Mechanisms of Fear Reduction

CYNTHIA L. LANCASTER and MARIE-H. MONFILS

Abstract

Over the past century, numerous theories have been advanced toward a unified account of fear reduction and have achieved various degrees of empirical support. Here, we first provide a brief overview of the basic models that account for fear acquisition, then we provide a review of several of the most prominent theories of fear reduction, and finally, we describe important cutting-edge directions for future research.

Although it is clear that fear reduction occurs across a broad range of biological organisms (Harris, 1943), there is no single, widely accepted theory for the mechanism of fear reduction. Over the past century, numerous theories have been advanced and have achieved various degrees of empirical support. Here, we first provide a brief overview of the basic models that account for fear acquisition, then we provide a review of several of the most prominent theories of fear reduction, and finally, we describe important cutting-edge directions for future research.

We have divided the theories into three broad categories: procedural models, cognitive models, and neurobiological models. Procedural models, as defined here, focus primarily on which particular procedures are seen as most critical for achieving fear reduction. This perspective aligns well with early work in behaviorism, a movement that focused on observable procedures and responses. Cognitive models, as defined here, propose that changes in conscious cognition play a key role in fear reduction. Note that cognitive models imply the importance of cognitive change as a *mechanism*, though cognitive change might be facilitated by behavioral methods (cf., Bandura, 1977). The perspective of cognitive models aligns closely with the cognitive movement and more recent research in psychotherapy. Neurobiological models provide theories on the specific mechanisms of neural plasticity and the changes of biological systems that are associated with fear reduction. Behavioral neuroscience and nonhuman animal models of

Emerging Trends in the Social and Behavioral Sciences. Edited by Robert Scott and Stephen Kosslyn.
 ISBN 978-1-118-90077-2.

fear reduction have provided the foundation for the development of the neurobiological models.

> "Behavior therapists have had more success in developing clinical treatments for phobia than in explaining how fear is learned and unlearned."
>
> —*Reiss (1980, p. 380)*

FEAR ACQUISITION

Pavlov's (1927) model of classical conditioning is one of the most important accounts of fear acquisition. According to this model, fear can be acquired through pairing an initially neutral stimulus (conditioned stimulus (CS)) with another inherently aversive stimulus (unconditioned stimulus (US)). For example, in the famous little Albert experiments, young Albert learned to fear a white rat though classical conditioning (Watson & Rayner, 1920). At first, Albert showed signs of interest in the rat, but after being repeatedly presented with a loud noise, the presence of the rat alone elicited distress. Albert had thus learned to fear the rat, and this fear response generalized to similar stimuli. Since the little Albert experiments, extensive data have been gathered through additional studies of Pavlovian conditioning, and have provided insight into the brain mechanisms of fear acquisition as well as avenues to reduce the intensity of fear memories. Rodents, such as rats and mice, are popular choices for studying fear in nonhuman animals because they are relatively easy to house and care for and have stereotyped responses that can be easily quantified. Rodent fear can be measured in a variety of ways, but one of the most popular methods is measuring freezing behavior, a stereotypical behavioral response to fear in a number of species.

In addition to classical conditioning, many animal models of learning use paradigms involving indirect measures of fear, such as conditioned suppression or avoidance behavior. Owing to the focus on fear reduction mechanisms in this essay, these paradigms will not be reviewed in further detail. Research has also clearly demonstrated that fear can be acquired through mechanisms other than classical conditioning, such as vicarious learning (Askew & Field, 2008). Furthermore, as described by Mowrer's (1960) two-factor theory, after the fear is acquired through classical conditioning, it can be maintained by operant conditioning or, specifically, learned avoidance. When the feared stimulus is confronted and the escape response follows, escape is negatively reinforced by removal of the feared stimulus and fear response. The frequency of the avoidance response then reduces the opportunity for confrontation with the feared stimulus, a procedure that is central to fear reduction (see the section titled "Procedural Models" for further details).

Appropriate fear responses are essential to survival, but this adaptive mechanism sometimes goes awry, and the associated memories are extremely persistent. As such, reducing fear responses is often a difficult task both in the laboratory and in clinical settings. Many aspects of animal models of fear conditioning resemble pathological fear and anxiety conditions seen in humans (Rosen & Schulkin, 1998; Wolpe, 1981; for review, see Delgado, Olsson, & Phelps, 2006), so optimizing fear reduction in these models is critical to our effort to understand and treat fear conditions in the clinic.

