



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, & Echiverri, 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 unsigned (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-Buckling 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

expression of fear on the one hand, and conditional associations indicative of underlying learning on the other hand, may not always change in concordance (Beckers, Kryptos, Boddez, Effting, & Kindt, 2013). Fear expressed at follow-up (as the critical index of the strength and consolidation of extinction learning) appears to be more likely to be influenced by factors such as passage of time, context shifts, adverse events or relearning than by the level of fear experienced at the end of extinction/exposure. Some aspects of the inhibitory learning model overlap with cognitive models that emphasize behavioral testing to disconfirm beliefs and assumptions (Salkovskis et al., 2006). However, the inhibitory learning model is not restricted to behavioral testing as a strategy for generating inhibitory associations, nor is it limited to testing of explicitly stated cognitions.

Therapeutic strategies for enhancing inhibitory learning and its retrieval

(1) Expectancy violation. The first strategy is to design exposures that maximally violate expectancies regarding the frequency or intensity of aversive outcomes (Davey, 1992; Gallistel & Gibbon, 2000; Rescorla & Wagner, 1972). This strategy derives from the premise that the mismatch between expectancy and outcome is critical for new learning (Rescorla & Wagner, 1972) and for the development of inhibitory expectancies that will compete with excitatory expectancies. The more the expectancy can be violated by experience, the greater the inhibitory learning. We found that this approach yielded as much long-term benefit at follow-up with just one trial of exposure per two days compared to repeated trials of exposure each day for acrophobia (Baker et al., 2010). Deacon et al. (2013) found that interoceptive exposure that continued until the patient's expectancy of an aversive outcome reached less than 5% was superior to standard interoceptive exposure. In this approach, exposure tasks are designed to accommodate "what do you need to learn" rather than by fear reduction or "stay in the situation until fear declines" as would be predicated from an habituation-based model of exposure therapy. For example, for persons who irrationally expect to become erratic and hurt themselves due to prolonged anxiety, anxiety is induced for prolonged durations in order to fully violate expectancies regarding their behavior. Clinically, it is important that the client identify the US when predicting the expectancy to be violated. For example, for clients with social anxiety, predicting that they will "get anxious" during a social interaction would not be sufficient, whereas predicting that they would be ignored or otherwise rejected would be sufficient. The expectancy violation approach ties exposure parameters directly to consciously stated expectancies for aversive events. As such, it overlaps with models in which exposure is used for belief disconfirmation, and which was shown to be superior to habituation approaches in one small study (see Salkovskis et al., 2006). As further evidence against habituation approaches, neither fear reduction nor ending fear levels predict long term outcome from extinction or exposure (Baker et al., 2010; Culver et al., 2012; Kircanski et al., 2012; Plendl & Wotjak, 2010; Prenoveau et al., 2013; Rescorla, 2006 – discussed in prior sections). Moreover, exposure strategies that specifically impede habituation were found to be more effective than strategies that do not (Culver et al., 2012; Kircanski et al., 2012; Lang & Craske, 2000 – reviewed in subsequent sections). In the expectancy violation approach, the end of an exposure trial is determined by conditions that violate expectancies and not by fear reduction; for example, exposure is continued for the duration determined to most effectively violate expectancies rather than whether fear has declined. Learning is centered around whether the expected negative outcome occurred or not, or was as 'bad' as expected (i.e., was

'manageable or not'). Thus, following each exposure trial, the learning is consolidated by asking participants to judge what they learned regarding the non-occurrence of the feared event, discrepancies between what was predicted and what occurred, and the degree of "surprise" from the exposure practice (e.g., Rescorla & Wagner, 1972). Indeed, mental rehearsal, or in this case mental rehearsal of the inhibitory CS-no US association, is an important component of memory consolidation (Joos, 2011; Meeter & Murre, 2004).

A key aspect of an expectancy violation model is to facilitate attention to both the CS and the non-occurrence of the US. Error-correction models (e.g., Rescorla & Wagner, 1972) posit an important role for the salience of the CS such that any change in associative strength (e.g., extinction learning) will be directed to the cue that is most salient (Mackintosh, 1975; Pearce & Hall, 1980). Inasmuch as extinction learning represents the formation of a non-contingent relationship between CS and US, awareness of both the CS and the non-occurrence of the US are essential. This may be one reason why distraction is such a pernicious safety behavior, as it can reduce awareness of the CS, or the CS-no US relationship.¹ It may also explain the limitations of habituation based models, since habituation is enhanced by a procedure which is likely to reduce the salience of the stimulus (i.e., repeated exposure to the same stimulus). We return to the importance of salience below, when referring to occasionally reinforced extinction as a strategy for enhancing inhibitory learning.

Within the expectancy violation model, graduated exposure may be employed to conditions under which the feared outcome is judged most likely to occur (i.e., the conditions that provide optimal violation of expectancy). However, in contrast to an habituation-based model, the graduated approach is tied to 'violating' conditions per se (e.g., duration of exposure) and not necessarily tied to fear level (i.e., repeat each item on a fear hierarchy enough times for fear to decline before proceeding to the next hierarchy item). For example, for persons who fear having a heart attack from a panic attack in an elevator, exposure may be conducted to progressively lengthier trials in the elevator even though fear does not decline with each exposure trial. Notably, sustained arousal throughout extinction is associated with less fear at retest in animals (Rescorla, 2006) and in humans (Culver, Stephens, & Craske, in preparation-a), arousal consolidates extinction memories (Cain, Blouin, & Barad, 2004) and in several of our studies, failure to habituate throughout exposure therapy was not associated with poorer outcomes (e.g., Culver et al., 2012; Kircanski et al., 2012; Lang & Craske, 2000).

The basic premise of the violation of expectancy approach, which is that extinction learning is enhanced by the mismatch between expectancy and experience, implies that strategies that reduce expectancy prior to extinction can negatively impact extinction learning. To this end, traditional cognitive interventions designed to lessen probability overestimation (e.g., "I am unlikely to be bitten by the dog") and perceived negative valence (e.g., "It is not so bad to be rejected") may be deleterious to inhibitory learning when employed prior to, or during, exposures. That is, cognitive interventions may reduce the expectancy of a negative outcome before exposure and thereby lessen the mismatch between initial expectancy and actual outcome. Thus, we now confine our

¹ Notably, a recent meta-analysis of distraction during exposure for specific phobias indicated that uninstructed exposure outperformed distracted exposure on behavioral outcomes, but, under specific conditions of interactive distraction and repeated exposure trials, distracted exposure outperformed focused exposure on behavioral and distress outcomes (Podina, Koster, Philippot, Dethier, & David, 2013).

“cognitive” interventions to post-exposure questioning in order to facilitate memory consolidation.

Habituation approaches to exposure posit that exposure to a given item continues for long enough for fear to decline and for the number of occasions necessary for fear to be significantly lessened. In an inhibitory learning model, exposure continues for the length of time predetermined as an adequate test of a stated expectancy, and continues for the number of occasions necessary for expectancies to be lessened.

(2) Deepened extinction. A second strategy is “deepened extinction” (Rescorla, 2000, 2006), in which either multiple fear CSs are first extinguished separately before being combined during extinction, or a previously extinguished cue is paired with a novel CS. This has been shown to reduce spontaneous recovery and reinstatement of fear in animals (Rescorla, 2006) and humans (Culver, Vervliet, & Craske, in preparation-b). Wherever possible, we combine multiple cues (internal and/or external) during exposure therapy, after initially conducting some exposure to each cue in isolation. However, it is important that both stimuli predict the same US. Interceptive exposure to feared bodily sensations (such as caffeine consumption), and in vivo exposure to feared external agoraphobic situations (such as shopping in a crowded mall) followed by inclusion of interoceptive exposure during in vivo exposure (drinking coffee whilst in the shopping mall) is an example of deepened extinction for panic disorder and agoraphobia (Barlow & Craske, 1994). Another example would be exposure to one specific type of spider, then a second distinctly different spider, followed by exposure to both spiders at the same time. A third example would be exposure to an obsession (such as an obsession of stabbing a loved one), exposure to a cue that triggers obsessions (such as a knife in the presence of a loved one), followed by exposure to both the obsession and the knife in the presence of the loved one. Although deepened extinction is presumed to exert its effects through augmented violation of expectancies, the procedure could be implemented without specifically asking clients to identify their expectancies beforehand. Thus, deepened extinction represents one way in which an inhibitory learning approach extends beyond behavioral testing for the purpose of belief disconfirmation.

(3) Occasional reinforced extinction. A third strategy just gaining evidence in human studies is occasional reinforced learning during extinction. Occasional reinforced extinction involves occasional CS-US pairings during extinction training (Bouton, Woods, & Pineno, 2004). The benefits may derive from an expectancy violation effect in which the participant is less likely to expect the next CS presentation to predict the US because CS-US pairings have been associated with both further CS-US pairings and CS-no US pairings (Bouton et al., 2004). Alternatively, the procedure of occasional reinforcement during extinction may enhance salience of the CS which in turn contributes to new learning about the CS (Pearce & Hall, 1980). As with animal studies (Bouton et al., 2004), we found that occasional reinforced extinction sustained fear arousal during extinction but attenuated the subsequent reacquisition of fear in a human conditioning study (Culver et al., under review). The phenomenon of rapid reacquisition is most likely in the presence of repeated aversive outcomes (e.g., social rejection in the case of social anxiety disorder and panic attacks in the case of panic disorder). It may also be likely to occur in the context of dangerous environments that lead to retraumatization, although the approach of occasionally reinforced extinction is ethically prohibitive in such cases. In the case of social anxiety, an individual may successfully extinguish fear responding in social situations only to have that fear response return quickly after just one subsequent pairing of a social situation with a negative outcome (e.g., rejection or negative evaluation). Although further evidence is warranted, the clinical

translation of occasional reinforced extinction is the addition of occasional social rejections and “shame attacks” in exposures to social situations, or the deliberate induction of panic attacks (e.g., such as by substances like yohimbine) in exposures to feared situations for panic disorder. We routinely conduct such reinforced exposure and even encourage clients to seek the opportunity for occasional negative outcomes for the reasons stated. Although occasional reinforced extinction is presumed to exert its effects at least partly through augmented violation of expectancies, the procedure could be implemented without specifically asking clients to identify their expectancies beforehand. Thus, occasional reinforced extinction represents another way in which an inhibitory learning approach extends beyond behavioral testing for the purpose of belief disconfirmation.

