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The Effects of Attention Allocation on Fear Extinction

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by

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ABSTRACT OF THE DISSERTATION

The Effects of Attention Allocation on Fear Extinction

by

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Doctor of Philosophy in Psychology

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Professor Michelle G. Craske, Chair

The dissertation consists of one experimental study investigating the potential method for improving exposure therapy outcomes for anxiety disorders through the manipulation of attention allocation. If proven effective, these methods can be utilized to optimize exposure therapy for anxiety disorders or be considered as adjunct interventions intended to compliment and enhance exposure therapy.

Although the literature on fear conditioning and the literature on selective attention to threat have received wide support and have consequently implicated the treatment of anxiety disorders in their own right, there is very little integration and reconciliation of the two into a more parsimonious, yet comprehensive, theory of anxiety development, maintenance, and treatment. This study attempted to integrate both bodies of work by examining the effect of attention bias modification training upon Pavlovian fear extinction. This study tested the prediction that training attention towards the threat cue

(CS+) would facilitate fear extinction; whereas training attention towards the safety cue (CS-) would impair fear extinction. High trait-anxious participants (N=44) were trained to attend towards the threat cue, towards the safety cue, or equally to both cues in a modified dot probe task. Next, transfer of attention processing tendencies was examined in a differential conditioning paradigm across 2 visits. Results indicated participants trained to attend towards the safety cue demonstrated temporary enhancement of extinction performance, but it did not persist through Visit 2. On the other hand, participants trained to attend towards the threat cue demonstrated reduced fear responding on an expectancy measure of conditioning that did not become apparent until Visit 2. These results suggest that attention training towards the threat cue may benefit extinction learning in the long-term and that attention training towards the safety cue may provide short-term relief from fear.

The dissertation of Betty Liao is approved.

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Liao, B. & Craske, M.G. The Impact of State Anxiety on Fear Inhibition. (2013). *Journal of Experimental Psychopathology, 4(2),* 148-160.

Craske, M.G., **Liao, B.,** Brown, L., & Vervliet, B. (2012). Role of Inhibition in Exposure Therapy. *Journal of Experimental Psychopathology, 3(3), 322-345.*

Prenoveau, J.M., Craske, M.G., **Liao, B.**, & Ornitz, E.M. (In Press). Human fear conditioning and extinction: Timing is everything . . . or is it? *Biological Psychology.*

Niles, A., LeBeau, R., **Liao, B.,** Glenn, D., & Craske, M.G. (2012). Dimensional Indicators of Generalized Anxiety Disorder Severity for DSM-V. *Journal of Anxiety Disorders, 26,* 279-286.

Prenoveau, J.M., Craske, M.G., **Liao, B.**, & Ornitz, E.M. (2011). Associative Conditioning Deficits: Caused by or Cause of Anxiety? *Journal of Experimental Psychopathology, 2,* 432-448.

LeBeau, R., Glenn, D., **Liao, B.,** Wittchen, H.U., Beesdo, K., Ollendick, T., & Craske, M. (2010) Specific Phobia: A review of DSM-IV Specific Phobia and preliminary recommendations for DSM-V. *Depression and Anxiety* 27:148-167.

Lewis- Fernández, R., Hinton, D.E., Laria, A.J., Patterson, L.H., Hofmann, S.G., Craske, M.G., Stein, D.J., Asnaani, A., & **Liao, B**. (2010) Culture and the anxiety disorders: Recommendations for DSM-V. *Depression and Anxiety* 27: 212-229.

Falk, E.B.C., Rameson, L., Berkman, E.T., **Liao, B.**, Kang, Y., Inagaki, T.K., & Lieberman, M.D. (2009) The Neural Correlates of Persuasion: A common network across cultures and media. *Journal of Cognitive Neuroscience 22:11,* 2447–2459.

INTRODUCTION

Introduction: Attentional Bias and Anxiety

A normative function of the mechanisms underlying fear is to facilitate detection of danger in the environment and to help the organism respond effectively to threatening situations. Biases in processing threat-related information have been assigned a prominent role in the etiology and maintenance of anxiety disorders (Beck, 1976; Eysenck, 1992; Mathews, 1990; Mathews & MacLeod, 2002; Williams, Watts, MacLeod, & Mathews, 1988). Specifically, it is hypothesized that the selective attention towards threat-related stimuli is moderated by trait anxiety (including both clinically anxious and trait-anxious individuals; Bar-Haim et al., 2007), which is considered a personality trait that predisposes an individual to respond with anxiety to novel or stressful situations (Spielberger et al., 1983), and is an important predisposition to the development of clinical anxiety (Barlow, 2002). It seems appropriate that the mechanisms underlying selective attention allocation towards threat postulated in the context of fear learning would be useful in understanding the development of and treatment for disordered anxiety.

Theoretical Models of Attentional Biases towards Threat in Anxiety

While there is robust evidence that biased attention towards threat is associated with anxiety, the underlying mechanisms by which this association operates are less clear. Over the last two decades, various attentional bias models have been proposed.

Williams, Watts, MacLeod, and Mathews' model (1988)

In this model, threat value of incoming stimuli is determined by an affective decision mechanism (ADF). This system produces an initial decision about whether the information is high or low in threat value based on stimulus features, but also on the individual's

current state anxiety. When a stimulus is appraised as highly threatening, a resource allocation mechanism (RAM) is triggered. When stimulus input is appraised as low threatening, attention is maintained to the task at hand and the new stimulus input is ignored. Furthermore, trait anxiety modulates the resource allocation mechanism in a way such that high trait anxious individuals allocate attention to threat (which results in an attentional bias), whereas low trait anxious individuals ignore threatening information. These directional biases are intensified by increased state anxiety. That is, under stress, high trait anxious individuals become more vigilant, whereas low trait anxious individuals become more avoidant of threat. While this model has inspired thorough investigation, one point under major criticism is the assumption that low trait anxious individuals will direct their attention away from threat, regardless of the severity of threat. It seems implausible that severe threat will not attract attention. Indeed, Wilson and MacLeod (2003) demonstrated that low trait anxious individuals displayed bias towards severe threat, but not towards moderate threat. Despite this limitation, the model has strongly influenced contemporary other models of attentional bias to threat. Additionally, an important implication of this model is that the bias in the RAM underlies individual differences in vulnerability to anxiety, thereby suggesting that treatment should target this particular mechanism, for example, by attentional retraining, to get high trait-anxious individuals to stop orienting to threat and to adopt instead the avoidant attentional style of low anxious individuals.

Mogg and Bradley's cognitive-motivational model (1998)

In this model, attention to threat is considered to be a normal and adaptive mechanism. It draws strongly on neurobiological work of LeDeoux (1996) who

demonstrated that threat can be processed through two different neural pathways: (1) a fast and crude analysis of stimulus features related to previous encountered threat, and (2) a slower, more detailed analysis of stimulus input based on contextual information and information stored in long-term memory. In Mogg and Bradley's model, attention to threat is determined by two systems: first being a valence evaluation system that is responsible for the appraisal of stimuli, which includes preconscious appraisal of stimuli parameters and conscious appraisal based on current state anxiety, contextual information, prior learning history, and biological preparedness. Furthermore, trait anxiety moderates the reactivity of valence evaluation such that high trait anxious individuals have a heightened sensitivity to threat, resulting in mild threat cues being more readily appraised as high threat relative to low trait anxious individuals. Second, output from the valence evaluation system feeds into a goal engagement system, which determines the allocation processing resources. If a stimulus is appraised as threatening, current behavior will be interrupted and attention will be allocated to the new stimulus input. On the other hand, if a stimulus is appraised to be low in threat, further processing of the stimulus will be inhibited, attention will be maintained at ongoing tasks, and current behavior will not be disrupted. In contrast to Williams et al's model (1988), Mogg and Bradley would argue that it would be fruitless to retrain anxious individuals to avoid attending to threat (once the stimuli are appraised as having high threat value) as the selective allocation of attention towards threat is inherently an adaptive mechanism of evolution. Instead, therapeutic efforts may be more usefully targeted in those processes involved in the *appraisal* of stimulus value (such as in cognitive restructuring).