FOUNDATIONAL THEORIES OF FEAR REDUCTION

Procedural Models

Extinction. One of the most established approaches to reduce fear is through extinction trials, as the procedure is called in the animal literature, or through exposure therapy, as the procedure is called in the psychotherapy literature. During extinction trials, repeated presentations of the CS in the absence of the US lead to a progressive decrease in expression of fear of the stimulus (Bouton & Bolles, 1979; Pavlov, 1927; Rescorla & Heth, 1975). For example, a fear response to a tone, conditioned through repeated pairing with an electrical shock, can be extinguished by repeatedly presenting the tone without the shock.

Unfortunately, as initially suggested by Pavlov, extinction procedures do not seem to modify the original fear memory trace. Instead, extinction trials produce a new, separate, inhibitory memory that is stored in parallel to the original fear memory (Bouton, 2002; Pavlov, 1927). This is evident at the behavioral level when the fear response returns through the phenomena of spontaneous recovery, reinstatement, and renewal. Spontaneous recovery is brought about by the passage of time; reinstatement is return of fear following a stressful and triggering event; and renewal is the return of fear in contexts other than the extinction context. Research on neural mechanisms underlying extinction also suggests that the extinction memory is processed differently than the original fear memory and serves to inhibit responding to the original memory (for review, see Quirk, Garcia, & Gonzalex-Lima, 2006).

Reciprocal Inhibition and Counterconditioning. Wolpe (1954) proposed that fear reduction observed during various psychotherapies could be explained by the mechanism of reciprocal inhibition. Reciprocal inhibition suggests that fear reduces when the presentation of a fear-provoking stimulus is paired with a response that is physiologically incompatible with fear, such as relaxation induced by a procedure such as progressive muscle relaxation (Wolpe, 1961). This view was inspired by the law of reciprocal innervation (Ciuffreda & Stark, 1975), which states that when one muscle is activated,

its opposite muscle relaxes automatically to allow for smooth flexing of the limb. Wolpe (1968) noted that each pairing of the competing response with the target stimulus would progressively weaken the relationship between the stimulus and the fear response, resulting in the conditioned inhibition of fear. This theory led to Wolpe's development of systematic desensitization, which consists of graduated, imaginal exposure to a feared stimulus while maintaining a relaxed state. Wolpe (1968) used the term counterconditioning interchangeably with reciprocal inhibition. However, others (e.g., Davison, 1968) suggest that counterconditioning is different because it does not assume an underlying mechanism of physiological incompatibility; instead, it simply states that through conditioning, the feared stimulus is gradually paired with a new emotional state (e.g., pleasure, if the feared stimulus is presented consistently with an enjoyable food; Jones, 1924).

Both of these models fell out of favor when evidence demonstrated that procedures such as flooding and implosive therapy also led to fear reduction (e.g., Boulougouris, Marks & Marset, 1971; Emmelkamp, 1974; Keane, Fairbank, Caddell, & Zimering, 1989). In contrast to systematic desensitization, flooding and implosive therapy are not graduated and do not involve the practice of relaxation during exposure to the feared stimulus. Therefore, the reciprocal inhibition and counterconditioning models cannot explain the fear reduction observed after flooding and implosive therapy. Furthermore, the mechanism of reciprocal inhibition cannot account for fear extinction observed in animal models.

Habituation. It is important to note that in the literature at large, habituation is sometimes referred to as a mechanism, and in other cases it is referred to as an outcome—simply the observed reduction in fear response over. We will focus our critique on habituation as a mechanism, defined by Harris (1943) as "response decrement due to repeated stimulation" (p. 387). This theory posits that repeated stimulation (i.e., repeated presentation of a feared stimulus) is the central procedure necessary for fear reduction. Similar to reciprocal inhibition, this model was inspired by analogy to a physiological phenomenon. Specifically, fear reduction is seen as analogous to the refractory period in the nerve–muscle response, in which the nerve or the muscle is incapable of response as a result of recent stimulation. The habituation model suggests that the fear response cannot be maintained indefinitely, so after a prolonged fear response during confrontation with the feared stimulus, the body will eventually fatigue and the fear response will temporarily reduce as a result. Although habituation provides an explanation for the recovery of fear that can occur after the passage of time (Lader and Mathews, 1968), it cannot explain the reduction in fear that can last for years after the completion of

exposure therapy (e.g., Fava *et al.*, 2001). In addition, using the habituation model to explain long-term fear reduction directly contradicts the finding that exposure treatment terminated after 50% reduction in fear is not less effective than treatment terminated after 100% reduction in fear (Rachman, Robinson, & Lopatka, 1987).

