, M. L. (2004). Toward a unified treatment for emotional disorders. *Behavior Therapy*, *35*, 205–230.

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1182.

Barsky, A. J., & Ahern, D. K. (2004). Cognitive behavior therapy for hypochondriasis: A randomized controlled trial. *Journal of the American Medical Association*, *291*, 1464–1470.

Barsky, A. J., & Klerman, G. L. (1983). Overview: Hypochondriasis, bodily complaints, and somatic styles. *American Journal of Psychiatry*, *140*, 273–283.

Baxter, L. R., Schwartz, J. M., Bergman, K. S., Szuba, M. P., Guze, B. H., Mazziotta, J., . . . & Phelps, M. E. (1992). Caudate glucose metabolic rate changes with both drug and behavior therapy for obsessive-compulsive disorder. *Archives of General Psychiatry*, *49*, 681–689.

Beauregard, M. (2007). Mind does really matter: Evidence from neuroimaging studies of emotional self-regulation, psychotherapy, and placebo effect. *Progress in Neurobiology*, *81*, 218–236.

Beauregard, M., Levesque, J., & Bourgouin, P. (2001). Neural correlates of conscious self-regulation of emotion. *Journal of Neuroscience*, *21* RC165 (1–6).

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

Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Gusman, F. D., Charney, D. S., et al. (1995). The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress*, *8*, 75–90.

Bonanno, G. A., Papa, A., Lalande, K., Westphal, M., & Coifman, K. (2004). The importance of being flexible: The ability to both enhance and suppress emotional expression predicts long-term adjustment. *Psychological Science*, *15*, 482–487.

Bouton, M. E. (1993). Context, time, and memory retrieval in the interference paradigms of Pavlovian learning. *Psychological Bulletin*, *114*, 80–99.

Bouton, M. E., & Bolles, R. C. (1979). Role of conditioned contextual stimuli in reinstatement of extinguished fear. *Journal of Experimental Psychology: Animal Behavior Processes*, *5*, 368–378.

Bouton, M. E., & Swartzentruber, D. (1991). Sources of relapse after extinction in Pavlovian and instrumental learning. *Clinical Psychology Review*, *11*, 123–140.

Brody, A. L., Saxena, S., Schwartz, J. M., Stoessel, P. W., Maidment, K., Phelps, M. E., . . . & Baxter, L. R. (1998). FDG-PET predictors of response to behavioral therapy and pharmacotherapy in obsessive compulsive disorder. *Psychiatry Research*, *91*, 127–139.

Bunge, S. A., Ochsner, K. N., Desmond, J. E., Glover, G. H., & Gabrieli, J. D. E. (2001). Prefrontal regions involved in keeping information in and out of mind. *Brain*, *124*, 2074–2086.

Butler, A. C., Chapman, J. E., Forman, E. M., & Beck, A. T. (2006). The empirical status of cognitive-behavioral therapy: A review of meta-analyses. *Clinical Psychology Review*, *26*, 17–31.

Chambless, D. L., Baker, M. J., Baucom, D. H., Beutler, L. E., Calhoun, K. S., Crits-Christoph, P., . . . & Woody, S. R. (1998). Update on empirically validated therapies, II. *The Clinical Psychologist*, *51*, 3–16.

Charney, D. S. (2004). Psychobiological mechanisms of resilience and vulnerability: Implications for successful adaptation to extreme stress. *American Journal of Psychiatry*, *161*, 195–216.

Clark, D. M., Salkovskis, P. M., Hackmann, A., Wells, A., Fennell, M., Ludgate, J., . . . & Gelder, M. (1998). Two psychological treatments for hypochondriasis: A randomised controlled trial. *British Journal of Psychiatry*, *173*, 218–225.

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York: Guilford Press.

Cole, D. A., & Maxwell, S. E. (2003). Testing mediational models with longitudinal data: Questions and tips in the use of structural equation modeling. *Journal of Abnormal Psychology*, *112*, 558–577.

Davidson, R. J. (2000). Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience. *Psychological Bulletin*, *126*, 890–909.

Dirikx, T., Hermans, D., Vansteenwegen, D., Baeyens, F., & Eelen, P. (2004). Reinstatement of extinguished conditioned responses and negative stimulus valence as a pathway to return of fear in humans. *Learning and Memory*, *11*, 549–554.