Mathews and Mackintosh's model (1998)

This model predicts that an attentional bias may be triggered when threat has to compete with other stimuli or task-demands. A threat evaluation system (akin to the affective decision system in William's (1988) model) automatically evaluates the stimulus and the output feeds into a distracter/threat representation system. The interference caused by the distraction representation is countered up to a certain degree by voluntary effort aimed at attending to the current task at hand. In accounting for attentional bias, it is postulated that the output of the threat evaluation system is strengthened by anxiety level. More specifically, stimulus input needs to exceed a certain threshold before output will flow from the threat evaluation system into the distraction representation system. A heightened anxiety level lowers the threshold value from the threat evaluation system and causes an increased output of this system. This model further proposes that strong danger cues will attract attention in everyone, whereas weak danger cues will only do so in individuals with a heightened anxiety level (such as those who are trait-anxious). This model shares many similar features with Mogg et al's model (1998). Specifically, they both recognize that the threat value of a stimulus event can be evaluated in two distinct pathways: either via higher level cortical processes, or via a less precise but quicker "short-cut" route directly from the thalamus to the amygdala. With that said, this model emphasizes the automatic nature of the threat evaluation mechanism relatively more than previous models.

Eysenck e*t al.'s attentional control theory (2007)*

This model posits that anxiety disrupts two central executive functions related to attentional control: inhibition and shifting. Inhibition refers to the ability to inhibit or

regulate dominant or automatic responses. Shifting refers to the adaptive ability to shift attention between tasks depending on the context. These two functions involve top-down (goal-oriented) and bottom-up (stimulus-driven) processing. Anxiety impairs the inhibition of top-down regulatory control, such that anxious individuals exhibit difficulties in disengaging their attention from distracting threat stimuli. Furthermore, anxiety heightens the degree to which attention is shifted from one task to another, so that stimulus-driven, bottom-up processing overshadows top-down inhibitory processing of automatic responses. Unlike aforementioned models, at the heart of attentional control theory is the impairment of the efficiency of central executive components of attention. The existence of a threat detection/evaluation mechanism and a goal engagement mechanism described in the aforementioned models are underlying assumptions of Eysenck's attentional control theory, but are not central to the theory itself.

Bar-Haim et al.'s model (2007)

Bar-Haim et al. propose an integrative model that incorporates several aspects of previous models and is consistent with the findings from their most recent meta-analysis (2007). According to this model, a threat evaluation system pre-attentively appraises stimuli in the environment on their degree of threat. Once a stimulus is tagged as having a high threat value, a resource allocation system is triggered and physiological alert state, interruption of current, ongoing activities, allocation of processing resources to the stimulus, and a conscious anxiety state will follow. These outcomes lead to strategic processing guided by a threat evaluation system. Within this system, assessment of contextual information, prior learning history, memory, and the assessment of the availability of coping resources take place. If the outcome from this guided, conscious

evaluation is low in threat, a negative feedback loop is triggered and the pre-attentive threat evaluation system re-corrects its initial evaluation and subsequences processing is terminated. On the other hand, if the outcome is appraised as high in threat, a high state of anxiety is likely to proceed. Individuals with trait anxiety or disordered anxiety demonstrate an automatic tendency to evaluate ambiguous or slightly threatening stimuli as high threat and consequently allocate more attentional resources to such stimuli. Furthermore, such individuals also demonstrate a tendency to consciously appraise stimuli as highly threatening despite contradictory evidence based on contextual information, prior learning, and available coping resources. Lastly, deficiencies in their ability to inhibit the predominant response will further perpetuate an anxious state despite conscious understanding of the irrational aspects of their threat evaluation. In summary, these models all posit a mostly preconscious threat detection mechanism responsible for directing and orienting attention towards threatening stimuli. Some posit a resource allocation mechanism that directs the use of available cognitive resources (Bar-Haim et al., 2007; Mogg & Bradley, 1998; Williams et al., 1988). Some posit a threat elaboration mechanism in which strategic processing evaluates the identified threat as either major or minor (Bar-Haim et al., 2007; Mogg & Bradley, 1998). Some posit a strategic goal engagement mechanism in which goals, beliefs, voluntary effort, or schematic processing can either maintain or override attentional bias to threat (Bar-Haim et al., 2007; Eysenck et al., 2007; Matthews & Mackintosh, 1998). In sum, the only consistent prediction across all the models is that a threat detection mechanism operates at the automatic stage of processing and underlies facilitated attention to threat. The types of downstream effects that an early threat detection system has on other anxiety-related processes (i.e., Pavlovian

fear conditioning), and thereby perpetuate anxiety and the eventual development of an anxiety disorder, are more unclear.

Empirical Evidence: Attentional Bias and Anxiety

A wealth of research has demonstrated that anxiety and fear are *associated* with selective attention for fear-relevant stimuli (see meta-analysis based on 172 studies by Bar-Haim et al., 2007). For example, attentional biases towards threat have been demonstrated in individuals experiencing post-traumatic stress disorder (PTSD; Buckley, Blanchard, & Neill, 2000), social phobia (Clark & McManus, 2002; Heinrichs & Hofman, 2001; Musa & Lepine, 2000), obsessive– compulsive disorder (OCD; Summerfeldt & Endler, 1998), generalized anxiety disorder (GAD; Mogg & Bradley, 2005), panic disorder and phobias (McNally, 1999), and in children (Ehrenreich & Gross, 2002; Waters et al., 2010). Many investigators used variations of the dot probe task (MacLeod et al., 1986) to confirm this association.

The dot probe task (Figure 2) displays two words on a computer screen with one at the top and one at the bottom (alternatively, the words may appear on the left and right side of the screen). Following a brief stimulus presentation duration (e.g., 500 ms), the stimuli disappear and a probe appears in a location previously occupied by one of the stimuli. The participant is asked to press a button indicating whether the top or bottom stimulus had been replaced by the probe. Attentional biases are inferred from different response times towards probes that replace threatening stimuli (i.e., congruent trials) compared to probes that replace neutral stimuli (i.e. incongruent trials). If an individual's attention is systematically drawn to the threat stimulus, response times will be shorter for probes that replace threatening stimuli compared to probes that replace neutral stimuli.

Modifying this basic design for anxious individuals, the stimuli that precede the probe have included emotional faces (e.g., Bradley et al., 1999), emotionally arousing photos from the International Affective Picture System (e.g., Koster et al., 2006), threat-relevant words (such as "heart attack" for panic patients; add citation), and conditioned (fear) stimuli (e.g., Pischek-Simpson et al., 2009).

While there is a large body of literature confirming the association between attentional biases towards threat and disordered anxiety, the assumption that attentional processes play a *causal* role in the emergence and maintenance of disordered anxiety is still speculative. The most powerful test of the causal hypothesis comes from studies that directly manipulate attentional biases and then observe the impact of such manipulations on the emotional experience. In essence, this involves inducing an attentional bias towards the to-be threat-relevant stimuli (relative to neutral stimuli) in healthy volunteers and then examining the effect the attentional bias induction has on anxiety level. For example, in healthy individuals, MacLeod and colleagues (2002) induced either an attentional bias towards threatening words or neutral words by using a modified version of the dot probe task. For the group that underwent threat bias induction, probes always appeared in the location of threat words. For another group, the probes always appeared in the location of neutral words, with the intention of inducing the reverse bias. In a subsequent non-contingent test phase, the former group was faster to detect targets in the location of new threatening words, and vice versa for the latter group, suggesting that differential attentional bias was successfully induced. Most critically, in a subsequent stress task involving difficult anagrams, the former group reported greater increases in negative mood than did the latter group. By implication, learning to attend to threat cues served to

influence how participants process the later stress task, leading to increase anxiety reactivity.

Attention Bias Modification Training (ABMT)

Variations of MacLeod's "attention retraining" dot-probe task (2002) is collectively known as Attention Bias Modification Training (ABMT) and it provides further support for the causal role of attention in anxiety development. Namely, if the *elimination* of an attentional bias towards threat can cause a *reduction* in anxiety symptomatology, then the *induction* of an attentional bias towards threat should also cause an *increase* in anxiety symptomatology. As noted, the dot probe task (MacLeod et al., 1986) has been adapted to not only *assess* attentional bias, but to also *modify* attentional bias by altering the contingency between the location of probes and disorder-relevant stimuli (MacLeod et al., 2002). If probes always replace neutral or positive stimuli, attention may be directed away from disorder-relevant stimuli with the hope that with practice, the anxiety vulnerability associated with an attentional bias towards threat will be reduced or eliminated altogether. In these neutral or positive training conditions, acquiring an attention bias away from disorder-relevant stimuli will facilitate faster reaction time on the task and then reduce dysfunctional emotional reactivity to a subsequent laboratory challenge. Researchers have been enthusiastic about using ABMT paradigm because of the methodological advantage of having an extremely well-matched control condition. In the control condition, probes replace neutral or positive and disorder-relevant stimuli with equal frequency. In other words, the control is simply the assessment version of the dot probe task. Work by Matthews & MacLeod (2002) demonstrated that multiple practice sessions designed to train attentional avoidance of threat in high-trait-anxious students decreased their anxiety

scores in response to an impending examination.