Cognitive Models

Self-Efficacy. Bandura (1977, 1983) proposed that a key mechanism of fear reduction (and other psychological changes) across various psychological treatments involves improvement in self-efficacy, defined as "the conviction that one can successfully execute the behavior required to produce the [desired] outcomes" (1977, p. 193). Bandura posits that change in self-efficacy can occur via performance accomplishments (e.g., exposure therapy), vicarious experience (e.g., witnessing another person confront the feared stimulus successfully), verbal persuasion (e.g., being told that one has the skill set to achieve the desired outcome), or emotional arousal (e.g., noticing that one is not physiologically aroused by a previously feared situation; Bandura, 1977). As one example of data that corroborate this theory, higher self-efficacy during confrontation with a feared stimulus predicts greater approach, and lower subjective and physiological fear response (e.g., Bandura, Reese & Adams, 1982). Self-efficacy has also been extended beyond the beliefs about the ability to cope behaviorally, to beliefs about the ability to cope with thoughts and feelings associated with fear while in the presence of the feared stimulus (Valentiner, Telch, Petruzzi & Bolte, 1996). Bandura (1997) provides a thorough review of the model and the supporting data for self-efficacy theory.

Expectancy Theory and Prediction Error Theory. In his description of expectancy theory, Reiss (1980) describes that the fear response is an algebraic sum of danger expectancy (e.g., "the spider will probably bite me") and the product of anxiety expectancy (e.g., "being in a room with a spider will probably make me feel anxious") with anxiety sensitivity (e.g., "the feeling of being anxious, itself, makes me anxious"). Note that anxiety expectancy will have no impact on an individual's fear response if the individual does not find the experience of anxiety to be threatening. On the basis of this model, the fear response should decrease under two conditions: (i) when the danger expectancy decreases (e.g., someone expects the spider to bite them, and it does not), and (ii) when the anxiety expectancy decreases if some level of anxiety sensitivity is present (e.g., one finds the experience of anxiety to be aversive, and is not as nervous as expected while

in the same room as a spider). Reiss (1991) later added other components to the expectancy model, such as social evaluation expectancy (e.g., "they will laugh at me when they see that I am anxious") and social evaluation sensitivity (e.g., "I will feel very embarrassed when they laugh at me").

Expectancy theory is directly in line with the prediction error model initially proposed by Rescorla and Wagner (1972). The prediction error model states that when the disparity between expectations and the occurrence of events is greater, more learning will occur. For example, when a CS is presented without the US during extinction training, expectation is violated and learning occurs. The prediction error theory would suggest that the most learning occurs early in an extinction session, as the mismatch between what is expected and what actually occurs attenuates across extinction trials.

Emotional Processing Theory. Emotional processing theory, as applied specifically to fear reduction by Foa and Kozak (1986), was based on the foundation of Lang's bioinformational theory (1977) and on Rachman's (1980) proposal of the emotional processing construct. In their explanation of emotional processing theory, Foa and Kozak acknowledge that some components of fear memory are not consciously accessible. However, this theory clearly recognizes the role of conscious cognitive processes as mechanisms of change, and is closely associated with psychotherapy research, and so has been grouped with the cognitive models.

Foa and Kozak (1986) defined emotional processing as, "the modification of memory structures that underlie emotions" (p. 20). Borrowing from the bioinformational account of fear, they stated that fear structures involve three basic representations: (i) characteristics of the feared stimulus; (ii) response to the feared stimulus (including verbal, physiological, and behavioral responses); and (iii) interpretive meaning of both the stimulus and the responses. They describe two key elements of a treatment that modifies the pathological fear structure: (i) initial activation of the fear structure during treatment, and (ii) confrontation with, and incorporation of, information that is incompatible with the fear structure. Finally, they proposed three signs that emotional processing is occurring (i) initial fear activation, (ii) within-session habituation, and (iii) between-session habituation.