Doss, B. D., & Atkins, D. C. (2006). Investigating treatment mediators when simple random assignment to a control group is not possible. *Clinical Psychology: Science and Practice*, *13*, 321–336.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of post-traumatic stress disorder. *Behaviour Research and Therapy*, *38*, 319–345.

Ehlers, A., Clark, D. M., Hackmann, A., McManus, F., & Fennell, M. (2005). Cognitive therapy for post-traumatic stress disorder: Development and evaluation. *Behaviour Research and Therapy*, *43*, 413–431.

Ehring, T., Ehlers, A., & Glucksman, E. (2008). Do cognitive models help in predicting the severity of posttraumatic stress disorder, phobia, and depression after motor vehicle accidents? A prospective longitudinal study. *Journal of Consulting and Clinical Psychology*, *76*, 219–230.

Falls, W. A., Miserendino, M. J. D., & Davis, M. (1992). Extinction of fear-potentiated startle: Blockade by infusion of an NMDA antagonist into the amygdala. *Journal of Neuroscience*, *12*, 854–863.

Felmingham, K., Kemp, A., Williams, L., Das, P., Hughes, G., Peduto, A., . . . & Bryant, R. (2007). Changes in anterior cingulate and amygdala after cognitive behavior therapy of posttraumatic stress disorder. *Psychological Science*, *18*, 127–129.

Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment*, *9*, 445–451.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, *99*, 20–35.

Frewen, P. A., Dozois, D. J. A., & Lanius, R. A. (2008). Neuroimaging studies of psychological interventions for mood and anxiety disorders: Empirical and methodological review. *Clinical Psychology Review*, *28*, 229–247.

Furmark, T., Tillfors, M., Marteinsdottir, L., Fischer, H., Pissiota, A., Langstrom, B., . . . & Fredrikson, M. (2002). Common changes in cerebral blood flow in patients with social phobia treated with citalopram or cognitive-behavioral therapy. *Archives of General Psychiatry*, *59*, 425–433.

Goldapple, K., Segal, Z., Garson, C., Lau, M., Bieling, P., Kennedy, S., . . . & Mayberg, H. (2004). Modulation of cortical-limbic pathways in major depression: Treatment-specific effects of cognitive behavioral therapy. *Archives of General Psychiatry*, *61*, 34–41.

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

Gross, J. J. (2002). The emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology*, *39*, 281–291.

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

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

Guastella, A. J., Richardson, R., Lovibond, P. F., Rapee, R. M., Gaston, J. E., Mitchell, P., . . . & Dadds, M. R. (2008). A randomized controlled trial of D-cycloserine enhancement of exposure therapy for social anxiety disorder. *Biological Psychiatry*, *63*, 544–549.

Hayes, S. C., Luoma, J. B., Bond, F. W., Masuda, A., & Lillis, J. (2006). Acceptance and commitment therapy: Model, processes, and outcomes. *Behaviour Research and Therapy*, *44*, 1–26.

Hermans, D., Dirikx, T., Vansteenwegenin, D., Baeyens, F., Van den Bergh, O., & Eelen, P. (2005). Reinstatement of fear responses in human aversive conditioning. *Behaviour Research and Therapy*, *43*, 533–551.

Hofmann, S. G. (2000). Self-focused attention before and after treatment of social phobia. *Behaviour Research and Therapy*, *38*, 717–725.

Hofmann, S. G. (2004). Cognitive mediation of treatment change in social phobia. *Journal of Consulting and Clinical Psychology*, *72*, 392–399.

Hofmann, S. G. (2007a). Cognitive factors that maintain social anxiety disorder: A comprehensive model and its treatment implications. *Cognitive Behaviour Therapy*, *36*, 193–209.

Hofmann, S. G. (2007b). Enhancing exposure-based therapy from a translational research perspective. *Behaviour Research and Therapy*, *45*, 1987–2001.

Hofmann, S. G. (2008a). Cognitive processes during fear acquisition and extinction in animals and humans: Implications for exposure therapy of anxiety disorders. *Clinical Psychology Review*, *28*, 200–211.

Hofmann, S. G. (2008b). Common misconceptions about cognitive mediation of treatment change: A commentary to Longmore and Worrell (2007). *Clinical Psychology Review*, *28*, 67–70.

Hofmann, S. G. (2011). *An introduction to modern CT; Psychological solutions to mental health problems*. Oxford, UK: Wiley.

Hofmann, S. G., & Asmundson, G. J. (2008). Acceptance and mindfulness-based therapy: New wave or old hat? *Clinical Psychology Review*, *28*, 1–16.

