



Maximizing exposure therapy: An inhibitory learning approach



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ABSTRACT

Exposure therapy is an effective approach for treating anxiety disorders, although a substantial number of individuals fail to benefit or experience a return of fear after treatment. Research suggests that anxious individuals show deficits in the mechanisms believed to underlie exposure therapy, such as inhibitory learning. Targeting these processes may help improve the efficacy of exposure-based procedures. Although evidence supports an inhibitory learning model of extinction, there has been little discussion of how to implement this model in clinical practice. The primary aim of this paper is to provide examples to clinicians for how to apply this model to optimize exposure therapy with anxious clients, in ways that distinguish it from a ‘fear habituation’ approach and ‘belief disconfirmation’ approach within standard cognitive-behavior therapy. Exposure optimization strategies include 1) expectancy violation, 2) deepened extinction, 3) occasional reinforced extinction, 4) removal of safety signals, 5) variability, 6) retrieval cues, 7) multiple contexts, and 8) affect labeling. Case studies illustrate methods of applying these techniques with a variety of anxiety disorders, including obsessive-compulsive disorder, posttraumatic stress disorder, social phobia, specific phobia, and panic disorder.

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Exposure therapy, or repeated approach toward fear provoking stimuli, has been a mainstay of cognitive behavioral therapy for anxiety disorders since its inception. Exposure takes various forms, including graduated versus intense (or flooding therapy), brief versus prolonged, with and without various cognitive and somatic coping strategies (as reviewed by Meuret, Wolitzky-Taylor, Twohig, & Craske, 2012), and imaginal, interoceptive or in vivo (or in real life). Exposure therapy has proven to be an effective treatment strategy for fear and anxiety disorders (Hofmann & Smits, 2008; Norton & Price, 2007). Our understanding of the mechanisms responsible for the effects of exposure therapy has evolved over the years (see Craske, Kircanski, et al., 2008; Craske, Liao, Brown, & Vervliet, 2012). The aims of the current paper are to review the inhibitory learning model of extinction as a mechanism for exposure therapy for fear and anxiety, and to detail the clinical application of this model. The translation is presented in a listing of specific behavioral strategies followed by their description in the context of case studies of panic disorder and agoraphobia, social anxiety disorder, posttraumatic stress disorder, obsessive

compulsive disorder and specific phobia. Other approaches to exposure therapy include habituation-based models, which emphasize reduction in fear throughout exposure, and behavioral testing to explicitly disconfirm threat-laden beliefs and assumptions (e.g., Foa & Kozak, 1986; Foa & McNally, 1996; Salkovskis, Hackmann, Wells, Gelder, & Clark, 2006). We have compared the inhibitory learning model to fear habituation and ‘belief disconfirmation using behavioral testing’ models in prior papers (i.e., Craske, Kircanski, et al., 2008; Craske, Waters, et al., 2008; Craske et al., 2012). In the discussion that follows, we present specific applications for ways in which the inhibitory learning model differs from these other models.

Inhibitory learning model of extinction

In a Pavlovian conditioning model, a neutral stimulus (the conditional stimulus, CS, such as a neutral picture) is followed by an aversive stimulus (the unconditional stimulus, US, such as an electric shock). After a number of such pairings, the neutral CS will come to elicit anticipatory fear reactions (or a conditional response, CR). The CR is presumed to depend upon the CS becoming a reliable predictor of the US. An association is posited between the memory representations of the CS and the US such that presentations of the

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CS will indirectly activate the memory of the US. Hence, by ‘thinking’ about the aversive US, fear is elicited. Fear conditioning is considered a valid model for many of the anxiety disorders, including panic disorder, social anxiety disorder, specific phobia, obsessive compulsive disorder, and posttraumatic stress disorder (Grillon, 2008). One powerful way to reduce conditional fear reactions is through extinction, in which the CS is repeatedly presented in the absence of the associated aversive event (the US). Exposure therapy, wherein an individual is repeatedly exposed to fear provoking stimuli in the absence of repeated aversive outcomes, is the clinical proxy of extinction and indeed exposure therapy, first proposed by Wolpe (1958) in the form of systematic desensitization, was derived from early models of extinction learning.

Inhibitory learning is regarded as being central to extinction (Bouton, 1993; Miller et al., 1988; Wagner, 1981), although additional mechanisms, such as habituation, are likely to be involved (Myers & Davis, 2007). Within a Pavlovian conditioning approach, the inhibitory learning models mean that the original CS-US association learned during fear conditioning is not erased during extinction, but rather is left intact as new, secondary inhibitory learning about the CS-US develops – specifically, that the CS no longer predicts the US (e.g., Bouton, 1993; Bouton & King, 1983). Research into the neural mechanisms underlying fear extinction support an inhibitory model, since the amygdala, which is particularly active during fear conditioning (Shin & Liberzon, 2010), appears to be inhibited by cortical influences identified as occurring from the medial prefrontal cortex as a result of extinction learning (Milad et al., 2007, 2009).