Foa, Huppert, and Cahill (2006) provided an update of emotional processing theory that incorporates recent research findings. In line with the evidence for inhibitory learning (see the section titled "New Inhibitory Learning" for a review), they described that modifying the fear structure may involve the addition of new learning that completes with old learning, rather than the elimination or replacement of previous associations in the fear structure. Furthermore, they recognized that research to date does

not provide strong support for the originally posited association between within-session habituation and emotional processing. They accounted for this finding by explaining that the critical mechanism of emotional processing is encountering disconfirming information. Therefore, within-session habituation should only be important for individuals who hold the belief that their anxiety will continue indefinitely until they escape the feared situation.

Emotional processing theory has been a heavily influential theory, in part, because of its integrative nature. For example, the habituation, expectancy, and prediction error models can all be incorporated into emotional processing theory, under the central concept of mechanisms by which disconfirming information is encountered and/or incorporated. Despite its strengths, emotional processing theory has several areas for continued growth. A review of the literature conducted by Craske *et al*. (2008) found that within-session habituation is not indicative of emotional processing, and that initial fear activation correlates with emotional processing in some studies but not others. Foa, Huppert, and Cahill (2006) interpreted this type of evidence to suggest that proposed correlates of emotional processing (e.g., within session habituation) may not provide reliable evidence of the emotional processing. As evidence contrary to the proposed theory begins to emerge, clearly outlining central components of the theory that are falsifiable will be crucial to the continued development of innovative and integrative theories such as emotional processing.

Neurobiological Models

New Inhibitory Learning. New inhibitory learning was proposed as a mechanism of fear reduction to explain the observation that extinguished fear can later reemerge through processes such as spontaneous recovery, renewal, and reinstatement. Essentially, extinction training is thought to produce a new memory (of the feared stimulus as being safe), which acts to suppress the fear response (Bouton & Swartzentruber, 1991). The original fear association remains intact and can reemerge whenever the inhibitory memory fails to activate. It is theorized that the new inhibitory memory is context specific, so fear is more likely to emerge in contexts dissimilar to those of extinction training. For example, if a patient successfully extinguished fear of tarantulas during exposure therapy in their therapist's office, the patient might experience a return of fear when encountering tarantulas outside the office because the inhibitory memory might fail to activate. The persistence of the original association in combination with a new inhibitory memory explains why extinguished fear might later reemerge. Today, this model of fear reduction is widely accepted by researchers who work with fear models in humans and other animals (Bouton, 2002; Craske *et al.*, 2008; Foa, Huppert, & Cahill, 2006).

Fear Memory Unlearning/Erasure. In the context of psychotherapy, exposure therapy (also called extinction training) is one of the most empirically supported methods to reduce fear. However, as explained earlier, inhibitory learning produced by extinction training is not equivalent to unlearning or erasure of fear, so it is always possible that the fear response may reemerge (Bouton, 2002). Other lines of research suggest that certain time windows leave memories more susceptible to persistent (possibly permanent) disruption. For example, pharmacological agents applied shortly after learning can prevent consolidation (i.e., strengthening/solidifying) of a fear memory. Another such window of opportunity is during reconsolidation.

Reconsolidation is a mechanism by which a memory becomes susceptible for disruption or updating every time it is retrieved. When memory reactivation experiments were in their infancy, the most prominent methods of inducing amnesia after a reactivation for previously learned fear events involved administering electroconvulsive shock (ECS) or hypothermia after a reactivation of the memory (Mactutus, Riccio, & Ferek, 1979; Misanin, Miller, & Lewis, 1968).

In 2000, Nader and colleagues performed an important study in which they convincingly demonstrated that when a memory is retrieved, it enters a reconsolidation period during which it requires new protein synthesis before becoming re-encoded into long-term storage. When protein synthesis is prevented through the infusion of a pharmacological agent, the freezing response to the CS was drastically reduced the next day. Nader, Schafe and Le Doux (2000) also showed that the amnesic effect is sensitive to the time between reactivation and drug injection; 6 hours after retrieval, the memory is no longer susceptible to pharmacological blockade. This study reinvigorated a field that had been somewhat dormant for a number of years. Since Nader *et al.*'s publication, the number of studies testing reconsolidation blockade of fear memory has grown exponentially, and researchers have tested a variety of potential pharmacological agents for this use (e.g., Duvarci & Nader, 2004). Unlike inhibitory learning, reconsolidation blockade does not seem to be susceptible to the return of fear in studies using a number of pharmacological agents.