(4) Removal of safety signals. A fourth strategy is the prevention or removal of “safety signals” or “safety behaviors.” Common safety signals and behaviors for clients with anxiety are the presence of another person, therapists, cell phones, medications, or food or drink. For persons who expect aversive outcomes contingent upon fear itself (i.e., “fear of fear”, such as individuals with panic disorder who fear losing control should they panic, or individuals with social anxiety who fear humiliation should they exhibit anxiety), reduction of fear itself could become a safety signal. In the experimental literature, safety signals alleviate distress in the short term, but when they are no longer present, the fear returns (Lovibond, Davis, & O’Flaherty, 2000). This effect is believed to derive in part from interference with the development of inhibitory associations. In phobic samples, the availability and use of safety signals and behaviors has been shown to be detrimental to exposure therapy (Sloan & Telch, 2002), whereas instructions to refrain from using safety behaviors improved outcomes (Salkovskis, 1991). However, recent data have presented contradictory findings (Rachman, Shafran, Radoomsky, & Zysk, 2011). Specifically, the use of hygienic wipes following exposures for individuals with contamination fears did not lead to any more spontaneous recovery of fear or disgust than exposure without hygienic wipes. Similarly, Deacon and colleagues have failed to replicate the deleterious effect of continuing to engage in safety behaviors (including the availability of the safety behavior but without actual engagement of it) during exposure in claustrophobic fear (Deacon, Sy, Lickel, & Nelson, 2010; Sy, Dixon, Lickel, Nelson, & Deacon, 2011). However, the ability of safety behaviors to mitigate extinction learning likely varies depending on the ratio of inhibition and excitation in a given trial. That is, the presence of inhibitory stimuli (i.e., stimuli that decrease the likelihood that the US will be delivered) will mitigate extinction learning inasmuch as they decrease the expectation of the US, and the discrepancy between what is predicted and what actually occurs determines the degree of associative change. The impact of inhibitory stimuli on extinction learning will therefore depend on the number and strength of inhibitory stimuli versus the number and strength of excitatory stimuli (i.e., stimuli that predict the US; Rescorla & Wagner, 1972). The general consensus remains that safety signals or behaviors should gradually be phased out over the course of exposure therapy (Hermans, Craske, Mineka, & Lovibond, 2006). Gradual phasing is recommended only in order to reduce treatment attrition. If willing, immediate removal of safety signals is preferred.

(5) Variability. A fifth strategy involves stimulus variability throughout exposure since varying the to-be-learned task enhances retention of learned non-emotional material (Magill & Hall, 1990; Schmidt & Bjork, 1992; Shea & Morgan, 1979). Variability is believed to enhance the storage capacity of newly learned information (Bjork & Bjork, 1992, 2006), pair the information to-be-learned with more retrieval cues (Estes, 1955), or generate a rule that captures the invariance among tasks (Schmidt & Bjork, 1992),

which renders the information more retrievable at a later point in time. Although this strategy did not originate from associative conditioning models, the effects can be explained by context retrieval models of extinction as well (Bouton, 1993), since variability is more likely to characterize contexts in which phobic stimuli are encountered once exposure therapy is complete. Hence, variability during exposure may offset context renewal effects after exposure. We found that variability in terms of timing between exposure sessions (i.e., progressively longer intervals between exposure sessions) led to superior outcomes at follow-up than nonvariable-massed exposure in spider fearful samples (Rowe & Craske, 1998; Tsao & Craske, 2000). Also, variability in terms of the stimuli used during exposure led to positive outcomes in terms of spontaneous recovery in spider fearful and height fearful samples (Lang & Craske, 2000; Rowe & Craske, 1998), although a third study of contaminant anxiety showed trends only (Kircanski et al., 2012). Traditional exposure proceeds steadily from one hierarchy item to the next, with each item repeated a number of times until anxiety decreases. Instead, in variable exposure, exposure is conducted to items from the hierarchy in random order, without regard to fear levels or fear reduction, although usually beginning with the least anxiety producing item to avoid treatment refusal. We routinely conduct exposure with varying stimuli, for varying durations, at varying levels of intensity, or select items from a fear hierarchy out of order, rather than continuing exposure in one situation until fear declines before moving to the next situation. Notably, such variability typically elicits higher levels of physiological arousal and subjective anxiety during exposure that fail to habituate (e.g., Kircanski et al., 2012; Lang & Craske, 2000), and yet produce beneficial effects in the long term.

Furthermore, greater variability in fear levels throughout exposure (i.e., repeated increases following decreases in minute to minute fear levels) is a positive predictor of outcomes in contaminant anxiety and public speaking anxiety (Culver et al., 2012; Kircanski et al., 2012). Conceivably, emotional state (i.e., fear level) serves as a retrieval cue and varying levels of fear are likely to occur in situations following exposure therapy where retrieval is required (Bjork & Bjork, 1992, 2006). Thus, variation in fear level throughout exposure will offset context renewal once exposure therapy is completed. Variability in emotional state may also enhance salience of the phobic stimulus and thereby enhance learning of inhibitory associations. We routinely encourage variability in fear response during exposures, such as by conducting “unpredictable” lengths of exposures to phobic stimuli (with clients’ agreement to the general principles beforehand).

(6) Retrieval cues. One option for enhancing retrieval of extinction learning and offsetting context renewal is to include retrieval cues (of the CS-no US association) during extinction training to be used in other contexts once extinction is over (Brooks & Bouton, 1994; Dibbets & Maes, 2011; Vansteenwegen et al., 2006). One risk of retrieval cues, however, is that they may acquire an inhibitory value and become a safety signal (Dibbets, Havermans, & Arntz, 2008). Retrieval cues differ from safety signals in that they retrieve the CS-no US relationship (i.e., act as an occasion setter), whereas safety signals are directly associated with the non-occurrence of the US. For example, a therapist’s office where previous exposure sessions were conducted can act as a retrieval cue for a new exposure, whereas benzodiazepines (e.g., in the case of panic disorder) could act as a safety signal. In clinical analog anxious samples, the effects of a retrieval cue (distinctive pen and clip board) upon context renewal were very weak in one study (Culver et al., 2011), although instructions to mentally reinstate what was learned during exposure (an instructional retrieval cue) had more robust effects in reducing context renewal in another study (Mystkowski, Craske, Echiverri, & Labus, 2006). In the

treatment of anxiety disorders, this approach prescribes that individuals carry cues (e.g., wrist band) with them to remind them of what they learned during exposure therapy (as long as the cues do not become safety signals), or are prompted to remind themselves of what they learned in exposure therapy each time they encounter previously feared sensations or situations. However, these strategies are best employed as a relapse prevention skill. Using retrieval cues early in therapy, while the focus is on acquisition of extinction learning, may negatively impact progress as these cues can reduce the expectancy of the aversive event (and therefore mitigate expectancy violation effects). In addition, any retrieval cues should be used sparingly to mitigate their likelihood of becoming a conditioned inhibitor or safety signal.

(7) Multiple contexts. Context renewal involves the return of fear to a phobic stimulus when it is encountered in a context (internal or external) that differs from the context in which exposure therapy was conducted (Mineka, Mystkowski, Hladek, & Rodriguez, 1999; Mystkowski et al., 2002; Rodriguez, Craske, Mineka, & Hladek, 1999). Multiple contexts have been shown to offset context renewal in rodent samples (e.g., Gunther, Denniston, & Miller, 1998), in human laboratory studies (e.g., Bandarian Balooch & Neumann, 2011; Bandarian Balooch, Neumann, & Boschen, 2012) and in a clinical analog study of exposure therapy (Vansteenwegen et al., 2007). On the other hand, one conditioning study in rodents (Bouton, García-Gutiérrez, Zilski, & Moody, 2006) and another conditioning study in humans (Neumann, Lipp, & Cory, 2007) failed to demonstrate detectable benefits of multiple contexts throughout extinction on context renewal, suggesting that the effects are unstable. The clinical translation involves conducting interoceptive, imaginal, and in vivo exposures in multiple different contexts, such as when alone, in unfamiliar places, or at varying times of day or varying days of the week.

(8) Reconsolidation. A recent (re-)discovery is that retrieving already stored memories induces a process of *reconsolidation* (Nader, Schafe, & Le Doux, 2000), since the memory is written into long term memory again, requiring de novo neurochemical processes. Thus, it may be possible to change memories during the reconsolidation time frame upon retrieval. Monfils, Cowansage, Klann, and LeDoux (2009) used a behavioral strategy for this purpose, hypothesizing that novel information presented during the reconsolidation window may be incorporated into the memory and change it. Thus, extinction during a reconsolidation window may weaken the fear memory itself. Monfils et al. found that a brief presentation of the CS 30 min prior to sustained extinction trials significantly reduced spontaneous recovery, renewal, reinstatement and rapid reacquisition in a rodent sample. This effect has since been demonstrated in healthy human samples (Agren et al., 2012; Schiller et al., 2010). The clinical implication is to introduce the phobic stimulus for a brief period 30 min before repeated trials of exposure. However, as with many of the other strategies listed above, there is a need for further evidence. For example, the findings regarding pre-exposure reconsolidation has not been replicated in all cases (Chan, Leung, Westbrook, & McNally, 2010). Furthermore, the same results occur whether the brief exposure to the CS occurred in the window before extinction or in a window after completion of extinction (Baker, McNally, & Richardson, 2013; Ponnusamy et al., 2011), which suggests that the results may relate more to enhancing the retrieval of the extinction learning rather than erasing acquisition learning. Also, in clinical practice, most clients will retrieve their fear memories to a certain degree whenever they enter treatment sessions. The question is what type, degree, or frequency of retrieval opens the reconsolidation window and provides the opportunity to update the underlying memories (Vervliet, Craske, & Hermans, 2013).