Hofmann, S. G., Ellard, K. K., & Siegle, G. J. (in press). Neurobiological correlates of cognitions in fear and anxiety: A cognitive-neurobiological information-processing model. *Cognition and Emotion*.

Hofmann, S. G., Meuret, A. E., Rosenfield, D., Suvak, M. K., Barlow, D. H., Gorman, J., . . . & Woods, S. W. (2007). Preliminary evidence for cognitive mediation during cognitive behavioral therapy for panic disorder. *Journal of Consulting and Clinical Psychology*, *75*, 374–379.

Hofmann, S. G., Meuret, A. E., Smits, J. A. J., Simon, N. M., Pollack, M. H., & Eisenmenger, K. (2006). Augmentation of exposure therapy for panic disorder with D-cycloserine. *Archives of General Psychiatry*, *63*, 298–304.

Hofmann, S. G., Moscovitch, D. A., Kim, H. J., & Taylor, A. N. (2004). Changes in self-perception during treatment of social phobia. *Journal of Consulting and Clinical Psychology*, *72*, 588–596.

Hofmann, S. G., Pollack, M. H., & Otto, M. W. (2006). Augmentation treatment of psychotherapy for anxiety disorders with D-cycloserine. *CNS Drug Reviews*, *12*, 208–217.

Hofmann, S. G., & Smits, J. A. J. (2008). Cognitive-behavioral therapy for adult anxiety disorders: A meta-analysis of

randomized placebo-controlled trials. *Journal of Clinical Psychiatry*, *69*, 621–632.

Hollon, S. D., & Beck, A. T. (1986). Cognitive and cognitive-behavioral therapies. In S. L. Garfield, & A. E. Bergin (Eds.), *Handbook of psychotheraphy and behavior change* (3rd ed., pp. 443–482). New York: Wiley.

Kaysen, D., Scher, C. D., Mastnak, J., & Resick, P. (2005). Cognitive mediation of childhood maltreatment and adult depression in recent crime victims. *Behavior Therapy*, *36*, 235–244.

Kenny, D. A., Korchmaros, J. D., & Bolger, N. (2003). Lower level mediation in multilevel models. *Psychological Methods*, *8*, 115–128.

Kraemer, H. C., Wilson, T., Fairburn, C. G., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry*, *59*, 877–883.

Kushner, M. G., Kim, S. W., Donahue, C., Thurus, P., Adson, D., Kotlyar, M., . . . & Foa, E. B. (2007). D-cycloserine augmented exposure therapy for obsessive-compulsive disorder. *Biological Psychiatry*, *62*, 835–858.

Ledgerwood, L., Richardson, R., & Cranney, J. (2003). D-cycloserine facilitates extinction of conditioned fear as assessed by freezing in rats. *Behavioral Neuroscience*, *117*, 341–349.

Ledgerwood, L., Richardson, R., & Cranney, J. (2004). D-cycloserine and the facilitation of extinction of conditioned fear: Consequences for reinstatement. *Behavioral Neuroscience*, *118*, 505–513.

LeDoux, J. (1996). *The emotional brain: The mysterious underpinnings of emotional life*. New York: Touchstone.

Lieberman, M. D. (2007). Social cognitive neuroscience: A review of core processes. *Annual Review of Psychology*, *58*, 259–289.

Linden, D. E. J. (2006). How psychotherapy changes the brain: The contribution of functional neuroimaging. *Molecular Psychiatry*, *11*, 528–538.

Martin, S. D., Martin, E., Rai, S. S., Richardson, M. A., & Royall, R. (2001). Brain blood flow changes in depressed patients treated with interpersonal psychotherapy or venlafaxine hydrochloride—Preliminary findings. *Archives of General Psychiatry*, *58*, 641–648.

Martinez, M. P., & Botella, C. (2005). An exploratory study of the efficacy of a cognitive-behavioral treatment for hypochondriasis using different measures of change. *Psychotherapy Research*, *15*, 392–408.

Milad, M. R., Rauch, S. L., Pitman, R. K., & Quirk, G. J. (2006). Fear extinction in rats: Implications for human brain imaging and anxiety disorders. *Biological Psychology*, *73*, 61–71.

Mineka, S., & Zinbarg, R. (2006). A contemporary learning theory perspective on the etiology of anxiety disorders. *American Psychologist*, *61*, 10–26.

