



The effect of positive mood induction on reducing reinstatement fear: Relevance for long term outcomes of exposure therapy



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ABSTRACT

While exposure therapy is effective in treating anxiety, fear can return after exposure. Return of fear can be understood through mechanisms of extinction learning. One form of return of fear is reinstatement, or, the fear that results from an unsignaled unconditional stimulus (US) presentation after extinction. Though the conditional response (CR; e.g., fear) typically reduces during extinction, the excitatory conditional stimulus (CS+) valence remains negative. The more negative the CS+ valence after the end of extinction, the greater the fear at reinstatement. The current study evaluated the degree to which positive mood induction (positive imagery training; PIT) compared to control (positive verbal training; PVT) before extinction a) decreased CS+ negative valence during extinction and b) reduced reinstatement fear. Compared to PVT, PIT a) increased positive affect, b) decreased post-extinction CS+ negative valence, and c) reduced reinstatement responding as measured by eye blink startle reflex (when shock was used at reinstatement) and self-report fear (regardless of reinstatement US type). Results suggest that increasing positive affect prior to exposure therapy could reduce relapse through reinstatement.

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1. Introduction

Exposure therapy is well-established as an effective therapeutic strategy for anxiety disorders (Hofmann & Smits, 2008; In-Albon & Schneider, 2007). However, a number of individuals experience a return of fear following successful conclusion of treatment (Craske & Mystkowski, 2006; Rachman, 1989). Thus, there is a need to understand the mechanisms responsible for return of fear and to develop interventions that reduce its occurrence and enhance long-term treatment gains. In the model of exposure therapy, return of fear is understood as reactivation of conditional threat associations that compete with the non-threat-based associations developed through extinction (Hermans, Craske, Mineka, & Lovibond, 2006). The purpose of the current study is to evaluate one possible method (i.e., positive mood induction before extinction) of reducing return of fear following extinction.

Models of extinction emphasize inhibitory learning mechanisms (Bouton, 1993; Wagner, 1981), although additional

mechanisms, such as habituation, may also be involved (Myers & Davis, 2007). Within a classical conditioning approach, the original conditional stimulus (CS)/unconditional stimulus (US) association learned during acquisition of threat responding¹ is not erased during extinction, but rather is left intact while a new, secondary CS+/NoUS inhibitory association develops (e.g., Bouton, 1993; Bouton & King, 1983). This means that individuals have two memories of the CS+: one in which it predicts an aversive event and a separate memory in which it predicts no aversive event. The relative strength between these two memories determines how much threat responding occurs. In these studies, a CS+ is associated with the occurrence of the US, whereas a CS− is associated with the absence of the US. The inhibitory association is dependent on both the CS+ and the context in which the CS+ is presented, whereas the initial excitatory association is independent of context (Bouton, 2004). Since the original excitatory meaning (CS+/US) is not

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¹ Following premises set forth by LeDoux (2014), we will use the term “threat responding” to encompass both the conscious, emotional experience of being afraid and the unconscious, biological responses. The term “fear” conditioning” will be replaced by “threat conditioning.” The term “fear” will henceforth refer to the conscious emotion of being afraid.

erased by extinction, it can be retrieved following extinction, as evidenced by increased conditional threat responding. In the context of exposure, the retrieval of the excitatory CS+/US association translates to a return of fear and relapse (Vervliet, Hermans, & Craske, 2013).

Several phenomena demonstrate retention of the original excitatory CS+/US association. These include *spontaneous recovery* (Quirk, 2002), which is observed clinically as increasing threat responding with increasing intervals of time since the end of exposure therapy and the next time the phobic stimulus is encountered. For example, an individual who completes treatment for phobia of public speaking will likely have greater threat responding when giving a public speech months after treatment compared to a public speech immediately after the last exposure session. Retention of the CS+/US association is also apparent in *renewal* of threat responding due to a change in context between extinction and extinction retest (Bouton, 1993). Contexts may be exteroceptive cues (e.g., a room, place, environment, or other external background stimuli; Bouton, 1993) and interoceptive cues, such as drug state (Bouton, Kenney, & Rosengard, 1990; Overton, 1985). The clinical translation of context renewal is exemplified by return of fear in a public speaking situation (e.g., a wedding) that differs from the public speaking practiced in exposure therapy (e.g., clinic rooms; Culver, Stoyanova, & Craske, 2011).

A third demonstration of CS+/US retention is *rapid reacquisition*, in which the CS+ and US are re-paired following extinction (Kehoe & Macrae, 1997). Clinically, an individual who undergoes therapy for a phobia of dogs may experience rapid reacquisition if attacked by a dog after completion of exposure therapy. Finally, unpaired US presentations (without the presence of the CS+) after extinction can lead to a *reinstatement* of threat responding (Rescorla & Heth, 1975). For example, an individual who is treated for specific phobia for dogs after being attacked by a dog may experience reinstatement of fear of dogs following being bitten by a snake. Reinstatement has been long established in animal studies and more recently in human conditioning studies (e.g., Dirikx, Hermans, Vansteenwegen, Baeyens, & Eelen, 2004, 2007; Hermans et al., 2005; LaBar & Phelps, 2005; Norrholm et al., 2006; Van Damme, Crombez, Hermans, Koster, & Eccleston, 2006; Zbozinek, Prenoveau, Liao, Hermans, & Craske, 2015). The current study addresses new ways to mitigate the effects of reinstatement.