It is important to add that the strength or age of a memory can influence whether a reconsolidation-based paradigm can effectively lead to fear reduction (for a review, see Alberini, 2005). Furthermore, although many drugs are effective in reducing fear to a simple CS during reconsolidation in rodent studies, those same drugs are often toxic to humans (propranolol being an exception; Pitman *et al.*, 2002). Therefore, pharmacological reconsolidation blockade provides an excellent avenue for understanding the mechanisms of fear memory reconsolidation and for developing animal models of fear reduction; however, the ability to translate this into a clinical setting remains

questionable and not without risk (see the section titled "Cutting-Edge Paradigms").

COMMENTS ON REVIEWED MODELS

Procedural Models for Fear Reduction Are Insufficient

Procedural models for fear reduction are insufficient, because each procedure can be described on the level of neurobiological modeling, and conscious cognitive mechanisms also play an important role. Conscious cognitive processes probably include processes that are initiated by the cortex, which may sometimes override more automatic processes, or work in conjunction with unconscious/automatic processes. However, procedural models provide an excellent foundation for the investigation of fear reduction mechanisms because they outline the situations in which changes in the memory trace are most likely to occur. Procedural models can be viewed as the context in which both unconscious and conscious pathways of fear reduction operate.

We Assume There Are Neurobiological Mechanisms for the Cognitive Models Described

In a sense, cognitive models could also be deemed insufficient because we assume that changes in cognitive processes are mediated by neurobiological mechanisms, as with any psychological process. We predict that we will understand more about the neurobiological models that underlie the changes in cognitive processes over time, particularly as we develop more sophisticated, noninvasive tools to study neurobiological mechanisms. However, the neurobiological models involved in updating of cognitive processes are probably so complex and individuated that they are unlikely to replace cognitive models in a reductionistic manner. It may be more effective to conceptualize each of these models as representing different levels of understanding or different levels of detail (e.g., understanding change in memory structures on a macro vs micro level).

CUTTING-EDGE PARADIGMS

With extinction providing a practical but not necessarily permanently effective method to reduce fear, and reconsolidation blockade providing an effective but somewhat impractical method for targeting the original fear memory (owing to the fact that many reconsolidation blocking drugs cannot be used in humans), a more effective behavioral technique is much desired.

Monfils, Cowansage, Klann, and LeDoux (2009) addressed this issue by combining the strengths of both reconsolidation and extinction methods into a behavioral paradigm to permanently reduce fear. In this paradigm, they found that using extinction training during the reconsolidation window allows for a persistent updating of the memory and prevents the return of fear. Similar to previous experiments that attempted to reduce fear after a reactivation session, Monfils *et al*. (2009) found that the effectiveness of the retrieval+extinction paradigm was restricted to a specific temporal range between the retrieval and the extinction session. A number of additional studies have since been published, most of which have replicated the retrieval+extinction effect described by Monfils *et al*. (e.g., Clem & Huganir, 2010; Rao-Ruiz *et al.*, 2011; but see Chan, Leung, Westbrook, & McNally, 2010). It should also be noted that extinction applied shortly after fear conditioning (before consolidation) has also shown success in preventing the return of fear (Myers, Ressler, & Davis, 2006; see Chang & Maren, 2009).

The retrieval+extinction technique allows for a behavioral paradigm to permanently attenuate fear expression to a conditioned CS in a manner that does not involve drugs or surgery by slightly modifying the timing between the first and the second presentation of the CS (e.g., conducting the retrieval trial, which is the first CS presentation, between 10 min and 1 h prior to extinction training). This method has also been successful in extinguishing shock-conditioned fear in humans (Schiller *et al.*, 2010). Owing to the fact that the retrieval+extinction might prevent the return of fear using a protocol that is methodologically similar to exposure therapy, there is significant enthusiasm from a number of clinicians in translating the initial findings into therapeutic avenues (see also Graff *et al.*, 2014—an important recent study that combines the retrieval+extinction approach with a pharmacological manipulation to successfully target older memories in rodents).

CONCLUSIONS AND FUTURE DIRECTIONS

Although some of the tools we have at our disposal are effective in decreasing fear responding, it is clear that they do not work in all cases, and even for those in which they work, return of fear is often a concern. Moving forward, we need to better identify the parameters in which various mechanisms operate. For example, under what conditions are inhibitory learning, reconsolidation update, or both operating as the mechanisms of fear reduction?