Due to the potential treatment implications of ABMT, it has grown rapidly over the last decade, and two quantitative reviews of the literature have been published. Hakamata and colleagues (2010) conducted a specific literature review of the effect of ABMT on attention bias and anxiety based on 12 datasets. They found that ABMT resulted in a large effect on attentional bias (d=1.16, CI=0.82-1.50) and a medium effect on anxiety (d=0.61, CI=0.42-0.81). Furthermore, effect sizes for anxiety were moderated by several variables: (1) larger in clinical population (d=.78), (2) larger after stressful task (d=.77), (3) larger on trait anxiety (d=1.06) than state anxiety (d=.41), (4) larger to words (d=1.29) than to faces (d=.37), and (5) larger in top-down (d=.79) than in left-right (ns) presentational form of cues/probes.

Extending the findings by Hakamata and colleagues, Hallion and Ruscio (2011) examined the effect of ABMT on depression in addition to anxiety based on a meta-analysis of 45 studies. Overall, ABMT had a medium effect on biases (g = 0.49) that was stronger for interpretation (g = 0.81) than for attention (g = 0.29) biases. ABMT further had a small effect on anxiety and depression (g = 0.13), although this effect was more reliable when symptoms were assessed after participants experienced a stressor (g = 0.23). These findings were broadly consistent with cognitive theories of anxiety and depression that propose an interactive effect of cognitive biases and stressors on these symptoms. However, the small effect sizes observed here suggest that this effect may be more modest than previously believed. Based on the aforementioned reviews, the magnitude of the efficacy of ABMT is unclear given that Hakamata suggested large effects on attention and medium effects on anxiety while Hallion and Ruscio suggested small effects on both anxiety

and attention. Additionally, the Hakamata review did not apply standard fail-safe N guidelines when interpreting effect sizes. Furthermore, the Hallion and Ruscio review did not examine a number of task characteristics as potential moderators (e.g., population characteristics, probe/cue type and location, etc.); thus, the moderators taken into account by Hakamata await replication in a larger sample. Such findings will have direct implications for how future ABMT should be delivered. But, most critically, the next step in attentional bias and modification research is to elucidate how such bias may independently contribute to or synergistically interact with other etiological models (i.e., fear conditioning) to the development and perpetuation of anxiety. Similarly, how such bias may contribute to or interact with other anxiety treatment models (i.e., fear extinction) to the reduction of anxiety symptoms is also unclear.

Fear Conditioning and Anxiety

Pavlovian conditioning is a widely accepted model for the etiology of anxiety disorders. During aversive conditioning, repeated pairing of a neutral stimulus (conditional stimulus; CS) with an intrinsically aversive stimulus (unconditional stimulus; US) leads the CS to elicit a conditional fear response (CR). The early conditioning models assumed that traumatic conditioning experiences were both necessary and sufficient for the development of phobic fears and other anxiety disorders (Watson & Rayner, 1920). The paradigm shifted when Eysenck (1979) proposed that pathological anxiety results from excessive fear acquisition to aversive stimuli in susceptible individuals relative to non-anxious individuals. In other words, anxious individuals were hypothesized to show enhanced conditionability to cues that signal threat (CS+). Contemporary learning theories expanded on these models by incorporating the role of fear incubation (Eysenck, 1979),

evolutionarily prepared aversive associations (e.g., Ohman, 1986; Seligman, 1971), failure to inhibit fear to safety cues (Davis et al., 2000), associative learning deficits (Grillon, 2002), and stimulus generalization (Mineka & Zinbarg, 1996) in the formation and persistence of anxiety disorders. Davis, Falls, and Gewirtz (2000) identified a failure to inhibit or suppress the CR in the presence of safety signals, such as the CS- or the CS during fear extinction, as a mechanism by which pathological anxiety may develop. A meta-analysis of the conditioning literature concluded that anxious individuals, relative to healthy controls, show "deficits in inhibition" of fear CR to a CS that predicts safety from threat (i.e., CS-) during fear conditioning *and* to CS that no longer signals threat (i.e., during extinction) (Lissek et al., 2005). It is now believed that impaired inhibitory mechanisms may be central to development of various anxiety disorders. Inhibitory learning means that the original CS-US association learned during fear conditioning is not erased during extinction, but rather is left intact as a new, secondary inhibitory learning about the CS-no US relationship develops (e.g., Bouton & King, 1983; Bouton, 1993). It is this secondary, CS-no US association that forms the basis for fear inhibition. In other words, this model implies that at the end of extinction, the CS engages in two competing processes simultaneously: an excitatory association from fear acquisition and an inhibitory association from fear extinction. Consequently, even though fear expressly subsides by the end of fear extinction, retention of at least part of the original association can be uncovered by various procedures, with each one showing a continuing persistence of the original excitatory association after extinction. These procedures include spontaneous recovery, context renewal, rapid reacquisition and reinstatement (Bouton, 1993). Exposure therapy is an efficacious treatment for anxiety disorders and there is abundant evidence that it is more

effective than either wait-list or attention placebo controls (Norton & Price, 2007; Hofmann & Smits, 2008) or active treatment comparisons (Tolin, 2010). During exposure therapy, the patient gradually confronts anxiety-provoking cues (internal or external) that are perceived to lead to an aversive outcome. With repeated practice, cues that were originally anxiety-provoking (CS) will no longer elicit such reactivity (CR) because patients learn that the likelihood of an aversive event (US) happening due to encountering such cues is minimal (or non-existent). This process of fear reduction is believed to operate in part through extinction processes (Bouton, 2001; Mineka, 2006). Although exposure therapy is an efficacious behavioral treatment (Barlow et al., 2002; Chambless & Hollon, 1998), not every patient benefits (non-response rate vary from 10-40%; Craske, 1999). Also, of those patients who do benefit, many experience a subsequent return of fear (Rachman, 1989). Better understanding of fear extinction mechanisms will help to optimize exposure therapy parameters, which in turn could lead to more efficacious treatments and longer-lasting outcomes.

Theoretical Models of Pavlovian Conditioning

It is apparent that better understanding of Pavlovian conditioning processes will not only illuminate the mechanisms by which anxiety is developed and sustained, it will also help to optimize exposure therapy parameters, which in turn could lead to more efficacious treatments.

Rescorla-Wagner model (1972)

This model has generated many new ideas within the field of learning and behavior since its conception. Although it has failed to address a number of important issues (see Miller, Barnet, & Grahame, 1995), it has continued to be the standard against which

subsequent theories are measured. Rescorla and Wagner created a formal mathematical model to compute the amount of learning expected to occur with each trial of conditioning (ΔV). The model is based on the critical assumption that the degree to which learning occurs to the CSs presented during a conditioning trial depends on how "surprising" the US is (λ – V). By definition, an event (λ) is surprising if it is different from what is expected (V). With regard to fear conditioning, an unexpectedly aversive US is the basis for excitatory fear conditioning and will lead to increased reactivity, and an unexpectedly neutral US (or the absence of the aversive US) is the basis for inhibitory fear conditioning and will lead to a reduction in reactivity. This maps on well to rates of learning in human fear conditioning experiments where the most learning occurs at the beginning of the phase when the presentation of the US is the most surprising during conditioning (and its absence is most surprising during extinction), and then the rate of learning slows with each subsequent trial until what is expected (V) grows to match what actually happened (λ) and the surprise term (λ – V) becomes zero, at which point no more learning can occur. While the level of surprise of the US is necessary for learning to occur, other factors, such as the relative salience of the CS (α) and the relative strength of the US (β), will also determine the rate of fear conditioning. Putting everything together, the formal mathematical model can be expressed as: $V = (\alpha*\beta)(\lambda - V)$. The Rescorla-Wagner model assumes that the salience or the "attention-gettingness" of the CS is a constant throughout learning. In other words, organisms are assumed to allocate the same amount of attention to CSs across fear conditioning (including extinction) trials.