Therapeutic strategy for enhancing inhibitory regulation

Social neuroscience has identified another strategy for enhancing inhibitory regulation that involves linguistic processing, or affect labeling. Affect labeling may work to augment associative inhibitory processes within extinction or may work in an independent but complementary manner to extinction learning. A number of studies have shown that linguistic processing activates a region of the cortex, the right ventrolateral prefrontal cortex, that reduces activity in the amygdala, thereby attenuating anxious responding (Lieberman et al., 2007). It appears that engaging the executive functioning cortical areas of the brain works to dampen the limbic system activity. In two studies, we have shown benefits of affect labeling as individuals are exposed to feared stimuli. Tabibnia, Lieberman, and Craske (2008) found that repeated evocative spider images paired with word labels, negatively valenced and irrelevant to the images (e.g., “bomb” and “war”), produced a greater reduction in subsequent skin conductance response (SCR) to the images, one week later, than unpaired images. Furthermore, Kircanski et al. (2012) found added benefits of affect labeling in a sample of individuals with spider phobias as they underwent exposure therapy. In comparison to cognitive reappraisal of thoughts, distraction, and exposure alone, affect labeling during exposure was found to reduce skin conductance and increase approach behavior at one week follow up in a context different than the exposure context (Kircanski et al., 2012). These data suggest that linguistic processing in the form of labeling, as opposed to more traditional cognitive therapy which attempts to change the content of appraisals, can improve outcomes from exposure. We routinely ask clients to state their emotional responses, without attempting to change their emotional responses, in the midst of exposure.

Case studies

In the following section, we present case examples of implementing an inhibitory learning based model of exposure therapy for a variety of anxiety disorders. This is not intended to be exhaustive but rather exemplary.

Obsessive compulsive disorder

Roberto is a 43-year-old father of two who sought therapy for intrusive thoughts related to physically harming his newborn son. While he believed that he would never actually harm his children, he was extremely distressed by these images. Specifically, he imagined suffocating his son while he slept. He often engaged in reassurance seeking from his wife, asking her to describe what a good father he was. In addition, when confronted with these intrusive images or thoughts, he attempted to bring to mind an image of a previous positive encounter with his children. As a result of these thoughts and images, Roberto significantly decreased the time he spent with his children, particularly when alone, and this caused much concern in his family. He had stopped tucking his children in at night, and refused to allow either child to sleep in his bed with him and his wife.

Session 1 entailed detailed discussion regarding the nature of associative learning, and how avoidance can interfere with new learning by preventing any violation in expectancy. Discussions de-emphasized the importance of immediate fear reduction and instead focused on strategies that while in the short term may elicit more distress, would lead to eventual fear reduction. That is, the therapist emphasized that the therapy would initially activate expectancies for negative outcomes – in order to get the optimal learning experience from exposure therapy – and that this may

cause more distress at first. The therapist further emphasized that fear would eventually reduce as a result of treatment, but that the mechanism underlying eventual fear reduction would be the continued expectancy violation. Roberto found the phrase “test it out” particularly helpful for remembering the rationale behind an inhibitory model of exposure.

Sessions 2–5 focused on in vivo exposure. Initial exposure exercises were based on spending time alone with his children, and specifically his infant son. These were chosen as they were deemed only “moderately difficult”. We find that beginning with moderately difficult exposures increases the likelihood of success and facilitates patient buy-in. However, we do not necessarily proceed up a hierarchy in a linear fashion consistent with the concept of variability discussed previously. For example, more difficult exposures, such as placing his hand on his son’s neck as he slept for a specified period of time, were completed early in therapy. Additional exposures included tucking his children in at night, reading news stories about parents harming their children and then playing with his son, and laying down with his children as they napped in his bed. Roberto worked to complete all of these exposures alone, as the presence of his wife acted as a safety signal that reduced his expectation that he would hurt his children. Further safety signals were gradually removed as well. Exposures extended to deliberately bringing to mind the intrusive violent images of suffocating his son prior to engaging in several of these tasks (see below). Roberto feared that if he did bring these images to mind, he might be more likely to actually perform a violent act. Thus, it was important to include these images in exposure sessions in order to maximize any violation of expectancy. Roberto’s therapist worked with him to develop a detailed imaginal script (including a variety of sensory elements) for use during exposure.

While at first glance these appear similar to exposures that may be conducted from the standpoint of habituation-based or cognitive models, several differences are important to note. First, prior to each exposure, Roberto learned to describe his feared outcome in order to facilitate expectancy violation. For example, Roberto reported that he was 80% certain he would attempt to suffocate his son if he placed his hand on his son’s neck for 10 min as he slept. The 10-min duration of the exposure was chosen as Roberto reported that shorter exposures did not increase his expectation of harming his son. Second, following each in-session exposure exercise, Roberto and his therapist engaged in lengthy discussion regarding the non-occurrence of his feared event. This represented an attempt to consolidate extinction learning. Open-ended questions such as “What did you fear would happen as a result of the exposure?” “What happened?” “How was that surprising?” and “What did you learn?” were used as part of an interactive discussion. Roberto was given monitoring sheets for between-session practices where he could list the anticipated negative outcome prior to exposure (e.g., suffocating his son), and engage in post-exposure consolidation. The latter involved Roberto listing whether his feared outcome occurred or not, citing evidence to support his awareness of the non-occurrence of the US (e.g., “How do you know your feared outcome did not occur?”), and describing what he learned from engaging in the exposure.

Third, Roberto was instructed to continue with a given exposure until his expectancy had been violated, or he had reached the agreed upon behavioral goal, regardless of his level of distress. Although fear often decreased across exposure trials, Roberto’s therapist noted that fear need not necessarily decline each time, as each instance of heightened expectation provided additional opportunities to enhance learning. Exposures to a given CS (e.g., touching his son’s neck as he slept) were repeated multiple times over the course of treatment; however, occasionally additional elements were added (increasing duration of the exposure, adding

additional cues—see below) to increase his expectation of a negative outcome.

Finally, cognitive restructuring was not employed prior to, or during, exposures, as this could reduce the expectancy of an aversive outcome and mitigate extinction learning. Rather, Roberto's therapist emphasized the importance of strategies that increase expectancy in order to maximize learning, and noted that certain strategies (e.g., safety behaviors, correcting probability overestimation) could negatively impact extinction learning. "Cognitive" strategies were confined to post-exposure discussions in order to facilitate consolidation of new learning.

Sessions 6–11 continued with in vivo exposure while incorporating several extinction enhancement strategies. In order to maximize extinction learning for a given CS, several conditional stimuli were included simultaneously in order to "overpredict" the occurrence of the US. This deepened extinction was accomplished in several ways. First, after conducting several exposures to cues in isolation, two cues were combined in compound. For example, Roberto initially conducted exposures to a) placing his hand on his son's neck as he slept and b) bringing to mind intrusive images, separately. These were then combined in a single exposure session. Second, cues that were extinguished in isolation were occasionally presented during a new exposure trial. This increases the expectancy for the novel CS while simultaneously maintaining its salience. For example, prior to bathing his infant son for the first time, Roberto combined this exposure with reading news stories about parents harming their children (which he had done previously).

Prior to termination, Roberto's therapist discussed the context dependent nature of extinction learning, and suggested several relapse prevention strategies. Specifically, Roberto worked to "mentally reinstate" previous extinction contexts by imagining, in detail, an exposure session that went well (i.e., his expectation was violated). He practiced this during several exposure trials during his last week of therapy, but was cautioned not to do this too often, nor to rely on it as a safety signal.

Examples of several exposure trials are shown in Table 1.

Post-traumatic stress disorder

Julia survived a sexual assault approximately one year ago, but is still troubled by intrusive images of the event and extensive situational avoidance. For example, she frequently avoids being alone, preferring to have one or more friends with her whenever in public. She reported avoiding any type of social interaction where others might be drinking, as she fears that alcohol may make a potential assailant more likely to act violently. In addition, she always carries pepper spray with her when she leaves the house. Julia reported that she is interested in pursuing a romantic relationship, but becomes highly fearful regarding interpersonal contact with a potential partner.

Session 1 entailed detailed discussion regarding the nature of associative learning, and how avoidance and safety behaviors can interfere with exposure by preventing violation in expectancy. In addition, Julia and her therapist developed a list of avoided situations along with the feared outcome associated with these situations. Although the hierarchy contained distress and expectancy ratings, exposures did not proceed linearly from the least distressing to the most distressing item, consistent with the concept of variability discussed previously.

Sessions 2–4 focused on in vivo exposures centered on expectancy violation while decreasing Julia's use of safety behaviors. Typical exposures included attending social gatherings alone, particularly in situations where individuals may be drinking socially, leaving her house without her pepper spray, and beginning

Table 1
Example OCD exposure exercises.

Session 3	
<u>BEFORE exposure:</u>	
Goal:	Place hand on son's neck for 10 min as he sleeps (4× over the course of the week) I will strangle him
What are you most worried will happen?	
On scale 0–100, how likely does this seem?	80%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	My hand never tightened around his neck
What did you learn?	I can be alone with my son as he sleeps and not hurt him
Session 4	
<u>BEFORE exposure:</u>	
Goal:	Read news stories of parents harming their children for 15 min and then play with kids for 10 min (3× over the course of the week) I will hurt my kids
What are you most worried will happen?	
On scale 0–100, how likely does this seem?	70%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	I never hurt my children, even when I was alone with them
What did you learn?	Reading stories about others hurting their kids doesn't mean I will
Session 6	
<u>BEFORE exposure:</u>	
Goal:	Imagine strangling my son for 5 min and then place hand on son's neck for 10 min as he sleeps (3× over the course of the week) I will strangle him
What are you most worried will happen?	
On scale 0–100, how likely does this seem?	90%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	My hand never tightened around his neck
What did you learn?	I was really anxious, but I didn't hurt my son. Just because I have thoughts about hurting him doesn't mean I will

to go on dates. Consistent with an inhibitory model, prior to engaging in exposures Julia was asked to state her feared negative outcome, and to track the non-occurrence of the US following each exposure.

Sessions 5–12 continued with in vivo exposure while also incorporating imaginal exposure to her trauma. Julia noted several concerns regarding engaging in imaginal exposure such as being unable to tolerate the distress associated with the exposure, and being too distressed to accomplish further tasks throughout the day. Julia's therapist worked with her to clarify and operationalize these expectations in order to "test them out". For example, her perceived inability to tolerate distress was related to a concern that the stress of the exposure would cause a "mental breakdown" and make her "go crazy". In order to target her concerns regarding being unable to accomplish tasks, Julia was asked to engage in minor tasks (e.g., cooking dinner, completing a work project) immediately following imaginal exposures. Julia's therapist also encouraged her to label her emotional experience prior to, and during, imaginal exposures to enhance inhibitory learning (i.e., affect labeling).