We should also seek to understand the influence of individual differences on the mechanisms of fear reduction. This returns to the much-repeated question, "What treatment works best for whom, and why?" but adds an emphasis on the possible biomarkers and/or *a priori* neural states that could

predispose an individual to responding optimally to one approach rather than another.

Ultimately, to effectively agree on a theory of what models and/or mechanisms best underlie fear reduction, we will also need to agree on an acceptable determinant for the said reduction. One determinant, and arguably the one that ultimately matters most, is subjective decrease in fear. Still, while evidence suggests that more than one type of approach may be successful in reducing fear, it is clear that early success of treatment (either within the treatment session or shortly after treatment) does not always reflect the long-term outcome. As such, developing additional means (e.g., physiological, behavioral, neural, or otherwise) of identifying long-term predictors of fear reduction will be crucial.

REFERENCES

Alberini, C. M. (2005). Mechanisms of memory stabilization: Are consolidation and reconsolidation similar or distinct processes? *Trends in Neuroscience*, *28*, 51–56.

Askew, C., & Field, A. P. (2008). The vicarious learning pathway to fear 40 years on. *Clinical Psychology Review*, *28*(7), 1249–1265.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, *84*(2), 191–215.

Bandura, A. (1983). Self-efficacy determinants of anticipated fears and calamities. *Journal of Personality and Social Psychology*, *45*(2), 464–469.

Bandura, A. (1997). *Self-efficacy: The exercise of control*. New York: Freeman.

Bandura, A., Reese, L., & Adams, N. E. (1982). Microanalysis of action and fear arousal as a function of differential levels of perceived self-efficacy. *Journal of Personality and Social Psychology*, *43*(1), 5–21.

Blanchard, R. J., & Blanchard, D. C. (1971). Defensive reactions in the albino rat. *Learning and Motivation*, *2*, 351–362.

Boulougouris, J. C., Marks, I. M., & Marset, P. (1971). Superiority of flooding (implosion) to desensitisation for reducing pathological fear. *Behaviour Research and Therapy*, *9*(1), 7–16.

Bouton, M. E. (2002). Context, ambiguity, and unlearning: Sources of relapse after behavioral extinction. *Biological Psychiatry*, *52*(10), 976–986.

Bouton, M. E., & Bolles, R. C. (1979). Contextual control of the extinction of conditioned fear. *Learning and Motivation*, *10*, 445–466.

Bouton, M. E., & Swartzentruber, D. (1991). Sources of relapse after extinction in Pavlovian and instrumental learning. *Clinical Psychology Review*, *11*(2), 123–140.

Chan, W. Y. M., Leung, H. T., Westbrook, F., & McNally, G. P. (2010). Effects of recent exposure to a conditioned stimulus on extinction of Pavlovian fear conditioning. *Learning and Memory*, *17*(10), 512–521.

Chang, C. H., & Maren, S. (2009). Early extinction after fear conditioning yields a context-independent and short-term suppression of conditional freezing in rats. *Learning & Memory*, *16*(1), 62–68.

Ciuffreda, K. J., & Stark, L. (1975). Descartes' law of reciprocal innervation. *American Journal of Optometry and Physiological Optics*, *52*(10), 663–673.

Clem, R. L., & Huganir, R. L. (2010). Calcium-permeable AMPA receptor dynamics mediate fear memory erasure. *Science*, *330*(6007), 1108–1112.

Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, *46*(1), 5–27.

Davison, G. C. (1968). A procedural critique of "Desensitization and the Experimental Reduction of Threat". *Journal of Abnormal Psychology*, *74*(1), 86–87.

Dawson, M. E., & Furedy, J. J. (1976). The role of awareness in human differential autonomic classical conditioning: The necessary-gate hypothesis. *Psychophysiology*, *13*(1), 50–53.

Delgado, M. R., Olsson, A., & Phelps, E. A. (2006). Extending animal models of fear conditioning to humans. *Biological Psychology*, *73*, 39–48.

Duvarci, S., & Nader, K. (2004). Characterization of fear memory reconsolidation. *The Journal of Neuroscience*, *24*, 9269–9275.

Emmelkamp, P. M. (1974). Self-observation versus flooding in the treatment of agoraphobia. *Behaviour Research and Therapy*, *12*(3), 229–237.