Mackintosh's model (1975)

Unlike the Rescorla-Wagner model where learning is driven by the effectiveness of

the US, attentional models of conditioning, namely, Mackintosh's model, suggest that learning depends on how well the CSs command one's attention (α). Specifically, attention to a given CS increases when that CS is the best predictor of the US, and decreases otherwise. This model can be expressed as $\Delta\alpha A>0$ if $|\lambda-VA|\geq|\lambda-VX|$, where αA is the CSA-specific learning rate, λ is the occurrence (λ =1) or absence of the US (λ =0), VA is the association of CSA with the US, and VX the association with the US of all CSs other than CSA. As one can see, the main change proposed by Mackintosh to the Rescorla-Wagner equation was that α is not a fixed function of the salience of the CS, but changes as a result of experience with the CS. Furthermore, it emphasized that attention to the CS is equivalent to its learning rate. The specific rule proposed captured the common intuition that subjects would learn to attend to relevant stimuli that predicted trial outcomes and ignore irrelevant stimuli that did not.

Pearce and Hall's model (1980)

Attentional models of conditioning differ in their assumptions about what determines the noticeability of the CS on a given trial (α). For example, Pearce and Hall assumed that the amount of attention a subject devotes to the CS on a given trial is determined by how surprising the US was on the preceding trial. Like many attentional models of conditioning (including Pearce & Hall, 1980), an important feature is that they assume that the level of surprise of the US on a given trial influences what is learned on the next trial. This differs from US-reduction models like the Rescorla-Wagner model, in which the level of the US surprise on a given trial determines what is learned on that given trial. This also differs from the Mackintosh model (1975) in that once sufficient learning has occurred and the US is no longer surprising, there should be little attention allocated to the

CS. In other words, this model would hypothesize that little attention would be directed towards the CS at the end of fear acquisition and extinction.

In summary, while researchers have built upon Rescorla and Wagner's model of conditioning by including a term to capture modifications in attention to the CS based on one's experience with the US after each learning trial (Pearce & Hall, 1980), many attentional models of conditioning (some of which were not included in this proposal) differ in what determines the attention allocated to the CS on a given trial. What is common to all models (i.e., Mackintosh, 1975; Pearce & Hall, 1980) is that they examine the causal effect of associative learning (whether or not the CS-US or CS-no US association is predictable) has on attention allocation. While it is universally assumed that paying attention to the CS and US are requisites for any learning to occur and procedures that disrupt attention to the CS are expected to also disrupt learning, these models do not explicitly delineate how such modifications to attention may enhance or impair Pavlovian conditioning and Pavlovian fear conditioning in particular. This is not particularly surprising as many models of Pavlovian conditioning were derived from animal research where measuring the animal's attention was difficult (or nearly impossible) to carry out on a trial to trial basis. However, in order to advance our knowledge of the etiological and treatment models of pathological anxiety based on basic science research of Pavlovian conditioning, theories of attention (broadly speaking) ought to be incorporated as sufficient data has accumulated from human research pointing to the association between disordered fear learning and an attentional bias towards the CS that predicts threat (Bar-Haim et al., 2007).

Attentional Bias and Fear Conditioning

Experimental research that integrates attentional bias and fear conditioning is relatively limited despite an obvious need to consider the role that cognitive processes, such as attention, play during Pavlovian fear conditioning in understanding the etiology, maintenance, and treatment of anxiety disorders. To date, only a handful of studies using the dot probe task or the spatial cueing task (visual task capable of teasing apart attention engagement and disengagement) have demonstrated increased attentional bias to the CS+ *after* fear conditioning relative to before conditioning and relative to the CS- (Koster et al., 2004; Van Damme et al., 2004a; Van Damme et al., 2004b; Hermans et al., 2005; Beaver et al., 2005; Van Damme et al., 2006; Pischek-Simpson et al., 2009). Furthermore, two studies also found evidence for the reduction of such bias towards the CS+ after fear extinction (Hermans et al., 2005; Van Damme et al., 2006). These studies converge in providing preliminary evidence for the causal impact of Pavlovian conditioning procedures on attention allocation to the cues presented. With that said, there is very little experimental research illuminating the role that attention allocation play in fear acquisition and extinction. No studies to date have manipulated attention allocation towards or away from a signal of threat during fear acquisition. It is perhaps obvious that attention to the task at hand is required for learning to occur. Therefore, it may be universally assumed that directing attention away from the threat-relevant cue will impair fear acquisition and directing attention towards the threat-relevant cue will enhance fear conditioning. What is more surprising is that only one study to date has examined the effect of training individuals to attend towards or away from an acquired signal of threat during extinction (Van Bockstaele et al., 2010) despite clear implications for exposure therapy. Van

Bockstaele and colleagues (2010) modified the spatial cueing task such that during fear extinction, participants were assigned to one of three groups: 1) attend towards threat, 2) attend away from threat, and 3) control. These groups differed in the frequency to which the CS+ was followed by the probe, as similarly described in MacLeod et al's (2002) attention bias modification training. Importantly, Van Bockstaele found that attending *towards* the CS+ during extinction enhanced extinction learning via participants' self-reported measures of CS-US contingency, and CS valence and arousal. While these findings await replications, these results were the first to suggest a causal role of attention allocation upon fear conditioning (i.e., extinction), provide further evidence for the interplay between attentional processes and fear learning, and provide preliminary evidence that attention modification training may optimize fear extinction (or exposure therapy). However, it must be noted that this finding is in contradiction with the growing body of literature based on MacLeod's AMBT which posited that directing attention *away* from threat-relevant stimuli can lead to reductions in anxiety (Hakamata et al., 2010). Clarification of the exact nature of the causal influence of attention allocation on fear extinction is urgently warranted given its clinical implications for anxiety disorders.

Theories of Attentional Bias on Fear Conditioning in Anxiety

Although a satisfactory theory that is capable of integrating both attentional and Pavlovian mechanisms of fear conditioning in explaining the development of anxiety is yet to be available, one can speculate as to what the role of a selective attention towards threat may play in explaining the inhibitory Pavlovian deficits (i.e., elevated responding to CS- during acquisition and impaired extinction to the CS+) exhibited by trait-anxious and clinically-anxious individuals. Due to an over-active, automatic threat detection mechanism

(Williams et al., 1988; Mogg & Bradley, 1998; Bar-Haim et al., 2007) and a difficulty in disengaging from threat once it is located, when an anxious individual undergoes fear conditioning, it is possible that the elevated fear responding to the CS- occurred as a result of not learning the safety of the CS- as attention continues to be allocated to the CS+. Similarly, during extinction, the attention may continue to be drawn towards the CS+ as the CS+ now represents two contradictory associations (an excitatory one from conditioning and an inhibitory one from extinction), and hence draws greater attentional resources from the anxious individual due to its ambiguous nature. This hypothesis would be consistent with attentional theories posited by Mogg et al (1998) and Mathews et al (1998), who predicted that the most robust difference between anxious and non-anxious individuals is the selective attention towards "weak" threat. However, instead of using the additional attentional resources allocated towards learning the "inhibitory" CS-no US contingency, a *preconscious* threat evaluation mechanism will automatically appraise a previously encountered threat as threatening when it is encountered again (Mathews & Mackintosh, 1998). Alternatively, the inhibitory learning during extinction that usually takes place in non-anxious individuals may be high jacked by the *conscious* appraisal of the CS+ as threatening in anxious individuals due to their prior learning about the CS+ (such as during fear acquisition), biological preparedness for hyper-arousal, among others. (Bar-Haim et al., 2007; Mogg & Bradley, 1998).