Julia reported a great deal of shame around her trauma, and reported fearing that others would judge her for actions she took, or didn't take, surrounding the assault. Initial exposures with her

therapist provided opportunities to violate this expectation, as the therapist's responses to disclosure (warmth and validation) were inconsistent with judgmental behaviors. Julia was encouraged to share her concerns and elements of her story with close friends to provide additional violation of this conditional association.

In addition to these concerns, Julia reported that the traumatic images were inherently aversive given their vivid nature. Julia's therapist discussed how repeated exposure would allow her to discriminate between the experience of the memory and the event itself (stimulus discrimination). Indeed, research suggests that repeated exposure leads to improved perceptual learning/stimulus discrimination (Tsodyks & Gilbert, 2004). Moreover, repeated exposure to the aversive elements of the memory may eventually reduce their salience, allowing the salience of non-threatening, contextual cues to come to the forefront. The addition of these contextual cues (e.g., the safety of the therapist's office) may facilitate attempts at discrimination. Although not directly related to inhibitory models discussed earlier, stimulus discrimination is an important concept in associative learning theories that may have relevance for intrusive images characterized by high degree of vividness.

This approach differed from an habituation-based model of exposure by targeting aspects of expectancy violation and stimulus discrimination, employing affective labeling, and tying exposure completion to behavioral goals rather than fear level. In addition, this approach differed from cognitive models inasmuch as it avoided inclusion of cognitive restructuring prior to, or during exposure, and employed exposure and extinction processes, rather than cognitive interventions, to target additional conditional reactions such as shame. Examples of several exposure trials are shown in Table 2.

Social phobia

Deandre is a 40-year-old male who was experiencing fears of social rejection and humiliation at treatment outset. Following an increase in social anxiety symptoms approximately one year ago, Deandre refused to apply for jobs or socialize with his wife's friends. His primary incentive for seeking treatment was the chance to repair his marriage, which had been strained over the past year due to his social avoidance.

Session 1 involved psychoeducation and treatment planning. The therapist discussed the prevalence, origins, and psychopathology of social phobia and the foundations of exposure-based psychotherapies (e.g., principles of associative conditioning). It was important to provide a detailed and frank description of what Deandre's responsibilities would be during exposure therapy in order to assess his willingness to follow through with a program that included behavioral assignments. One of the principles that Deandre carried forward from the initial session was the "personal scientist" approach to treatment, reflecting the emphasis on empiricism in this exposure therapy. That is, each exposure exercise was designed to evaluate a hypothesis, typically of the form "CS predicts US." In addition, the therapist explained that some exposures would entail sustained levels of fear and that the immediate goal of exposure was not fear reduction.

Sessions 2–5 were devoted to creating an inventory of feared social situations, collaboratively engineering the corresponding exposure exercises, and carrying out in- and between-session exposures. During the design of exposure exercises, Deandre's predicted fear level for each situation was recorded, but these predictions were not used to determine the order of exercises, as is common practice in habituation-based models. Instead, the emphasis was upon the hypothesis test, or learning, that needed to be accomplished in each scenario. The order of exposure exercises

Table 2
Example PTSD exposure exercises.

Session 3	
<u>BEFORE exposure:</u>	
Goal:	Go to a restaurant bar for 30 min, without cell phone or pepper spray I will be approached by drunken men who will grab me 60%
What are you most worried will happen?	
On scale 0–100, how likely does this seem?	
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	Although some men approached me, everyone was respectful and no one acted aggressively I can attend social events where people are drinking and still be safe
What did you learn?	
Session 5 (in session)	
<u>BEFORE exposure:</u>	
Goal:	Conduct imaginal exposure for 20 min (listen to recording 4× over the course of the week) I will be unable to handle the distress and will run out of the room 80%
What are you most worried will happen?	
On scale 0–100, how likely does this seem?	
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	I stayed the whole time I can begin to face these scary memories
What did you learn?	
Session 7 (in session)	
<u>BEFORE exposure:</u>	
Goal:	Conduct imaginal exposure for 20 min, then respond to work emails for 10 min. I will be unable to respond to emails effectively 70%
What are you most worried will happen?	
On scale 0–100, how likely does this seem?	
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	I reread the emails and they made sense. People responded to the emails as if they understood my emails I can still get things done after facing the memory
What did you learn?	

was guided by what Deandre judged to be the most pressing learning experience or hypothesis test for him at any given point in treatment. For instance, at treatment outset, Deandre was most concerned with learning that his wife's friends were not likely to humiliate him, and exposures initially concentrated on attending social events with his wife, even though such exercises were rated as more fear provoking than other avoided situations (e.g., encounters with grocery clerks).

The typical exposure exercise was fairly structured and involved multiple stages. First, the therapist established what Deandre expected to happen in the social situations he perceived as threatening. This prediction was recorded on a standard worksheet and labeled the *hypothesis*. One of his hypotheses was that if he expressed an opinion to a coworker, he would be regarded as incompetent, as evidenced by quizzical stares, raised eyebrows, and avoidance. (Deandre learned over successive exercises to render his hypotheses in behaviorally specific terms, given that vague hypotheses are exceedingly difficult to support or refute in any objective sense.) Second, Deandre performed the social behavior and observed the result. Like any good scientist, he recorded coworkers' responses on paper using objective language. The behavioral description of the response was treated as the result of

the hypothesis test, or *evidence*. Third, Deandre and the therapist compared the hypothesis with the evidence. At first, the therapist Socratically guided him through this process by asking such questions as “Is the evidence consistent with what you predicted?” and “Did you learn anything about your coworkers’ responses to you?” As therapy progressed, Deandre performed this *consolidation* on his own, with reference as needed to a standard set of questions about the CS-US association.

Although the consolidation process can be considered a form of cognitive therapy, there are several ways in which Deandre’s treatment diverged from traditional cognitive-behavioral therapies for social phobia. First, cognitive restructuring was not implemented prior to exposures to reduce anticipatory anxiety or otherwise prepare him for the exposure trials. The rationale behind this decision was to maintain US expectancy prior to exposure so that the trial could produce maximal *violation* of the expectancy. The statement “Does it really make sense to be afraid of next weekend’s party? What’s happened at the past few parties you’ve attended?” would be considered useful pre-exposure restructuring in some therapies, but would in fact be expected to limit the inhibitory learning thought to follow from expectancy violation. Second, cognitive restructuring in the midst of exposures was not encouraged. In traditional cognitive-behavioral therapy, Deandre might have been asked to attend to the non-threatening or positive elements of a social situation during an exposure exercise as a way of demonstrating that not all parts of the event were negative. However, this strategy also could distract Deandre from the CS-no US relation, and therefore could ultimately diminish inhibitory learning.

There were several features of these exposure trials that distinguished them from an habituation-based approach. The principal difference was the emphasis on expectancy violation, rather than fear levels, in the design and consolidation stages. The rigor with which the therapist elicited an objective, behaviorally-oriented hypothesis for each exposure exercise and the Socratic questioning regarding discrepancies between hypothesis and evidence followed from the centrality of expectancy violation to the inhibitory learning model, and is consistent with the “behavioral testing model” of exposure within cognitive behavioral therapy. Additionally, Deandre’s exposures were tailored, to some extent, to increase variability of fear induced both within and across exercises, and sometimes involved sustained fear, in ways that differed from both habituation-based and “behavioral testing” approaches to exposure therapy. For instance, there was no linear relation between the number of exposure exercises he completed and his initial or ending subjective fear ratings during a given exercise. He reached peak fear levels during some of his earliest exposures and some of his last. Also, during several exposures, Deandre reported elevated fear levels throughout. Since the length of exposures did not depend on subjective fear, many of them ended without significant decrements in fear ratings.

In Sessions 6–12, the therapist helped Deandre to design augmented exposures to enhance inhibitory learning. First, subtle safety behaviors related to Deandre’s speech in social interactions were identified and eliminated. For instance, Deandre was discouraged from fidgeting with his hands, wearing earphones, or bringing a magazine to read as a way of distracting himself during exposure exercises. As safety behaviors were eliminated, Deandre maximally attended to the associations (or lack thereof) between the CS (social environment) and the US (specific changes in the facial expressions and gaze of his interlocutor(s)). This change in attentional focus permitted maximum violation of a CS-US expectancies (i.e., hypotheses). Second, and along these same lines, Deandre’s therapist worked with him to counter his tendency to imaginably replay perceived negative aspects of social encounters

following exposures. To the extent that this “post event processing” interfered with his awareness of the non-occurrence of the US, it may have disrupted extinction learning. By refocusing his attention towards concrete behavioral indicators of the non-occurrence of rejection, Deandre was better able to discern the non-contingent relationship between social cues and aversive outcomes.

Third, Deandre carried out exposures that entailed a very high probability of negative social feedback. He performed several “shame attacks,” during which he deliberately acted in ways likely to elicit puzzled, embarrassed, or even scornful looks from others. Stated in terms of the inhibitory learning model, this procedure increased the chances of exposure to the US, or occasional reinforced extinction. An example shame attack was spending a therapy session in a building elevator and yelling out the floor numbers in a loud voice as people got on and off, paying special attention to riders’ facial and verbal reactions. Several other examples of exposure trials are presented in Table 3.

Table 3
Example social phobia exposure exercises.