Fava, G. A., Grandi, S., Rafanelli, C., Ruini, C., Conti, S., & Belluardo, P. (2001). Long-term outcome of social phobia treated by exposure. *Psychological Medicine*, *31*(5), 899–905.

Foa, E. B., Huppert, J. D., & Cahill, S. P. (2006). Emotional processing theory: An update. In B. O. Rothbaum (Ed.), *Pathological anxiety: Emotional processing in etiology and treatment* (pp. 3–24). New York, NY: Guilford.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, *99*(1), 20.

Gräff J., Joseph N. F., Horn M. E., Samiei A., Meng J., Seo J., … Tsai L. H. (2014). Epigenetic priming of memory updating during reconsolidation to attenuate remote fear memories. *Cell*, *156*(1–2):261–76.

Harris, J. D. (1943). Habituatory response decrement in the intact organism. *Psychological Bulletin*, *40*(6), 385–422.

Jones, M. C. (1924). A laboratory study of fear: The case of Peter. In *The pedagogical seminary* (pp. 308–315. Retrieved from http://psychclassics.yorku.ca/Jones/)

Keane, T. M., Fairbank, J. A., Caddell, J. M., & Zimering, R. T. (1989). Implosive (flooding) therapy reduces symptoms of PTSD in Vietnam combat veterans. *Behavior Therapy*, *20*(2), 245–260.

Lader, M. H., & Mathews, A. M. (1968). A physiological model of phobic anxiety and desensitization. *Behaviour Research and Therapy*, *6*(4), 411–421.

Mactutus, C. F., Riccio, D. C., & Ferek, J. M. (1979). Retrograde amnesia for old (reactivated) memory: Some anomalous characteristics. *Science*, *204*, 1319–1320.

Misanin, J. R., Miller, R. R., & Lewis, D. J. (1968). Retrograde amnesia produced by electroconvulsive shock after reactivation of a consolidated memory trace. *Science*, *160*, 203–204.

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.

Mowrer, O. H. (1960). *Learning theory and behavior*. Hoboken, NJ: John Wiley & Sons, Inc.

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

Nader, K., Schafe, G. E., & Le Doux, J. E. (2000). Fear memories require protein synthesis in the amygdala for reconsolidation after retrieval. *Nature*, *406*(6797), 722–726.

Pavlov, I. P. (1927). *Conditioned reflexes: An investigation of the physiological activity of the cerebral cortex* (G. V. Anrep, Trans.; Original work published 1927). Retrieved from: http://psychclassics.yorku.ca/Pavlov/

Pitman, R. K., Sanders, K. M., Zusman, R. M., Healy, A. R., Cheema, F., Lasko, N. B., … & Orr, S. P. (2002). Pilot study of secondary prevention of posttraumatic stress disorder with propranolol. *Biological Psychiatry*, *51*(2), 189–192.

Quirk, G. J., Garcia, R., & Gonzalez-Lima, F. (2006). Prefrontal mechanisms in extinction of conditioned fear. *Biological Psychiatry*, *60*, 337–343.

Rachman, S. (1980). Emotional processing. *Behaviour Research and Therapy*, *18*, 51–60.

Rachman, S., Robinson, S., & Lopatka, C. (1987). Is incomplete fear-reduction followed by a return of fear? *Behaviour Research and Therapy*, *25*(1), 67–69.

Rao-Ruiz, P., Rotaru, D. C., van der Loo, R. J., Mansvelder, H. D., Stiedl, O., Smit, A. B., & Spijker, S. (2011). Retrieval-specific endocytosis of GluA2-AMPARs underlies adaptive reconsolidation of contextual fear. *Nature Neuroscience*, *14*, 1302–1308.

Reiss, S. (1980). Pavlovian conditioning and human fear: An expectancy model. *Behavior Therapy*, *11*(3), 380–396.

Reiss, S. (1991). Expectancy model of fear, anxiety, and panic. *Clinical Psychology Review*, *11*(2), 141–153.

Rescorla, R. A., & Heth, C. D. (1975). Reinstatement of fear to an extinguished conditioned stimulus. *Journal of Experimental Psychology: Animal Behavior Processes*, *104*(1), 88–96.

Rescorla, R. A., & Wagner, A. R. (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In A. H. Black & W. F. Prokasy (Eds.), *Classical Conditioning II: Current Research and Theory* (pp. 64–99). New York: Appleton-Century-Crofts.