Bouton and colleagues propose that after extinction, the CS possesses two meanings; its original excitatory meaning (CS-US) as well as an additional inhibitory meaning (CS-no US). Therefore, even though fear subsides with enough trials of the CS in the absence of the US, retention of at least part of the original association can be uncovered by various procedures, with each one showing a continuing effect of the original excitatory association after extinction. First, conditional fear shows spontaneous recovery (Quirk, 2002), meaning that the strength of the CR increases in proportion to the amount of time since the end of extinction. Clinically, this effect parallels the return of fear that commonly occurs with the lapse of time since completion of exposure therapy (e.g., Craske & Mystkowski, 2006; Craske & Rachman, 1986). Thus, an individual whose fear of air travel significantly reduces by the end of exposure treatment is vulnerable to a return in fear of flying in the absence of repeated air travel following treatment completion.

Second, renewal of conditional fear occurs if the surrounding context is changed between extinction and retest (Bouton, 1993). In other words, fear extinction appears to be specific to the context in which extinction occurs. These effects have been observed in clinical analog samples undergoing exposure therapy and follow-up testing in the same versus different contexts (Culver, Stoyanova, & Craske, 2011; Mystkowski, Craske, & Echeverri, 2002; Mystkowski, Mineka, Vernon, & Zinbarg, 2003; Mystkowski et al., 2006). The clinical relevance of renewal arises when exposure therapy is completed in one or only a limited number of contexts (such as in the presence of a therapist or always immediately preceding or following a therapy session), such that fear is likely to return when the phobic stimulus is subsequently encountered in a different context (such as when alone or when unrelated to a therapy session).

Third, reinstatement of conditional fear occurs if unpaired (or unpaired) US presentations occur in between extinction and retest (Hermans et al., 2005; Rescorla & Heth, 1975; Van Damme, Crombez, Hermans, Koster, & Eccleston, 2006). The clinical

implication of reinstatement is that adverse events following exposure therapy may lead to a return of fear of the previously feared stimulus if it is encountered in an anxiety inducing context. For example, fear of asking questions in work meetings may resurge after being rejected in another social situation, or possibly after an unrelated adverse event such as a motor vehicle accident. Fourth, rapid reacquisition of the CR is seen if the CS-US pairings are repeated following extinction (Ricker & Bouton, 1996). The clinical application is that fears that have subsided may be easily and rapidly reacquired with re-traumatization, as may occur in combat situations or other dangerous environments.

Deficits in inhibition and anxiety disorders

A substantial number of individuals fail to achieve clinically significant symptom relief from exposure-based therapies (Arch & Craske, 2009) or experience a return of fear following exposure therapy (see Craske & Mystkowski, 2006). This may derive in part from the deficits in extinction learning (Craske, Waters, et al., 2008; Lissek et al., 2005) and more specifically, deficits in inhibitory learning and inhibitory neural regulation during extinction, that characterize individuals with anxiety disorders or elevated trait anxiety (e.g., Indovina, Robbins, Nunez-Elizalde, Dunn, & Bishop, 2011; Jovanovic et al., 2010; Milad et al., 2009, 2013; Rougemont-Bucking et al., 2011; see Craske et al., 2012 for a summary). In other words, anxious individuals show deficits in the mechanisms that are believed to be central to extinction learning – such deficits may not only contribute to poor response to exposure therapy but may also contribute to the development of excessive fear and anxiety in the first place.

As such, there is tremendous clinical value to optimizing inhibitory learning during exposure therapy in order to both enhance treatment efficacy in general and to compensate for the deficits that are present within the anxious individual. An exposure model that takes elements of inhibitory learning into account has the potential to offset the negative effects of spontaneous recovery, renewal, reinstatement and reacquisition. The goal is to enhance inhibitory learning (and possibly underlying neural inhibitory regulation) during exposure therapy and to enhance its retrieval following completion of exposure therapy.

Inhibitory learning versus habituation and behavioral testing approaches to exposure

Notably, the strategies listed below are not always consistent with an habituation-based model of exposure therapy, which rests upon fear reduction during exposure trials as a critical index of therapeutic change (e.g., Foa & Kozak, 1986; Foa & McNally, 1996; Lader & Matthews, 1968). Habituation models posit that fear reduction during an exposure trial is a necessary precursor to subsequent, longer lasting cognitive changes in the perceived harm associated with the phobic stimulus. The strategies that derive from inhibitory learning models do not emphasize fear reduction per se during exposure trials and instead sometimes use strategies designed to maintain elevated fear throughout exposure trials. In support, the amount by which fear has reduced at completion of extinction is not predictive of the amount of fear expressed at the follow-up extinction retest in either animals or human laboratory samples (Plendl & Wotjak, 2010; Prenoveau, Craske, Liao, & Ornitz, 2013; Rescorla, 2006). Similarly, the amount by which fear reduces by the end of an exposure trial or series of exposure trials is not predictive of the fear level expressed at follow-up assessment in fearful human samples (Baker et al., 2010; Culver, Stoyanova, & Craske, 2012; Kircanski et al., 2012). This is consistent with the notion of divergence in response systems, and that outward

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