Session 3	
<u>BEFORE exposure:</u>	
Goal:	Express a professional opinion to a coworker (4× over the course of the week)
What are you most worried will happen?	Coworker will stare at me contemptuously and walk away without responding
On scale 0–100, how likely does this seem?	95%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	Coworker responded immediately, agreed with my opinion, and we continued talking
What did you learn?	Coworkers do not always disregard my opinions
Session 4	
<u>BEFORE exposure:</u>	
Goal:	Predicting outcomes of sporting events to people at the local gym and bar (no alcohol)
What are you most worried will happen?	People will look at me scornfully (furrowed brows and squinted eyes) and turn away
On scale 0–100, how likely does this seem?	80%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	People responded with their own predictions. They did not appear scornful
What did you learn?	Strangers won’t necessarily reject my conversation
Session 9	
<u>BEFORE exposure:</u>	
Goal:	Ride elevator at the local mall for 30 min calling out the names of the floors in a loud voice (4× over the course of the week)
What are you most worried will happen?	People will look angrily at me, I will feel humiliated, and I will cry and leave the elevator
On scale 0–100, how likely does this seem?	90%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	I got more puzzled looks than angry ones. I DID feel humiliated, but I did not cry and I was able to remain in the elevator for 30 min
What did you learn?	Even when I feel humiliated, it’s a temporary state, and I can ultimately tolerate it

Specific phobia

Sharon is a 25-year-old female who sought treatment for a dog phobia. She had been afraid of dogs, especially large ones, since she witnessed her older sister being chased and bitten by a dog when Sharon was 10 years old. The phobia was problematic insofar as several of her closest friends had pet dogs at home and she refused to visit them, a decision that caused significant friction in those relationships. Additionally, she had recently quit her recreational soccer league—a very important leisure activity for Sharon—because her teammates regularly brought their dogs to games and practices.

Session 1 included thorough assessment of situational avoidance behaviors and discussion about how the exposure therapy model could help Sharon regain the social and leisure activities that were affected by the dog phobia. The therapist explained the parallels between systematic exposure exercises and hypothesis testing in scientific research. Sharon acknowledged that the exposure program would involve coming into contact with situations that were previously feared and avoided, and that the goal was to experience these situations in a way that allowed for new learning, rather than to achieve immediate fear reduction. Also in Session 1, the specific behavioral goals for the treatment were carefully defined. The therapist stated that therapy could be reasonably terminated at any point once the performance goals were met, but 6–12 sessions were recommended so that the basic principles of exposure, as well as the specific inhibitory learning strategies, could be communicated and rehearsed.

Sessions 2–5 were used for repeated practice of in vivo exposure. Sharon was taught to use a worksheet to record feared situations and what she hypothesized would occur in each situation. For one exercise, Sharon wrote that she avoided “standing on the sideline during a soccer match” because she predicted that “one of my teammates’ dogs will bite me.” The therapist was alert for opportunities to help Sharon increase the specificity of her hypotheses because, as in scientific research, hypotheses must be specific enough to be refutable. As such, Sharon was prompted to elaborate on the features of the soccer field sideline that were maximally predictive of a dog bite: “standing on the sideline within 10 yards of a dog for 10 min at a soccer match.” Sharon was then tasked with approaching this situation during a between-session soccer game and documenting the result on her worksheet. The therapist even challenged her to spend an extra 5 min in the sideline environment to provide an especially rigorous test of her dog bite hypothesis. During the following session, the therapist coached Sharon on how to methodically compare the results of the hypothesis test (i.e., evidence) with her hypothesis. Sharon reported that her experiential evidence refuted her hypothesis (i.e., no dog bite occurred), and she worked with the therapist to generate a revised, more plausible, characterization of the CS-US association (e.g., “I can stand next to a dog for the whole soccer game and it won’t bite me”). It was critical to note that this exposure and others like it do not involve remaining in the avoided situation until fear subsides. Instead, the exposures were geared toward expectancy violation; that is, the offset of the exposure exercise was determined by the specifications of the temporal hypothesis (dog bite will occur within 10 min). Indeed, Sharon reported that her fear had not decreased substantially at the termination of the sideline exposure.

Sessions 6–12 augmented exposures with strategies drawn from inhibitory learning research. The principle of multiple contexts was especially relevant: that is, the contexts of exposure were deliberately varied over time to enhance retrieval and generalizability of inhibitory learning. For instance, exposures were designed to have Sharon approach dogs of various sizes and in multiple environments, especially in situations that were likely to be most

important to Sharon after therapy was completed (e.g., at friends’ houses and at the soccer field). She also completed exposure exercises by herself (e.g., without friends present at between-session exercises), as having others present to appease an aggressive dog could have served as a safety signal and prevented full violation of her expectation of a dog attacking her. Given that variability in exposure contexts was valued more than repeating an exposure in one context until fear subsided, fear levels were not uniformly lower as therapy progressed.

Exposures also varied with respect to internal contexts, most notably Sharon’s fear levels during the exercise. Although Sharon’s exposures began with smaller dogs to reduce the likelihood of treatment rejection, the therapist did not progressively assign slightly more feared exposure exercises over time in the sense of linearly following an exposure “ladder” or hierarchy. As a result, there was substantial variability in Sharon’s self-reported fear across exposure trials, and some trials terminated—after the goal of the exercise was reached and/or the stated expectancy was violated—when self-reported fear was relatively high. Examples of specific exposure trials are presented in Table 4.

Panic disorder

Charlie is a 43-year-old male who manages a construction company. While he had no major health concerns throughout his

Table 4

Example specific phobia exposure exercises.

Session 4	
<u>BEFORE exposure:</u>	
Goal:	Stand on the sideline within 10 yards of a dog for 15 min at a soccer match
What are you most worried will happen?	Before 10 min are up, a dog will bite me
On scale 0–100, how likely does this seem?	99%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	The dog never approached me
What did you learn?	I can probably stand on the sideline for a whole game and not get bitten
Session 5	
<u>BEFORE exposure:</u>	
Goal:	Pet [her friend] Katie’s dog for 30 min
What are you most worried will happen?	He will bite me
On scale 0–100, how likely does this seem?	85%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	He never bit me and seemed to enjoy my company (licked my hand, stayed in my lap)
What did you learn?	Some dogs do not bite when they are petted
Session 10	
<u>BEFORE exposure:</u>	
Goal:	Watch a whole soccer game (90 min) while seated on the ground, holding the leashes of two dogs [that belong to her teammates]
What are you most worried will happen?	Dog will bite me and I won’t be in a position to defend myself or run away
On scale 0–100, how likely does this seem?	70%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	The dogs did not make any menacing gestures and seemed to get used to me over time
What did you learn?	I can be in a relatively vulnerable position around dogs

Table 5
Example panic disorder exposure exercises.

Session 2	
<u>BEFORE exposure:</u>	
Goal:	Spin in a circle for 60 s
What are you most worried will happen?	I will have a stroke
On scale 0–100, how likely does this seem?	85%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	I remained conscious and didn't feel any pain.
What did you learn?	Feeling dizzy doesn't necessarily mean I will have a stroke
Session 8	
<u>BEFORE exposure:</u>	
Goal:	Go for a 15-min jog
What are you most worried will happen?	Having shortness of breath and a racing heart will make me have a heart attack
On scale 0–100, how likely does this seem?	75%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	My heart didn't stop
What did you learn?	Even when combined, I won't necessarily have a heart attack if I am short of breath and have a racing heart
Session 14	
<u>BEFORE exposure:</u>	
Goal:	Spin in a circle for 60 s and go for a 15-min jog without my pills or wife
What are you most worried will happen?	I may have a stroke or heart attack, and, if I do, I won't have my pills or wife with me to help me
On scale 0–100, how likely does this seem?	80%
<u>AFTER exposure:</u>	
Did what you were most worried about occur? Y or N	No
How do you know?	My heart didn't stop, I remained conscious, and I didn't feel any pain
What did you learn?	I probably won't have a stroke or heart attack, so I might not need my pills or wife present every time I feel these physical sensations

life, he experienced his first unexpected panic attack two years ago. Initially, these panic attacks occurred once every two months. In the past six months, however, his rate of panic attacks had increased to approximately once a week. Charlie was very worried about having additional panic attacks and what these panic attacks may mean for his health. He had visited doctors, and, despite favorable test results, was convinced his panic attacks would lead to one of two negative health outcomes: an imminent stroke or a heart attack. Specifically, he feared that interoceptive sensations (e.g., dizziness, shortness of breath, and racing heart) were either related to or could exacerbate an underlying medical condition. One doctor prescribed him benzodiazepines to reduce anxiety, which he took on an as-needed basis. Charlie had stopped exercising altogether, rarely engaged in hands-on construction work, and avoided playing effortful games with his children because he was afraid he would have a stroke or heart attack. To help him feel more comfortable, Charlie's wife joined him on visits to construction sites – where the dust could make it difficult for Charlie to breathe – and she played with the children so Charlie could rest. Charlie's panic attacks caused him great distress and impacted his lifestyle, which is why he sought psychological treatment.

Session 1 included discussions about associative learning, how avoidance prevents extinction learning, and the importance of exposures for eventual (not immediate) fear reduction. Charlie's therapist incorporated three extinction enhancement strategies throughout treatment: violation of expectancies, deepened extinction, and the removal of safety signals.

Sessions 2–7 were primarily focused on interoceptive exposure through induction of dizziness, shortness of breath, and a racing heart. To induce dizziness, Charlie would spin in a circle. Because Charlie believed there was an 85% chance a stroke or heart attack would occur after 30 s of feeling dizzy, he engaged in an exposure that was longer in duration: 60 s. This was done to maximize Charlie's violation of expectancies. To induce shortness of breath, Charlie would breathe through a straw, visit a dusty construction site, or exercise. These exposures were similarly designed to endure beyond the point where Charlie believed he would experience a

stroke or heart attack. Lastly, to induce racing heart, Charlie would drink caffeine. He drank more caffeine than the amount he expected would lead to a heart attack or stroke. Two sessions were devoted to each of these three interoceptive symptoms to violate the expectancy that Charlie would experience a stroke or heart attack.

Starting with Session 4, exposures included the gradual removal of safety signals, Charlie began attending therapy without his wife waiting outside of the room and was asked to no longer bring his benzodiazepines to session. By Session 7, Charlie was also able to conduct between-session exposures without the presence of safety signals.

Sessions 8–14 focused on deepened extinction, which involved combining multiple feared stimuli that have been extinguished in isolation in order to enhance extinction learning. For Charlie, this initially meant combining shortness of breath with accelerated heart rate. These exposures included drinking caffeine prior to exercising or playing with his children. Once Charlie completed these exposures, the therapist designed exposures to add dizziness (e.g., spinning in a circle before exercising or playing with his children). This combination made Charlie think there was a 99% chance he would have a stroke or heart attack. Thus, all three of Charlie's feared interoceptive symptoms were included in one exposure to maximize the violation of expectancies.