Moore, E. L., & Abramowitz, J. S. (2007). The cognitive mediation of thought-control strategies. *Behaviour Research and Therapy*, *45*, 1949–1955.

Myers, K. M., & Davis, M. (2002). Behavioral and neural analysis of extinction. *Neuron*, *36*, 567–684.

Myers, K. M., Ressler, K. J., & Davis, M. (2006). Different mechanisms of fear extinction dependent on length of time since fear acquisition. *Learning and Memory*, *13*, 216–223.

Nakao, T., Nakagawa, A., Yoshiura, T., Nakatani, E., Nabeyama, M., Yoshizato, . . . & Kanba, S. (2005). Brain activation of patients with obsessive-compulsive disorder during neuropsychological and symptom provocation tasks before and after symptom improvement: A functional magnetic imaging study. *Biological Psychiatry*, *57*, 901–910.

Nakatani, E., Nakagawa, A., Ohara, Y., Goto, S., Uozumi, N., Iwakiri, M., . . . & Yamagami, T. (2003). Effects of behavior therapy on regional cerebral blood flow in obsessive-compulsive disorder. *Psychiatry Research*, *124*, 113–120.

Northoff, G., Heinzel, A., de Greck, M., Bermpohl, F., Dobrowolny, H., & Panksepp, J. (2006). Self-referential processing in our brain—Meta-analysis of imaging studies on the self. *NeuroImage*, *31*, 440–457.

Ochsner, K. N., Bungem, S. A., Gross, J. J., & Gabrieli, J. D. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience*, *14*, 1215–1229.

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

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

Öst, L. G. (2008). Efficacy of the third wave of behavioral therapies: A systematic review and meta-analysis. *Behaviour Research and Therapy*, *46*, 296–321.

Paquette, V., Levesque, J., Mensour, B., Leroux, J. -M., Beaudoin, G., Bourgouin, P., . . . & Beauregard, M. (2003). Change the mind and you change the brain: Effects of cognitive-behavioral therapy on the neutral correlates of spider phobia. *NeuroImage*, *18*, 401–409.

Pitman, R. K. (1997). Overview of biological themes in PTSD. *Annals of the New York Academy of Sciences*, *821*, 1–9.

Popper, K. R. (1959). *The logic of scientific discovery*. New York: Basic Books.

Prasko, J., Horacek, J., Zalesky, R., Kopecek, M., Novak, T., Paskova, B., . . . & Hoschl, C. (2004). The change of regional brain metabolism (^{18}FDG PET) in panic disorder during the treatment with cognitive-behavioral therapy or antidepressants. *Neuroendocrinology Letters*, *5*, 340–348.

Price, D. D. (2000). Psychological and neural mechanisms of the affective dimension of pain. *Science*, *288*, 1769–1772.

Rachman, S., & Wilson, G. T. (2008). Expansion in the provision of psychological treatment in the United Kingdom. *Behaviour Research and Therapy*, *46*, 293–295.

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy*, *35*, 7410–7456.

Rescorla, R. A., & Heth, C. D. (1975). Reinstatement of fear to an extinguished conditioned stimulus. *Journal of Experimental Psychology: Animal Behavior Processes*, *1*, 88–96.

Resick, P. A., & Schnicke, M. K. (1993). *Cognitive processing therapy for rape victims*. Newbury Park, CA: Sage.

Ressler, K. J., Rothbaum, B. O., Tannenbaum, L., Anderson, P., Graap, K., Zimand, E., . . . & Davis, M. (2004). Cognitive enhancers as adjuncts to psychotherapy: Use of D-cycloserine in phobic individuals to facilitate extinction of fear. *Archives of General Psychiatry*, *61*, 1136–1144.

Robbins, S. J. (1990). Mechanisms underlying spontaneous recovery in autoshaping. *Journal of Experimental Psychology: Animal Behavior Processes*, *16*, 235–249.

Rodriguez, B. I., Craske, M. G., Mineka, S., & Hladeck, D. (1999). Context-specificity of relapse: Effects of therapist and environmental context on return of fear. *Behaviour Research and Therapy*, *39*, 845–862.

Roemer, L., & Orsillo, S. M. (2002). Expanding our conceptualization of and treatment for generalized anxiety disorder: Integrating mindfulness/acceptance-based approaches with existing cognitive-behavioral models. *Clinical Psychology: Science and Practice*, *9*, 54–68.