Rosen, J. B., & Schulkin, J. (1998). From normal fear to pathological anxiety. *Psychological Review*, *105*(2), 325–350.

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.

Valentiner, D. P., Telch, M. J., Petruzzi, D. C., & Bolte, M. C. (1996). Cognitive mechanisms in claustrophobia: an examination of Reiss and McNally's expectancy model and Bandura's self-efficacy theory. *Cognitive Therapy and Research*, *20*(6), 593–612.

Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology*, *3*(1), 1–14.
Wolpe, J. (1954). Reciprocal inhibition as the main basis of psychotherapeutic effects. *Archives of Neurology and Psychiatry*, *72*(2), 205.
Wolpe, J. (1961). The systematic desensitization treatment of neuroses. *The Journal of Nervous and Mental Disease*, *132*(3), 189–203.
Wolpe, J. (1968). Psychotherapy by reciprocal inhibition. *Conditional Reflex: A Pavlovian Journal of Research & Therapy*, *3*(4), 234–240.
Wolpe, J. (1981). The dichotomy between classical conditioned and cognitively learned anxiety. *Journal of Behavior Therapy and Experimental Psychiatry*, *12*(1), 35–42.

CYNTHIA L. LANCASTER SHORT BIOGRAPHY

Cynthia L. Lancaster received her bachelor's degree from Trinity University, where she completed a study on the topic of mirror exposure for body image dissatisfaction. She then worked as a research assistant at the University of Texas Health Science Center at San Antonio, primarily studying the etiology and treatment of posttraumatic stress disorder in active duty military personnel. She is currently in the clinical psychology doctoral program at the University of Texas at Austin, where she studies the mechanisms and augmentation of exposure therapy for anxiety disorders.

MARIE-H. MONFILS SHORT BIOGRAPHY

Marie-H. Monfils is an assistant professor in the Department of Psychology at the University of Texas at Austin. She received her PhD in behavioral neuroscience from the Canadian Centre for Behavioural Neuroscience, under the supervision of Bryan Kolb and Jeffrey Kleim, and her master's degree from the University of Calgary with Cam Teskey. She conducted a postdoctoral fellowship at New York University in Joseph LeDoux's laboratory. Her laboratory is currently pursuing three research streams: (i) investigating post-consolidation manipulations that can persistently attenuate fear memories, (ii) isolating the mechanisms that underlie social transmission of fear, and (iii) examining the interactions between early experience and fear learning in adulthood. More details about the Monfils Fear Memory Lab may be found at http://homepage.psy.utexas.edu/HomePage/Group/MonfilsLAB/Site/Monfils_Lab.html.

RELATED ESSAYS

What Is Neuroticism, and Can We Treat It? *(Psychology)*, Amantia Ametaj *et al*.
Aggression and Victimization *(Psychology)*, Sheri Bauman and Aryn Taylor

Delusions *(Psychology)*, Max Coltheart
Depression *(Psychology)*, Ian H. Gotlib and Daniella J. Furman
Positive Emotion Disturbance *(Psychology)*, June Gruber and John Purcell
Mental Imagery in Psychological Disorders *(Psychology)*, Emily A. Holmes *et al*.
Normal Negative Emotions and Mental Disorders *(Sociology)*, Allan V. Horwitz
Dissociation and Dissociative Identity Disorder (DID) *(Psychology)*, Rafaële J. C. Huntjens and Martin J. Dorahy
Computer Technology and Children's Mental Health *(Psychology)*, Philip C. Kendall *et al*.
Understanding Risk-Taking Behavior: Insights from Evolutionary Psychology *(Psychology)*, Karin Machluf and David F. Bjorklund
Evolutionary Perspectives on Animal and Human Personality *(Anthropology)*, Joseph H. Manson and Lynn A. Fairbanks
Disorders of Consciousness *(Psychology)*, Martin M. Monti
Cognitive Remediation in Schizophrenia *(Psychology)*, Clare Reeder and Til Wykes
Cognitive Bias Modification in Mental *(Psychology)*, Meg M. Reuland *et al*.
Clarifying the Nature and Structure of Personality Disorder *(Psychology)*, Takakuni Suzuki and Douglas B. Samuel
Rumination *(Psychology)*, Edward R. Watkins
Emotion Regulation *(Psychology)*, Paree Zarolia *et al*.