Notably, the exposures above differed from habituation-based models in an important way. Though Charlie rated his fear level before and after each exposure, *within-session* fear reduction was de-emphasized (e.g., how afraid he was of dizziness at the beginning and end of one session or one exposure). Instead, Charlie's therapist encouraged strategies that continually *increased* expectation and fear in order to facilitate extinction learning.

In addition, this approach differed from cognitive models which emphasize reappraisal of catastrophic misinterpretations and attention to possible signs of environmental safety prior to, or during exposure (Clark & Beck, 2010). For example, in traditional cognitive-behavioral interventions for panic disorder, a client may be asked to evaluate the likelihood that an elevated heart rate

would lead to a heart attack by examining evidence: “How many times have you had an elevated heart rate? How many heart attacks have you had? What were the results of your last physical?” Reducing catastrophic appraisals and directing attention to possible safety signals in the environment before exposure therapy may inadvertently impact extinction learning by reducing expectancy and mitigating attention to excitatory conditional stimuli. However, such cognitive reappraisal can be conducted post-exposure in order to consolidate the learning that has taken place.

Examples of exposure trials are presented in Table 5.

Summary

The translation from extinction learning to exposure therapy for fear and anxiety disorders involves directly targeting the initial acquisition, consolidation, and later retrieval of new learning. While the focus of the exposure may differ depending on the psychological condition being treated, in each case exposure therapy will generally contain the following elements. First is the specific goal of the exposure therapy: together, the therapist and client decide on the specific goal of the practice in terms of duration or behavioral goals in specific and measurable terms. Second is the anticipated negative outcome: the therapist elicits from the client the particular feared outcome of engaging in the task. Exposures are then designed in such a way and proceed until a given anticipation or expectation is violated. Third is recognition and consolidation of the non-occurrence of the anticipated event: following completion of an exposure practice, therapists aid clients in discussing the non-occurrence of the feared event. This reflects consolidating the new learning regarding the non-contingent relationship between the conditional stimulus and the unconditional stimulus. In addition, exposure includes “inhibitory learning enhancement and inhibitory regulation enhancement strategies”, including deepened extinction (or exposure to multiple feared cues), occasionally reinforced extinction (or occasional exposure to aversive outcomes), weaning from safety signals, stimulus and response variability, retrieval cues, multiple contexts, and affect labeling. Table 6 summarizes these strategies along with “catch phrases” we have found useful in expressing their rationale to clients.

Framing exposure within a modern learning theory perspective holds numerous advantages including providing a parsimonious explanation for shared elements of traditional exposure (or,

behavioral experiments), while simultaneously explaining their shortcomings. In addition, it ties clinical research to the wealth of research on learning theory in animal and human populations. Third, it holds promise for improving the efficacy of exposure-based procedures through selective targeting of associative learning mechanisms. Associative learning theories provide a parsimonious explanatory model from which to situate exposure processes. However, additional translational research is needed to further elucidate the optimal conditions necessary for enhancing inhibitory regulation and the precise methods for implementing these strategies in routine clinical care.

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References

- Agren, T., Engman, J., Frick, A., Björkstrand, J., Larsson, E. M., Furmark, T., et al. (2012). Disruption of reconsolidation erases a fear memory trace in the human amygdala. *Science*, 21, 1550–1552. <http://dx.doi.org/10.1126/science.1223006>.
- Arch, J. J., & Craske, M. G. (2009). First-line treatment: a critical appraisal of cognitive behavioral therapy developments and alternatives. *Psychiatric Clinics of North America*, 32(3), 525–547. <http://dx.doi.org/10.1016/j.psc.2009.05.001>.
- Baker, K. D., McNally, G. P., & Richardson, R. (2013). Memory retrieval before or after extinction reduces recovery of fear in adolescent rats. *Learning & Memory*, 20(9), 467–473. <http://dx.doi.org/10.1101/lm.031989.113>.
- Baker, A., Mystkowski, J., Culver, N., Yi, R., Mortazavi, A., & Craske, M. G. (2010). Does habituation matter? Emotional processing theory and exposure therapy for acrophobia. *Behaviour Research and Therapy*, 48, 1139–1143. <http://dx.doi.org/10.1016/j.brat.2010.07.009>.
- Bandarian Balooch, S., & Neumann, D. L. (2011). Effects of multiple contexts and context similarity on the renewal of extinguished conditioned behaviour in an ABA design with humans. *Learning and Motivation*, 42(1), 53–63. <http://dx.doi.org/10.1016/j.lmot.2010.08.008>.
- Bandarian Balooch, S., Neumann, D., & Bosch, M. J. (2012). Extinction treatment in multiple contexts attenuates ABC renewal in humans. *Behaviour Research and Therapy*, 50, 604–609. <http://dx.doi.org/10.1016/j.brat.2012.06.003>.
- Barlow, D. H., & Craske, M. G. (1994). *Mastery of your anxiety and panic II*. Albany, NY: Graywind Publications Incorporated <http://dx.doi.org/10.1017/S1352465809005141>.
- Beckers, T., Krypotos, A. M., Boddez, Y., Effting, M., & Kindt, M. (2013). What's wrong with fear conditioning? *Biological Psychology*, 92(1), 90–96. <http://dx.doi.org/10.1016/j.biopsycho.2011.12.015>.
- Bjork, R. A., & Bjork, E. L. (1992). A new theory of disuse and an old theory of stimulus fluctuation. In A. Healy, S. Kosslyn, & R. Shiffrin (Eds.), *From learning processes to cognitive processes: Essays in honor of William K. Estes*. Hillsdale, NJ: Erlbaum.
- Bjork, R. A., & Bjork, E. L. (2006). Optimizing treatment and instruction: implications of a new theory of disuse. In L. G. Nilsson, & N. Ohta (Eds.), *Memory and society: Psychological perspectives*. New York: Psychology Press.
- Bouton, M. E. (1993). Context, time, and memory retrieval in the interference paradigms of Pavlovian learning. *Psychological Bulletin*, 114, 80–99. <http://dx.doi.org/10.1037/0033-2909.114.1.80>.
- Bouton, M. E., García-Gutiérrez, A., Zilski, J., & Moody, E. W. (2006). *Extinction in multiple contexts does not necessarily make extinction less vulnerable to relapse*.
- Bouton, M. E., & King, D. A. (1983). Contextual control of the extinction of conditioned fear: tests for the associative value of the context. *Journal of Experimental Psychology: Animal Behavior Processes*, 9, 248–265. <http://dx.doi.org/10.1037/0097-7403.9.3.248>.
- Bouton, M. E., Woods, A. M., & Pineño, O. (2004). Occasional reinforced trials during extinction can slow the rate of rapid reacquisition. *Learning and Motivation*, 35(4), 371–390. <http://dx.doi.org/10.1016/j.lmot.2004.05.001>.
- Brooks, D. C., & Bouton, M. E. (1994). A retrieval cue for extinction attenuates response recovery (renewal) caused by a return to the conditioning context. *Journal of Experimental Psychology: Animal Behavior Processes*, 20, 366–379. <http://dx.doi.org/10.1037/0097-7403.20.4.366>.
- Cain, C. K., Blouin, A. M., & Barad, D. (2004). Adrenergic transmission facilitates extinction of conditional fear in mice. *Learning & Memory*, 11, 179–187. <http://dx.doi.org/10.1101/lm.71504>.
- Chan, W. Y., Leung, H. T., Westbrook, R. F., & McNally, G. P. (2010). Effects of recent exposure to a conditioned stimulus on extinction of Pavlovian fear conditioning. *Learning and Memory*, 17, 512–521. <http://dx.doi.org/10.1101/lm.191250>.
- Clark, D. A., & Beck, A. T. (2010). *Cognitive therapy of anxiety disorders: Science and practice*. New York, NY: Guilford Press.
- Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy.

Table 6
Strategies for enhancing inhibitory learning.

Strategy	Description	Catch-phrase
Expectancy violation	Design exposures to violate specific expectations	<i>Test it Out</i>
Deepened extinction	Present two cues during the same exposure after conducting initial extinction with at least one of them	<i>Combine It</i>
Reinforced extinction	Occasionally present the US during exposures	<i>Face Your Fear</i>
Variability	Vary stimuli and contexts	<i>Vary It Up</i>
Remove safety behaviors	Decrease the use of safety signals and behaviors	<i>Throw It Out</i>
Attentional focus	Maintain attention on the target CS during exposure	<i>Stay With It</i>
Affect labeling	Encourage the clients to describe their emotional experience during exposure	<i>Talk It Out</i>
Mental reinstatement/ retrieval cues	Use a cue present during extinction or imaginatively reinstate previous successful exposures	<i>Bring It Back</i>