Neural Bases of Fear Conditioning and Selective Attention to Threat

The neural bases of fear conditioning and extinction have been well established in human samples. The amygdala plays a primary role in fear conditioning. In healthy humans, fMRI studies show elevated amygdala activation during conditioning (for a review,

Shin & Liberzon, 2010). Conditioned fear is presumed to be mediated by the transmission of sensory information about the CS and US to the amygdala and the subsequent control of fear reactions via projections from the amygdala to hypothalamic and brainstem regions that regulate behavioral, endocrine, and autonomic responses (e.g., skin conductance responding (SCR); LeDoux, 2000). In support, amygdala activity has been positively correlated with SCRs during fear conditioning (LaBar et al., 1998; Phelps et al., 2001). Other regions associated with fear conditioning are the insular cortex (e.g., Shin & Liberzon, 2010; Phelps et al., 2004) and the dorsal and rostral anterior cingulate (ACC; e.g., LaBar et al., 1998; Phelps et al., 2004). The ventral medial PFC (vmPFC) appears to mediate extinction. Human neuroimaging studies have associated greater vmPFC activation with extinction and at extinction recall (e.g., Milad et al., 2007; Phelps et al., 2004). Specifically, the vmPFC modulates fear CR through descending inhibitory projections to the central nucleus of the amygdala (CeA; e.g., Maren & Quirk, 2004; Sotres-Bayon et al., 2009). Furthermore, the dorsal ACC and the vmPFC have been implicated during extinction by down-regulating the expression of the fear CR from the amygdala (e.g., Carlsson et al., 2006; Phelps et al., 2004). Such regions activated during extinction may also serve as the neurobiological basis for fear inhibition to safety cues (CS-) during conditioning. The neural correlates of anxiety disorders may involve hyper-activation of the amygdala during fear conditioning and extinction and hypo-activation of frontal regions during extinction (e.g., Bishop et al., 2004; Bishop et al., 2007). For example, high trait anxiety is related to amygdala hyperactivity during the processing of aversive and neutral stimuli (Bishop et al., 2004; Etkins et al., 2004; Dickie & Armony, 2008) *and* even during the extinction of conditioned fear (Barrett & Armony, 2009). Anxiety also correlated with decreased activity

in the dACC during extinction (Sehlmeyer et al., 2010) and decreased activity in vmPFC during extinction (Bremner et al., 2005) and extinction recall (Milad et al., 2007; 2009). Neurocircuitry of selective attention to threat in anxiety suggests a pathway between the amygdala and PFC that corresponds with the neural correlates of extinction learning. That is, evidence to date indicates that anxious individuals show an increased amygdala response to both attended and unattended threat stimuli (fearful faces; Bishop et al., 2004) and hypoactivation in prefrontal control mechanisms in response to attentional competition from threat-related distracters. For instance, when the frequency of threat-related distracters was increased, anxious individuals showed both reduced activity in the and lateral prefrontal regions and the rostral ACC (Bishop et al., 2004; Bishop et al., 2006; Hull, 2002; Shin et al., 2001). These findings parallel the findings reported for fear conditioning and extinction in anxious adults (Bremner et al., 2005). This commonality at the neural level possibly reflects a greater conceptual link than is sometimes recognized between fear conditioning and selective attention to threat. A closer integration of attentional and Pavlovian conditioning models of anxiety disorders may help inform etiology and provide insight into discordant findings at the behavioral, affective, and cognitive level when examined in isolation from one another.

Specific Aims

The broad goal of this study was to elucidate the interaction between attentional and Pavlovian processes in fear learning. While attentional and Pavlovian processes may reciprocally contribute to the development and maintenance of anxiety, this study intends to emphasize the causal impact of attention on Pavlovian fear conditioning. This goal was achieved by (1) directly examining the causal influence of differential fear acquisition on

attention allocation to safety vs. threat cues as measured by autonomic reactivity and self-report, and (2) evaluating the effect of attention retraining (towards threat or safety cues) on self-reported, physiological, and behavioral measures of fear extinction.

Hypotheses

(1) Differential fear acquisition on attention allocation to safe vs. threat cue**.** Consistent with extant data (e.g., Pischek-Simpson, 2009; Van Damme et al., 2006), we predicted that a selective attention towards threat (CS+) relative to a neutral stimulus will occur as a result of undergoing fear acquisition. The association between the CS+ and the US will make the CS+ a stimulus of high threat value; therefore directing one's attention towards a threatening stimulus may be adaptive. Also consistent with extant literature (Pischek-Simpson, 2009; Van Damme et al., 2006), We also predicted that participants will *not* selectively attend to the CS- relative to a neutral stimulus. The CS- predicts the absence of threat just as a neutral stimulus could also predict the absence of threat as it was never paired with the US during fear acquisition. (2) Effect of attention retraining (towards threat, towards safety, or neither) on measures of fear extinction. We predicted that attentional retraining will have differential effects on fear extinction. Namely, training participants to attend *towards* the CS+ will enhance fear extinction (compared to attending towards the CS- or to neither) as attention is needed to detect the contingencies, or lack thereof, between a stimulus and its negative outcome (Van Bockstaele et al., 2010). Although a number of studies have demonstrated the beneficial effects of ABMT on cognitive and behavioral responses to a subsequent stressor (see review by Hakamata et al., 2010), implying that attention retraining (i.e., directing attention away from the CS+) may also benefit fear extinction (if fear extinction is conceptualized as a stressor), the

results from Van Bockstaele et al (2010) provided direct evidence against ABMT on fear extinction. Furthermore, we also predicted that training individuals to attend towards the CS- would impair fear extinction relative to training individuals to attend towards the CS+.

METHOD

Design

Participants were randomly assigned to one of three experimental groups. Namely, participants underwent one of three attention bias modification training (ABMT): (1) Training towards the CS+ (TD), (2) Training towards the CS- (TS), or (3) Training to attend equally to the CS+ and CS- (CON). The distribution of participants across the three groups was as follows: TD = 15, TS = 15, CON = 14.

Participants

Forty-four high-trait anxious University of California at Los Angeles undergraduates participated for course credit. They ranged in age from 18 to 24 years old (*M*ean=19.89, *SD*=1.34) and consisted of 28 females (70.%) and 16 males (30%). Self-endorsed ethnic breakdown of the sample was as follows: 34.1% Asian, 27.3% Caucasian, 15.9% Hispanic/Latin American, 9.1% African American, 4.6% Middle Eastern, and 9% multiethnic.

Participants were recruited if they scored in the top quartile of scores on the State-Trait Anxiety Inventory-Trait (i.e., score $\geq$45). Exclusion criteria for study participation included 1) any heart, respiratory, or neurological problems, 2) currently in treatment for an emotional problem, 3) pregnancy, and 4) current or history of addiction.

Measures

Self-Reported Questionnaires

Trait anxiety and depression were measured, given evidence for their relationship with fear learning (Otto et al., 2007; Prenoveau et al., 2011). Trait anxiety was assessed using the State –Trait Anxiety Inventory (STAI; Spielberger et al., 1970), which possesses good reliability and validity. Vulnerability to anxiety was assessed using the Behavioral Inhibition Scale (BIS; Carver & White, 1994), which has acceptable reliability and convergent and divergent validity. Depressive symptomatology was assessed using the Beck Depression Inventory II (BDI-II; (Beck et al., 1996), which has excellent test-retest reliability, high internal consistency, and moderate to high convergent validity. Since mindfulness and other acceptance-based approaches have also been hypothesized to strengthen extinction learning (Treanor, 2011), trait-mindfulness was also measured by using the Mindful Attention and Awareness Scale (Brown & Ryan, 2003).

Self-Reported Measures

Subjective Units of Discomfort (SUD)

Throughout the experiment, state anxiety levels were assessed using a 100-point Likert Subjective Unit of Discomfort (SUD) scale (0 = 'no discomfort', 25 = 'little discomfort', 50 = 'moderate discomfort', 75 = 'strong discomfort', 100 = 'extreme discomfort'). SUD was assessed at 7 time points across the experiment.

Valence and Arousal Ratings

Subjective ratings of valence and arousal of the CSs were assessed using an 11-point Likert scale (0 = 'not at all fearful' or 'not at all unpleasant', 10 = 'very fearful' or 'very unpleasant'; Prenoveau et al., 2011).

US Expectancy Rating (EXP)

Participants' expectancy of receiving a shock (the US) was rated during CS presentations and inter-trial intervals (ITIs). Ratings were made by using a Biopac Subject Feedback Device (TSD115) to slide a pointer along an analog scale between the extremes of 0='certain no shock' and 9='certain shock' with a midpoint of 4.5='uncertain.' A signal (i.e., 'shock?') appeared onscreen at specific times (see *Procedure* for details) to prompt participants to make a rating. At these times, they were instructed to make their rating based on their expectancy of receiving the US in "the next few moments" (Prenoveau et al., 2011). Participants were given 5 seconds to make a response by moving the pointer. They were instructed to leave the pointer at that point on the slider until prompted to make the next response. Finally, the raw data was re-centered at 4.5 prior to data analysis, so that positive values would indicate increasing expectancy of US delivery and negative values would indicate decreasing expectancy of US delivery.