There has been little investigation of the effects of a reinstating US that is different from the acquisition US. Yet, reinstatement by a US that differs from the acquisition US would offer a theoretical model for the occasions when clients experience a return of fear following exposure therapy due to an aversive event (e.g., car accident) that differs from the original acquisition event (e.g., social ridicule). In one animal study, a novel US at reinstatement (i.e., klaxon [loud horn]) that differed from the original US during acquisition (i.e., electric shock) reinstated conditional threat responses to the CS+ (Rescorla & Heth, 1975). In one human study, a reinstating US that was different from the acquisition US elicited as great an increase in skin conductance responding to the CS+ as reinstatement with the original US. However, US expectancy increased only for the reinstatement US, regardless of acquisition US (Sokol & Lovibond, 2012). These studies suggest that a reinstating US that differs from the original acquisition US can increase conditional threat responding without increasing expectancy of the acquisition US. The current study evaluated the role of US type at reinstatement (i.e., electric shock, loud scream sound).

Evaluation of CS+ valence in relation to phenomena such as spontaneous recovery has suffered methodological limitations (Dirikx et al., 2004, 2007; Hermans et al., 2005; Zbozinek et al., 2015) or is nonexistent in the case of rapid reacquisition and renewal. However, the more negatively the CS+ is valenced at the

end of extinction, the greater the threat responding after reinstatement (Dirikx et al., 2004, 2007; Hermans et al., 2005; Zbozinek et al., 2015). Hermans and colleagues (e.g., Dirikx, et al., 2004) utilized the network model of emotions (Lang, Bradley, & Cuthbert, 1990) to develop the valence-arousal model of reinstatement. In this model, emotions are located on a valence (positive, negative) \times arousal (high, low) orthogonal matrix, with fear being located in the negative valence and high arousal quadrant (Lang, Greenwald, Bradley, & Hamm, 1993). Extinction learning decreases arousal towards the CS+, as shown by attenuated skin conductance response (SCR; e.g., Bradley, Cuthbert, & Lang, 1990). However, even though CS+ valence may become less negative by the end of extinction, it typically remains more negative than before acquisition (Dirikx et al., 2004). The combination of increased arousal that is evoked by the arousing properties of the unpaired US and persistent negative valence of the CS+ is posited to lead to reinstatement of conditional fear responding (Dirikx et al., 2004; Dirikx et al., 2007).

The valence-arousal model of reinstatement raises the possibility that strategies designed to decrease post-extinction negative valence of the CS+ may reduce the effects of reinstatement. Positive mood induction increases positive valence towards a specific stimulus (Erez et al., 2002; Isen & Shalke, 1982). Furthermore, positive mood induction may activate additional neural pathways associated with enhancing extinction learning (e.g., ventromedial/medial prefrontal cortex and anterior cingulate cortex; Phan, Wager, Taylor, & Liberzon, 2002). We predict that positive mood induction may reduce reinstatement effects by decreasing negative valence towards the CS+. A number of methods have been shown to induce positive mood, such as watching positive films (e.g., Gross & Levenson, 1995) and positive imagery training (Holmes, Mathews, Dalgleish, & Mackintosh, 2006; Holmes, Mathews, Mackintosh, & Dalgleish, 2008; Pictet, Coughtrey, Mathews, & Holmes, 2011). We chose positive imagery training given the consistency with which it induces positive mood compared to a stringent comparison condition of positive verbal training (Holmes et al., 2006; Holmes, Lang, & Shah, 2009; Mathews, Ridgeway, & Holmes, 2013; Nelis, Vanbrabant, Holmes, & Raes, 2012).

We hypothesized that positive imagery training would increase positive affect relative to a control condition involving positive verbal training, consistent with prior research (e.g., Holmes, et al., 2006). Second, given that induction of positive mood has been shown to influence valence appraisals of specific stimuli (Erez et al., 2002; Isen & Shalke, 1982), we hypothesized that positive imagery training would decrease CS+ negative valence by the end of extinction training relative to positive verbal training. Third, we hypothesized that positive imagery training would decrease the effects of reinstatement compared to positive verbal training. Furthermore, we evaluated a reinstating US that was the same as the acquisition US (i.e., electric shock) versus different from the acquisition US (i.e., loud scream). We also tested the effects of positive imagery training relative to positive verbal training on spontaneous recovery to test for specificity of effects to reinstatement.

2. Methods

2.1. Participants

Participants (N = 100) were students from the University of California, Los Angeles, who participated for either 3 course credits, \$25 cash, or a combination. Six participants dropped out partway through the study, leaving 94 completers. Participants were 67.3% female; mean age 20.39 (SD = 2.66) years; and 4.3% African-American, 40.4% Asian or Asian-American, 20.2% Caucasian, 22.3% Hispanic or Latino, 7.4% Asian or Asian American and Caucasian,

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