- Behaviour Research and Therapy, 46, 5–27. <http://dx.doi.org/10.1016/j.brat.2007.10.003>.
- Craske, M. G., Liao, B., Brown, L., & Verliet, B. (2012). Role of inhibition in exposure therapy. *Journal of Experimental Psychopathology*, 3, 322–345. <http://dx.doi.org/10.5127/jep.026511>.
- Craske, M. G., & Mystkowski, J. (2006). Exposure therapy and extinction: clinical studies. In M. G. Craske, D. Hermans, & D. Vansteenwegen (Eds.), *Fear and learning: Basic science to clinical application*. Washington, DC: APA Books.
- Craske, M. G., & Rachman, S. (1986). Return of fear: heart rate responsivity and perceived skill. *British Journal of Clinical Psychology*, 26, 187–199. <http://dx.doi.org/10.1111/j.2044-8260.1987.tb01346.x>.
- Craske, M. G., Waters, A. M., Bergman, L. R., Naliboff, B., Lipp, O. V., Negoro, H., et al. (2008). Is aversive learning a marker of risk for anxiety disorders in children? *Behaviour Research and Therapy*, 46, 954–967. <http://dx.doi.org/10.1016/j.brat.2008.04.011>.
- Culver, N. C., Stephens, S., & Craske, M. G. (2014). *Occasional reinforced trials during extinction attenuates spontaneous recovery and rapid reacquisition* (in preparation-a).
- Culver, N. C., Stoyanova, M., & Craske, M. G. (2011). Clinical relevance of retrieval cues for attenuating context renewal of fear. *Journal of Anxiety Disorders*, 25, 284–292. <http://dx.doi.org/10.1016/j.janxdis.2010.10.002>.
- Culver, N., Stoyanova, M. S., & Craske, M. G. (2012). Emotional variability and sustained arousal during exposure. *Journal of Behavior Therapy and Experimental Psychiatry*, 2, 783–793. <http://dx.doi.org/10.1016/j.jbtep.2011.10.009>.
- Culver, N. C., Vervliet, B., & Craske, M. G. (2014). *Compound stimulus presentation deepens extinction learning* (in preparation-b).
- Davey, G. C. (1992). Classical conditioning and the acquisition of human fears and phobias: a review and synthesis of the literature. *Advances in Behaviour Research and Therapy*, 14, 29–66. [http://dx.doi.org/10.1016/0146-6402\(92\)90010-L](http://dx.doi.org/10.1016/0146-6402(92)90010-L).
- Deacon, B., Kemp, J. J., Dixon, L. J., Sy, J. T., Farrell, N. R., & Zhang, A. R. (2013). Maximizing the efficacy of interoceptive exposure by optimizing inhibitory learning: a randomized controlled trial. *Behaviour Research and Therapy*, 51, 588–596. <http://dx.doi.org/10.1016/j.brat.2013.06.006>.
- Deacon, B. J., Sy, J., Lickel, J. J., & Nelson, E. O. (2010). Does the judicious use of safety behaviors improve the efficacy and acceptability of exposure therapy for claustrophobic fear? *Journal of Behavior Therapy and Experimental Psychiatry*, 41, 71–80. <http://dx.doi.org/10.1016/j.jbtep.2009.10.004>.
- Dibbets, P., Havermans, R., & Arntz, A. (2008). All we need is a cue to remember: the effect of an extinction cue on renewal. *Behaviour Research and Therapy*, 46, 1070–1077. <http://dx.doi.org/10.1016/j.brat.2008.05.007>.
- Dibbets, P., & Maes, J. H. R. (2011). The effect of an extinction cue on ABA-renewal: does valence matter? *Learning and Motivation*, 42, 133–144. <http://dx.doi.org/10.1016/j.lmot.2010.12.003>.
- Estes, W. K. (1955). Statistical theory of distributional phenomena in learning. *Psychological Review*, 62, 369–377. <http://dx.doi.org/10.1037/h0046888>.
- Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: exposure to corrective information. *Psychological Bulletin*, 99, 20–35. <http://dx.doi.org/10.1037/0033-2909.99.1.20>.
- Foa, E. B., & McNally, R. J. (1996). Mechanisms of change in exposure therapy. In R. M. Rapee (Ed.), *Current controversies in the anxiety disorders* (pp. 214–227). New York, NY: Guilford Press.
- Gallistel, C. R., & Gibbon, J. (2000). Time, rate, and conditioning. *Psychological Review*, 107, 289–344.
- Grillon, C. (2008). Models and mechanisms of anxiety: evidence from startle studies. *Psychopharmacology*, 199(3), 421–437. <http://dx.doi.org/10.1007/s00213-007-1019-1>.
- Gunther, L. M., Denniston, J. C., & Miller, R. R. (1998). Conducting exposure treatment in multiple contexts can prevent relapse. *Behaviour Research and Therapy*, 36, 75–91. [http://dx.doi.org/10.1016/S0005-7967\(97\)10019-5](http://dx.doi.org/10.1016/S0005-7967(97)10019-5).
- Hermans, D., Craske, M. G., Mineka, S., & Lovibond, P. F. (2006). Extinction in human fear conditioning. *Biological Psychiatry*, 60(4), 361–368. <http://dx.doi.org/10.1016/j.biopsych.2005.10.006>.
- Hermans, D., Dirikx, T., Vansteenwegen, 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. <http://dx.doi.org/10.1016/j.brat.2004.03.013>.
- Hofmann, S. G., & Smits, J. A. (2008). Cognitive-behavioral therapy for adult anxiety disorders: a meta-analysis of randomized placebo-controlled trials. *Journal of Clinical Psychiatry*, 69, 621–632. <http://dx.doi.org/10.4088/JCP.v69n0415>.
- Indovina, I., Robbins, T. W., Nunez-Elizalde, A. O., Dunn, B. D., & Bishop, S. J. (2011). Fear-conditioning mechanisms associated with trait vulnerability to anxiety in humans. *Neuron*, 10, 563–571. <http://dx.doi.org/10.1016/j.neuron.2010.12.034>.
- Joos, E. (2011). *Repetitive thought in human (fear) conditioning: Strengthening the acquisition and extinction memory* (Doctoral dissertation).
- Jovanovic, T., Norrholm, S. D., Blanding, N. Q., Davis, M., Duncan, E., Bradley, B., et al. (2010). Impaired fear inhibition is a biomarker of PTSD but not depression. *Depression & Anxiety*, 27, 244–251. <http://dx.doi.org/10.1002/da.20663>.
- Kircanski, K., Mortazavi, A., Castriotta, N., Baker, A. S., Mystkowski, J. L., Yi, R., et al. (2012). Challenges to the traditional exposure paradigm: variability in exposure therapy for contamination fears. *Journal of Behavior Therapy and Experimental Psychiatry*, 43(2), 745–751. <http://dx.doi.org/10.1016/j.jbtep.2011.10.010>.
- Lader, M. H., & Mathews, A. M. (1968). A physiological model of phobic anxiety and desensitization. *Behaviour Research and Therapy*, 6, 411–421. [http://dx.doi.org/10.1016/0005-7967\(68\)90021-1](http://dx.doi.org/10.1016/0005-7967(68)90021-1).
- Lang, A. J., & Craske, M. G. (2000). Manipulations of exposure-based therapy to reduce return of fear: a replication. *Behaviour Research and Therapy*, 38, 1–12. [http://dx.doi.org/10.1016/S0005-7967\(99\)00031-5](http://dx.doi.org/10.1016/S0005-7967(99)00031-5).
- Lieberman, M. D., Eisenberger, N. I., Crockett, M. J., Tom, S. M., Pfeifer, J. H., & Way, B. M. (2007). Putting feelings into words. *Psychological Science*, 18, 421–428. <http://dx.doi.org/10.1111/j.1467-9280.2007.01916.x>.
- Lissek, S., Powers, A. S., McClure, E. B., Phelps, E. A., Woldehawariat, G., Grillon, C., et al. (2005). Classical fear conditioning in the anxiety disorders: a meta-analysis. *Behaviour Research and Therapy*, 43, 1391–1424. <http://dx.doi.org/10.1016/j.brat.2004.10.007>.
- Lovibond, P. F., Davis, N. R., & O'Flaherty, A. S. (2000). Protection from extinction in human fear conditioning. *Behaviour Research and Therapy*, 38, 967–983.
- Mackintosh, N. J. (1975). A theory of attention: variations in the associability of stimuli with reinforcement. *Psychological Review*, 82(4), 276. <http://dx.doi.org/10.1037/h0076778>.
- Magill, R. A., & Hall, K. G. (1990). A review of the contextual interference effect in motor skill acquisition. *Human Movement Science*, 9, 241–289. [http://dx.doi.org/10.1016/0167-9457\(90\)90005-X](http://dx.doi.org/10.1016/0167-9457(90)90005-X).
- Meeter, M., & Murre, J. M. J. (2004). Consolidation of long-term memory: evidence and alternatives. *Psychological Bulletin*, 130, 843–857. <http://dx.doi.org/10.1037/0033-2909.130.6.843>.
- Meuret, A. E., Wolitzky-Taylor, K. B., Twohig, M. P., & Craske, M. G. (2012). Coping skills and exposure therapy in panic disorder and agoraphobia: latest advances and future directions. *Behavior Therapy*, 43(2), 271–284. <http://dx.doi.org/10.1016/j.beth.2011.08.002>.
- Milad, M. R., Furtak, S. C., Greenberg, J. L., Keshaviah, A., Im, J. J., Falkenstein, M. J., et al. (2013). Deficits in conditioned fear extinction in obsessive-compulsive disorder and neurobiological changes in the fear circuit. *Journal of the American Medical Association*, 70, 608–618. <http://dx.doi.org/10.1001/jamapsychiatry.2013.914>.
- Milad, M. R., Pitman, R. K., Ellis, C. B., Gold, A. L., Shin, L. M., Lasko, N. B., et al. (2009). Neurobiological basis of failure to recall extinction memory in posttraumatic stress disorder. *Biological Psychiatry*, 66, 1075–1082. <http://dx.doi.org/10.1016/j.biopsych.2009.06.026>.
- Milad, M. R., Wright, C. I., Orr, S. P., Pitman, R. K., Quirk, G. J., & Rauch, S. L. (2007). Recall of fear extinction in humans activates the ventromedial prefrontal cortex and hippocampus in concert. *Biological Psychiatry*, 62, 446–454. <http://dx.doi.org/10.1016/j.biopsych.2006.10.011>.
- Miller, R. R., & Matzel, L. D. (1988). Contingency and relative associative strength. In S. B. Klein, & R. R. Mowrer (Eds.), *Contemporary learning theories: Pavlovian conditioning and the status of traditional learning theory* (pp. 61–84). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Mineka, S., Mystkowski, J. L., Hladek, D., & Rodriguez, B. I. (1999). The effects of changing contexts on return of fear following exposure therapy for spider fear. *Journal of Consulting and Clinical Psychology*, 67, 599–604. <http://dx.doi.org/10.1037/0022-006X.67.4.599>.
- Monfils, M. H., Cowansage, K. K., Klann, E., & LeDoux, J. E. (2009). Extinction-reconsolidation boundaries: key to persistent attenuation of fear memories. *Science*, 324, 951–955. <http://dx.doi.org/10.1126/science.1167975>.
- Myers, K. M., & Davis, M. (2007). Mechanisms of fear extinction. *Molecular Psychiatry*, 12, 120–150.
- Mystkowski, J. L., Craske, M. G., & Echiverri, A. M. (2002). Treatment context and return of fear in spider phobia. *Behavior Therapy*, 33, 399–416. [http://dx.doi.org/10.1016/S0005-7894\(02\)80035-1](http://dx.doi.org/10.1016/S0005-7894(02)80035-1).
- Mystkowski, J. L., Craske, M. G., Echiverri, A. M., & Labus, J. S. (2006). Mental reinstatement of context and return of fear in spider-fearful participants. *Behavior Therapy*, 37, 49–60. <http://dx.doi.org/10.1016/j.beth.2005.04.001>.
- Mystkowski, J. L., Mineka, S., Vernon, L. L., & Zinbarg, R. E. (2003). Changes in caffeine states enhance return of fear in spider phobia. *Journal of Consulting and Clinical Psychology*, 71, 243–250. <http://dx.doi.org/10.1037/0022-006X.71.2.243>.
- Nader, K., Schafe, G. E., & LeDoux, J. E. (2000). Fear memories require protein synthesis in the amygdala for reconsolidation after retrieval. *Nature*, 406, 722–726. <http://dx.doi.org/10.1038/35021052>.
- Neumann, D. L., Lipp, O. V., & Cory, S. E. (2007). Conducting extinction in multiple contexts does not necessarily attenuate the renewal of shock expectancy in a fear-conditioning procedure with humans. *Behaviour Research and Therapy*, 45, 385–394. <http://dx.doi.org/10.1016/j.brat.2006.02.001>.
- Norton, P. J., & Price, E. C. (2007). A meta-analytic review of adult cognitive-behavioral treatment outcome across the anxiety disorders. *Journal of Nervous and Mental Disease*, 195, 521–531.
- Pearce, J. M., & Hall, G. (1980). A model for Pavlovian learning: variations in the effectiveness of conditioned but not of unconditioned stimuli. *Psychological Review*, 87(6), 532. <http://dx.doi.org/10.1037/0033-295X.87.6.532>.
- Plendl, W., & Wotjak, C. T. (2010). Dissociation of within-and between-session extinction of conditioned fear. *The Journal of Neuroscience*, 30(14), 4990–4998. <http://dx.doi.org/10.1523/JNEUROSCI.6038-09.2010>.
- Podina, I. R., Koster, E. H., Philippot, P., Dethiuer, V., & David, D. O. (2013). Optimal attentional focus during exposure in specific phobia: a meta-analysis. *Clinical Psychology Review*, 33, 1172–1183.
- Ponnusamy, R., Zhuravka, I., Poulos, A. M., Huang, J., Wolke, D., O'Neil, P. K., & Fanselow, M. S. (August 2011). *Retrieval vs reconsolidation accounts of deepened extinction with reminder treatments*. Poster presented at the Gordon research conference.
- Prenoveau, J. M., Craske, M. G., Liao, B., & Ornitz, E. M. (2013). Human fear conditioning and extinction: timing is everything... or is it? *Biological Psychology*, 92(1), 59–68. <http://dx.doi.org/10.1016/j.biopsycho.2012.02.005>.