Roffman, J. L., Marci, C. D., Glick, D. M., Dougherty, D. D., & Rauch, S. L. (2005). Neuroimaging and the functional neuroanatomy of psychotherapy. *Psychological Medicine*, *35*, 1385–1398.

Salkovskis, P. M., Warwick, H. M., & Deale, A. C. (2003). Cognitive-behavioral treatment for severe and persistent health anxiety (hypochondriasis). *Brief Treatment and Crisis Intervention*, *3*, 353–367.

Schulz, S. M., Alpers, G. W., & Hofmann, S. G. (2008). Negative self-focused cognitions mediate the effect of trait social anxiety on state anxiety. *Behaviour Research and Therapy*, *48*, 438–449.

Schwartz, J. M., Stoessel, P. W., Baxter, L. R., Martin, K. M., & Phelps, M. E. (1996). Systematic changes in cerebral glucose metabolic rate after successful behavior modification of obsessive-compulsive disorder. *Archives of General Psychiatry*, *53*, 109–113.

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2001). *Mindfulness-based cognitive therapy for depression: A new approach to preventing relapse*. New York: Guilford Press.

Smits, J. A. J., Rosenfield, D., Telch, M. J., & McDonald, R. (2006). Cognitive mechanisms of social anxiety reduction: An examination of specificity and temporality. *Journal of Consulting and Clinical Psychology*, *74*, 1203–1212.

Stern, R., & Fernandez, M. (1991). Group cognitive and behavioural treatment for hypochondriasis. *British Medical Journal*, *303*, 1229–1231.

Straube, T., Glauer, M., Dilger, S., Mentzel, H. J., & Miltner, W. H. R. (2006). Effects of cognitive-behavioral therapy on brain activation in specific phobia. *NeuroImage*, *29*, 125–135.

Tang, T. Z., DeRubeis, R. J., Beberman, R., & Pham, T. (2005). Cognitive changes, critical sessions, and sudden gains in cognitive-behavioral therapy for depression. *Journal of Consulting and Clinical Psychology*, *73*, 168–172.

Taylor, S., & Asmundson, G. J. G. (2004). *Treating health anxiety: A cognitive-behavioral approach*. New York: Guilford Press.

Taylor, S., Asmundson, G. J. G., & Coons, M. J. (2005). Current directions in the treatment of hypochondriasis. *Journal of Cognitive Psychotherapy*, *19*, 291–310.

Walker, D. L., Ressler, K. J., Lu, K. T., & Davis, M. (2002). Facilitation of conditioned fear extinction by systemic administration or intra-amygdala infusions of D-cycloserine as assessed with fear-potentiated startle in rats. *Journal of Neuroscience*, *22*, 2343–2351.

Warwick, H. M., Clark, D. M., Cobb, A. M., & Salkovskis, P. M. (1996). A controlled-trial of cognitive-behavioural treatment of hypochondriasis. *British Journal of Psychiatry*, *169*, 189–195.

Warwick, H. M., & Salkovskis, P. M. (1989). Cognitive and behavioural characteristics of primary hypochondriasis. *Scandinavian Journal of Behaviour Therapy*, *18*, 85–92.

Wells, A., & Papageorgiou, C. (1998). Social phobia: Effects of external attention on anxiety, negative beliefs, and perspective taking. *Behavior Therapy*, *29*, 357–370.

Wilhelm, S., Buhlmann, U., Tolin, D. F., Meunier, S. A., Pearlson, G. D., Reese, H. E., . . . & Rauch, S. L. (2008). Augmentation of behavior therapy with D-cycloserine for obsessive-compulsive disorder. *American Journal of Psychiatry*, *165*, 335–341.

Wilson, K. G. (1997). Science and treatment development: Lessons from the history of behavior therapy. *Behavior Therapy*, *28*, 547–558.

Wilson, K. G., Hayes, S. C., & Gifford (1997). Cognition in behavior therapy: Agreements and differences. *Journal of Behavior Therapy and Experimental Psychiatry*, *28*, 53–63.

Woody, S. R., Chambless, D. L., & Glass, C. R. (1997). Self-focused attention in the treatment of social phobia. *Behaviour Research and Therapy*, *35*, 117–129.

World Health Organization (1992). *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva, Switzerland: Author.

World Health Organization (1993). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research*. Geneva, Switzerland: Author.

Zettle, R. D., & Hayes, S. C. (1986). Dysfunctional control by client verbal behavior: The context of reason giving. *Analysis of Verbal Behavior*, *4*, 30–38.

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