A short recognition questionnaire was administered after the Acquisition phase and Reinstatement phase to assess awareness of the CS-US contingency, using procedures described by Dawson and Reardon (1973).

Physiological Measures

Acoustic Startle Response (ASR)

Acoustic startle responses (ASRs) were measured in response to an acoustic startle probe, which was administered 7.5 seconds following each CS onset. The acoustic startle probe delivered to elicit the ASR consisted of 50 ms, 104 dB bursts of "white noise" with an instantaneous rise time delivered binaurally via stereophonic headphones. The ASR was measured by electromyogram (EMG) activity of the orbicularis oculi, which was recorded

from two miniature Ag-AgCl electrodes placed beneath the right eye approximately 10 mm apart edge-to-edge and 8 mm below the lower lid margin. The lateral electrode was placed 5 mm medial to the outer canthus. EMG was full-wave rectified and low and high frequency cut-off values were 30 Hz and 1000 Hz. The magnitude of the ASRs was based on the peak of the response during the 20 – 150 ms window following the probe. Each ASR was visually inspected prior to data inclusion. ASRs confounded by motor or electrical artifacts were excluded. Finally, raw data was natural-log transformed in order to normalize the distribution prior to statistical analysis.

Skin Conductance Response (SCR)

Skin conductance responses (SCRs) were measured at the onset of each CS. To measure SCRs, two disposable 1 cm diameter Ag-AgCl electrodes were placed on he distal phalanx of the index and middle fingers of the non-dominant hand. The raw data underwent a 2 Hz low-pass filter to filter out high frequency noise. The magnitude of the SCRs was calculated as the difference between the maximum skin conductance level (measured in microsiemens) within 1-6 seconds following CS onset and the lowest skin conductance level within the first 0.5 to 4 seconds following CS onset. The amplitude of the SCRs was range-corrected using the largest response elicited by the US for each individual participant for each experimental visit. For each participant, all SCRs were divided by that person's maximum SCR to the US. These range-corrected responses were then square root transformed in order to normalize the distribution prior to statistical analysis. SCRs were rejected for a given CS presentation if behavioral observations indicated movement. SCRs were scored as zero for a given CS presentation when there was no observable peak in skin conductance level within the 1-6 second window following CS onset.

Apparatus and Stimuli

The conditional stimuli (CS) were 4 geometric shapes: red square, blue triangle, purple diamond, and yellow trapezoid. The red square and purple diamond were always the CS+ (CS_A and CS_B), and the blue triangle and the yellow trapezoid were always CS- (CS_C and CS_D). After Acquisition, one set of CS+ and CS- (CS_A and CS_C) was used as stimuli during attention bias modification training and the other set of CS+ and CS- (CS_B and CS_D) underwent Extinction (counterbalanced across participants). An orange pentagon and a green circle (neutral stimuli; N1 and N2) were only presented during the Dot Probe Task (pre- and post-) and Attention Bias Modification Training (ABMT) and never during fear Acquisition, Extinction, and Reinstatement. All experimental stimuli were displayed on a 23-inch computer monitor located 3 feet in front of participants at eye level. The unconditional stimulus (US) was an electrical shock to the biceps that consisted of a train of 17 short pulses that summed to 500ms in duration. The ITIs involved presentation of a fixation cross. CS display, US delivery, ITI display, and US expectancy ratings were under the control of E-prime Stimulus Presentation Software (v2.0) installed on a Dell Inspiron Workstation computer.

Procedure

The experiment consisted of several phases that were completed over the course of two visits (scheduled 7 days apart). All participants were asked to abstain from ingesting stimulants (e.g., caffeine) for at least 1 hour prior to the first session. At the beginning of the first session, a trained research assistant described the study procedures to the participant. After obtaining informed consent, participants were administered a battery of self-reported measures: BDI, STAI, ASI, MAAS, and BIS.

Next, participants were seated in a comfortable chair that was situated approximately 3 feet from a computer monitor. Eight electrodes were placed on participants for recording. Two electrodes were needed for each of the following measures/stimuli: heart rate EKG, acoustic startle response (EMG), skin conductance response (SCR), and electrical stimulation. Leads were connected from these electrodes to the Biopac Acquisition System (MP150). Shock electrodes were attached to the biceps. To establish the level of electrical shock, participants underwent a work-up procedure where each individual selected a level that was "aversive, but tolerable and requires some effort to deal with" (Van Damme et al., 2005). This level was then applied throughout the experimental phases. Next, participants underwent 10 practice trials of the Dot Probe Task (DPT) to confirm comprehension of instructions.

During Visit 1, participants underwent 4 phases: Pre-Acquisition, Fear Acquisition, Attention Bias Modification Training (ABMT), and Extinction. Pre-acquisition consisted of one trial of each of the six experimental stimuli (4 CSs, N1, N2). Each trial was 8 seconds in duration. Duration of the inter-trial interval (ITI) was 20 seconds on average, and varied between 16 and 24 seconds. US expectancy ratings were prompted on the computer monitor immediately after each trial onset for 5 seconds. Following completion of the pre-acquisition phase, participants completed the 0-100 point SUD scale.

Next, participants were informed that now they may experience electrical shocks to their bicep. They were not explicitly informed of a CS-US relationship. Fear Acquisition consisted of 8 trials of each of the 2 CS+s and 2 CS-s, interspersed with ITIs. CS+ trials were composed of a red square and a purple diamond presented at the center of the computer screen, for 8 seconds, with the onset of the US (shock) at 7.5 seconds, and co-

termination of the CS+ stimuli and US. Twelve out of the 16 CS+ trials were reinforced by the US. CS- trials were composed of a blue triangle and a yellow trapezoid presented in the center of the computer screen, for 8 seconds, without the US. For the first five seconds after CS onset, participants were prompted to rate US expectancy (Figure 1). The ITIs, which were comprised of a blank monitor with a central fixation cross, ranged in duration from 16 to 24 seconds (average = 20 seconds). All participants received the same pseudo-random order of CS+ and CS- trials. The CS trial order consisted of 8 blocks, each containing one presentations of each trial type. Within each block, trial order was assigned randomly with the restriction that the same trial type was not repeated consecutively more than twice between blocks. Following completion of this phase, participants completed the SUD scale, valence and arousal ratings, and a recognition questionnaire of the CS-US contingency.

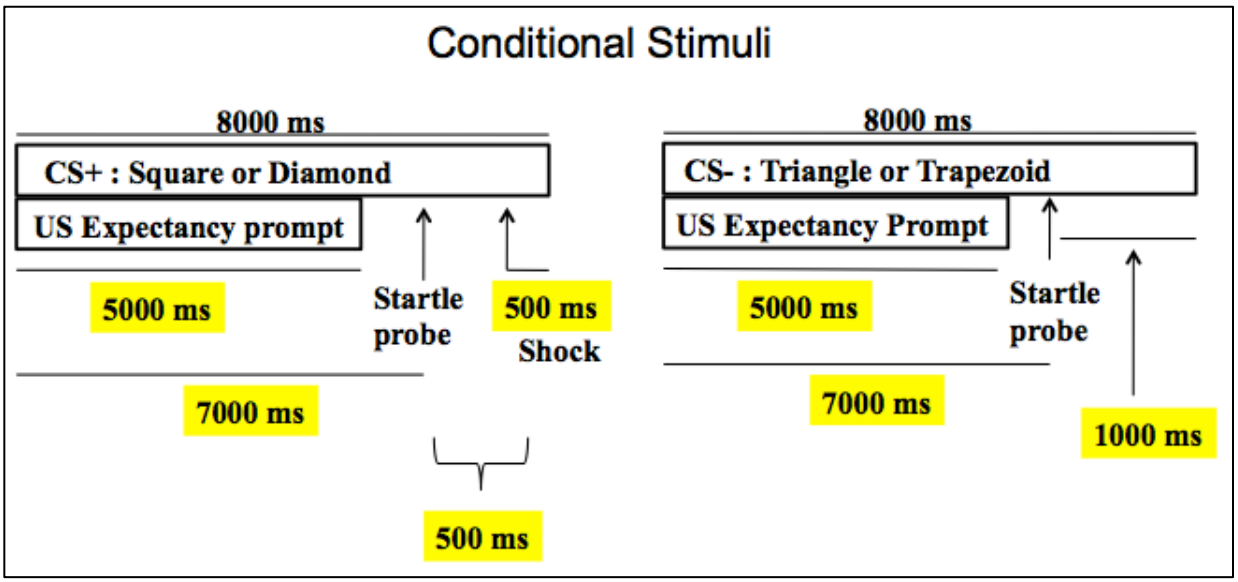


Figure 1.