- Quirk, G. J. (2002). Memory for extinction of conditioned fear is long-lasting and persists following spontaneous recovery. *Learning & Memory*, 9, 402–407. <http://dx.doi.org/10.1101/lm.49602>.
- Rachman, S., Shafraan, R., Radomsky, A. S., & Zysk, E. (2011). Reducing contamination by exposure plus safety behaviour. *Journal of Behavior Therapy and Experimental Psychiatry*, 42, 397–404. <http://dx.doi.org/10.1016/j.jbtep.2011.02.010>.
- Rescorla, R. A. (2000). Extinction can be enhanced by a concurrent excitator. *Journal of Experimental Psychology*, 26, 251–260.
- Rescorla, R. A. (2006). Deepened extinction from compound stimulus presentation. *Journal of Experimental Psychology: Animal Behavior Processes*, 32, 135–144. <http://dx.doi.org/10.1037/0097-7403.32.2.135>.
- Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: variations in the effectiveness of reinforcement and non-reinforcement. In A. H. Prokasy (Ed.), *Classical conditioning II: Current research and theory* (pp. 64–99). New York: Appleton-Century-Croft.
- 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.
- Ricker, S. T., & Bouton, M. E. (1996). Reacquisition following extinction in appetitive conditioning. *Animal Learning & Behavior*, 24, 423–436. <http://dx.doi.org/10.3758/BF03199014>.
- Rodriguez, B. I., Craske, M. G., Mineka, S., & Hladek, D. (1999). Context-specificity of relapse: effects of therapist and environmental context on return of fear. *Behaviour Research and Therapy*, 37, 845–862. [http://dx.doi.org/10.1016/S0005-7967\(98\)00106-](http://dx.doi.org/10.1016/S0005-7967(98)00106-).
- Rougemon-Bucking, A., Linnman, C., Zeffiro, T. A., Zeidan, M. A., Lebron-Milad, K., Rodriguez-Romaguera, J., et al. (2011). Altered processing of contextual information during fear extinction in PTSD: an fMRI study. *CNS Neuroscience & Therapeutics*, 17, 227–236. <http://dx.doi.org/10.1111/j.1755-5949.2010.00152.x>.
- Rowe, M. K., & Craske, M. G. (1998). Effects of varied-stimulus exposure training on fear reduction and return of fear. *Behaviour Research and Therapy*, 36, 719–734. [http://dx.doi.org/10.1016/S0005-7967\(97\)10017-1](http://dx.doi.org/10.1016/S0005-7967(97)10017-1).
- Salkovskis, P. M. (1991). The importance of behaviour in the maintenance of anxiety and panic: a cognitive account. *Behavioural Psychotherapy*, 19, 6–19. <http://dx.doi.org/10.1017/S0141347300011472>.
- Salkovskis, P. M., Hackmann, A., Wells, A., Gelder, M. G., & Clark, D. A. (2006). Belief disconfirmation versus habituation approaches to situational exposure in panic disorder with agoraphobia: a pilot study. *Behaviour Research and Therapy*, 45, 877–885. <http://dx.doi.org/10.1016/j.brat.2006.02.008>.
- Schiller, D., Monfils, M. H., Raio, C. M., Johnson, D. C., LeDoux, J. E., & Phelps, E. A. (2010). Preventing the return of fear in humans using reconsolidation update mechanisms. *Nature*, 463, 49–53. <http://dx.doi.org/10.1038/nature0863>.
- Schmidt, R. A., & Bjork, R. A. (1992). New conceptualization of practice: common principles in three paradigms suggest new concepts for training. *Psychological Science*, 3, 207–217. <http://dx.doi.org/10.1111/j.1467-9280.1992.tb00029.x>.
- Shea, J. B., & Morgan, R. L. (1979). Contextual interference effects on the acquisition, retention, and transfer of a motor skill. *Journal of Experimental Psychology: Human Learning and Memory*, 5, 179–187. <http://dx.doi.org/10.1037/0278-7393.5.2.179>.
- Shin, L. M., & Liberzon, I. (2010). The neurocircuitry of fear, stress, and anxiety disorders. *Neuropsychopharmacology*, 35, 169–191. <http://dx.doi.org/10.1038/npp.2009.83>.
- Sloan, T., & Telch, M. J. (2002). The effects of safety-seeking behavior and guided threat reappraisal on fear reduction during exposure: an experimental investigation. *Behaviour Research and Therapy*, 40, 235–251. [http://dx.doi.org/10.1016/S0005-7967\(01\)00007-9](http://dx.doi.org/10.1016/S0005-7967(01)00007-9).
- Sy, J. T., Dixon, L. J., Lickel, J. J., Nelson, E. A., & Deacon, B. J. (2011). Failure to replicate the deleterious effects of safety behaviors in exposure therapy. *Behaviour Research and Therapy*, 49(5), 305–314. <http://dx.doi.org/10.1016/j.brat.2011.02.005>.
- Tabibnia, G., Lieberman, M. D., & Craske, M. G. (2008). The lasting effect of words on feelings: words may facilitate exposure effects to threatening images. *Emotion*, 8(3), 307–317. <http://dx.doi.org/10.1037/1528-3542.8.3.307>.
- Tsao, J. C. I., & Craske, M. G. (2000). Timing of treatment and return of fear: effects of massed, uniform, and expanding spaced exposure schedules. *Behavior Therapy*, 31, 479–497. [http://dx.doi.org/10.1016/S0005-7894\(00\)80026-X](http://dx.doi.org/10.1016/S0005-7894(00)80026-X).
- Tsodyks, M., & Gilbert, C. (2004). Neural networks and perceptual learning. *Nature*, 431(7010), 775–781. <http://dx.doi.org/10.1038/nature03013>.
- Van Damme, S., Crombez, G., Hermans, D., Koster, E. H., & Eccleston, C. (2006). The role of extinction and reinstatement in attentional bias to threat: a conditioning approach. *Behaviour Research and Therapy*, 44, 1555–1563.
- Vansteenwegen, D., Vervliet, B., Hermans, D., Beckers, T., Baeyens, F., & Eelen, P. (2006). Stronger renewal in human fear conditioning when tested with an acquisition retrieval cue than with an extinction retrieval cue. *Behaviour Research and Therapy*, 44, 1717–1725. <http://dx.doi.org/10.1016/j.brat.2005.10.014>.
- Vansteenwegen, D., Vervliet, B., Iberico, C., Baeyens, F., Van den Bergh, O., & Hermans, D. (2007). The repeated confrontation with videotapes of spiders in multiple contexts attenuates renewal of fear in spider-anxious students. *Behaviour Research and Therapy*, 45(6), 1169–1179. <http://dx.doi.org/10.1016/j.brat.2006.08.023>.
- Vervliet, B., Craske, M. G., & Hermans, D. (2013). Fear extinction and relapse: state of the art. *Annual Review of Clinical Psychology*, 9, 215–248. <http://dx.doi.org/10.1146/annurev-clinpsy-050212-185542>.
- Wagner, A. R. (1981). SOP: a model of automatic memory processing in animal behavior. In N. E. Spear, & R. R. Miller (Eds.), *Information processing in animals: Memory mechanisms*. Hillsdale, NJ: Erlbaum.
- Wolpe, J. (1958). *Psychotherapy by reciprocal inhibition*. Stanford: Stanford University Press.