Following Acquisition, all participants underwent an assessment of attention allocation to one set of CS+ and CS- (CS_A and CS_C), and the neutral stimuli (N1, N2) by completing the Dot Probe Task (DPT). Each DPT trial began with a fixation cross presented in the center of the computer screen for 500 ms. Then, the cross was replaced by a geometric shape pair (CS+/neutral, CS-/neutral, or neutral/neutral), with one shape placed above and one below the fixation, for 500 ms. The shape pair then disappeared and a probe (i.e., vertical colon ":" or horizontal colon ". .") appeared immediately in the location previously occupied by one of the two shapes. The probe remained on the screen until the participants respond (up to 1.4s). Participants responded by clicking on either the left arrow key (for ". .") or up arrow key (for ":") on the keyboard by using their index finger for the left arrow key and their middle finger for the up arrow key. After the response, there was a 500 ms interval of a blank screen before the next trial began with a fixation cross. Response latencies to identify the probe were recorded. During DPT, participants were presented with one block of 120 trials comprising of three trial types (48 CS+/neutral, 48 CS-/ neutral, 24 neutral/neutral). For each trial type, the location of the cue (top or bottom) was equally distributed, and the location of the probe (top or bottom) was also equally distributed. Trials were presented in a new random order to each participant. Attentional biases were inferred from different response times towards probes that replace the danger cues (CS+; valid trials) compared to probes that replace the safety cues (CS-; valid trials). A faster response towards probes that replaced the CS+, compared to probes that replaced the CS-, reflected an attentional bias (see Figure 2).

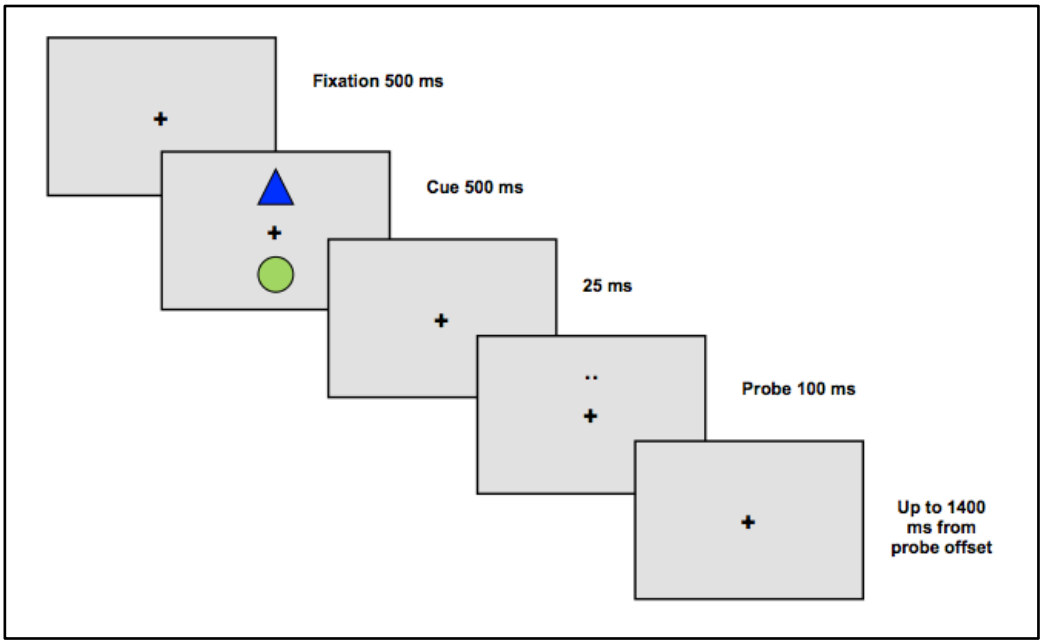


Figure 2.

Next, participants were randomly assigned to one of three attention bias modification training (ABMT) conditions. ABMT consisted of the DPT described above, but modified to facilitate an attention bias towards the CS+ (Towards Danger; TD), CS- (Towards Safety; TS), or equally between the CS+ and CS- (CON). In TD, the probe always replaced the CS+; in TS, the probe always replaced the CS-; and in CON, the probe replaced the CS+ and CS- equally often. In the TD group, there were 288 CS+/neutral trials (100% valid), 288 CS-/neutral trials (50% valid & 50% invalid) and 48 neutral/neutral trials (50% valid & 50% invalid). Cue location and probe location were counterbalanced between top and bottom, with the location of the CS+ (top or bottom) equally distributed, and divided over 3 blocks. In the TS group, there were 288 CS-/neutral trials (100% valid), 288 CS+/neutral trials (50% valid & 50% invalid) and 48 neutral/neutral trials (50% valid & 50% invalid). Cue location and probe location were counterbalanced between top and

bottom, with the location of the CS+ (top or bottom) equally distributed, and divided over 3 blocks. Thus, although there were no specific instructions to direct attention towards a certain cue type, the position of the probe (valid or invalid) should theoretically facilitate selective attention allocation. In the CON group, there were 288 CS+/neutral trials (50% valid & 50% invalid), 288 CS-/neutral trials (50% valid & 50% invalid) and 48 neutral/neutral trials (50% valid & 50% invalid). Cue location and probe location were counterbalanced between top and bottom, with the location of the CS+ (top or bottom) equally distributed, and divided over 3 blocks. Since valid and invalid trials were equally distributed for each CS+/neutral and CS-/neutral trials, no particular attention allocation (towards either the CS+ or the CS-) should theoretically be established. Following ABMT, participants underwent a second assessment of attention allocation. The post-ABMT DPT assessment was identical to the Pre-ABMT DPT assessment. Throughout Pre- and Post-DPT assessment phases and the ABMT phase, participants were given no instructions regarding the potential for US shock delivery.

Next, all participants underwent Extinction. Extinction was comprised of 8 CS+ and 8 CS- trials, that were structured the same as CS- trials in terms of trial duration and US expectancy prompts. Since the set of CS+ and CS- presented during ABMT have already functionally undergone fear extinction, the second set of CS+ and CS- that were not presented during ABMT were used for Extinction. Following completion of this phase, participants completed the SUD scale, valence and arousal ratings, and a recognition questionnaire of the CS-US contingency.

On Visit 2, all participants underwent Extinction Retest phase to determine the effectiveness of extinction learning (spontaneous recovery effect) and to investigate if an

unexpected US-only presentation would reinstate conditional responding to CSs (reinstatement effect). Upon arriving to the laboratory, electrodes were placed just as during Visit 1 for physiological recording and they sat in front of the same computer wear the same headphones. Participants were re-familiarized with the US expectancy rating scale and asked to again record their expectancy of receiving the US when prompted by the computer. Next, to habituate reactivity to the startle probe, participants underwent a Habituation phase where they heard 6 trials of the startle probe with only a fixation cross on the computer screen. The participants were told that they would not receive any electrical shock during this phase. Following Habituation, participants provided subjective valence and arousal ratings for each CS.

Next the Extinction Retest phase consisted of 8 presentations of each of the CS_B (CS+) and CS_D (CS-), that were structured the same as CS- trials (during Acquisition) in terms of trial duration, US expectancy prompts, and block randomization. The participants were instructed that they may receive electrical shock delivery to their bicep. Intertrial intervals (ITIs) were interspersed between CS trials. Following 4 CS_B and 4 CS_D presentations, all participants received one presentation of the US during the subsequent ITI. See Figure 3 for a flowchart of the experimental procedures across both visits.

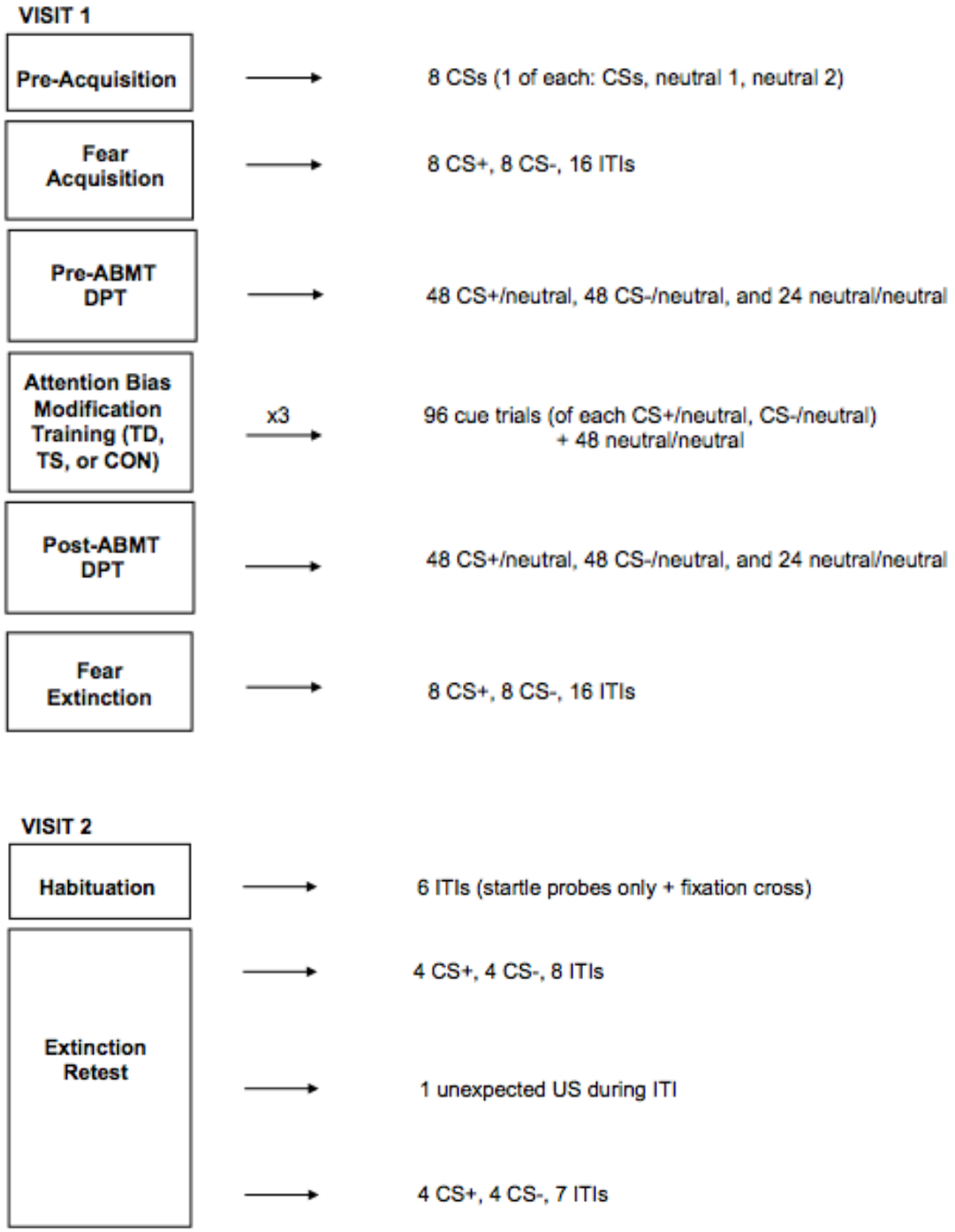

VISIT 1
Pre-Acquisition
8 CSs (1 of each: CSs, neutral 1, neutral 2)
Fear Acquisition
8 CS+, 8 CS-, 16 ITIs
Pre-ABMT DPT
48 CS+/neutral, 48 CS-/neutral, and 24 neutral/neutral
Attention Bias Modification Training (TD, TS, or CON)
x3
96 cue trials (of each CS+/neutral, CS-/neutral) + 48 neutral/neutral
Post-ABMT DPT
48 CS+/neutral, 48 CS-/neutral, and 24 neutral/neutral
Fear Extinction
8 CS+, 8 CS-, 16 ITIs
VISIT 2
Habituation
6 ITIs (startle probes only + fixation cross)
Extinction Retest
4 CS+, 4 CS-, 8 ITIs
1 unexpected US during ITI
4 CS+, 4 CS-, 7 ITIs

Figure 3.

Data Analysis Plan

General

Hypotheses conducted for baseline measures were assessed with one-way Analysis of Variance (ANOVA) to determine if baseline measures (e.g., BDI, muscle stimulation intensity, etc.) were different for participants prior to being assigned to different conditions of attention retraining.

Hypotheses that involved repeated measures across time were assessed with multilevel modeling (MLM). MLM is considered a superior method for analyzing longitudinal data as opposed to ordinary least square regression approaches for several reasons including its improved mechanism for handling missing data (not uncommon for psychophysiological measures) and its ability to handle repeated measures across time (Singer & Willett, 2003). Moreover, MLM divides variance across two levels. Level 1 contains variance attributed to intra-individual changes (e.g., repeated measures across time within the individual) and level 2 contains variation attributed to inter-individual differences (e.g., changes associated with attention training). Test statistics were reported in (1) χ^2 because linear mixed models in the statistical software packaged used for the analysis (i.e., STATA Corp.) cannot compute denominator degrees of freedom and thus cannot report F statistics; or (2) Z statistics for test of simple effects.

Baseline Measures

A one-way ANOVA was conducted for each to determine if baseline measures were different for participants prior to being assigned to different conditions of attention retraining. Baseline measures included: BDI, STAI-Trait Version, BIS, MAAS, ASI, muscle stimulation level, and intensity of muscle stimulation.

Subjective Units of Distress (SUD)

Phase was treated as an intra-individual (Level 1) variable, and CS-US Contingency Awareness during Fear Acquisition and Attention Bias Modification Training were included as inter-individual (Level 2) variables.

Valence and Arousal Ratings

For all analyses, phase was treated as an intra-individual (Level 1) variable, and CS-US Contingency Awareness during Fear Acquisition and Attention Bias Modification Training were included as inter-individual (Level 2) variables. A separate analysis including the aforementioned levels was conducted for valence ratings and arousal ratings.

Attention Bias Modification Training (ABMT)

For all analyses, phase was treated as an intra-individual (Level) variable, and Attention Bias Modification Training was included as an inter-individual (Level 2) variable. The intercept was the first time point (i.e., pre-ABMT DPT). The slope (i.e., change in Response Time) from pre-ABMT DPT to post-ABMT DPT was allowed to vary across participants. Prior to analysis, data was trimmed by excluding response times that were less than 250 ms or greater than 2000 ms. Responses where the probe was incorrectly identified were also excluded. Very few trials were excluded based on the aforementioned criteria.

Conditioning

For all analyses, Trial Order was treated as an intra-individual (Level 1) variable, and CS-US Contingency Awareness during Fear Acquisition and Attention Bias Modification Training were included as inter-individual (Level 2) variables. A separate analysis including the aforementioned levels was conducted for each of the following conditioning

measures: US Expectancy Rating, Acoustic Startle Response, and Skin Conductance Response, for both CS_B (CS+) and CS_D (CS-). Each analysis was first conducted to determine whether a linear or curvilinear model best represented intra-individual change over trials for each conditioning measure. We compared estimated R^2s corresponding to the amount of intra-individual variance in slope accounted for by linear versus curvilinear models as well as deviance statistics (Singer & Willett, 2003). Based on the initial model fitting, a linear or curvilinear model was selected for the analysis of each outcome measure. Then, Level 1 and Level 2 residuals were examined for model outliers and fit, and outliers (3 SD) were treated in the Winsor method and eliminated due to particularly strong and uncorrectable influence. Due to unexpected baseline differences between ABMT conditions, especially in BDI, BIS, and self-reported intensity of the US at Acquisition, the aforementioned variables were initially included as covariates in the model. After comparing variance-covariance structures by using the likelihood ratio test on each conditioning measure, we dropped the aforementioned covariates from the model because they did not explain significantly greater proportion of the variance in the model. CS-US Contingency Awareness from Acquisition was included at Level 2 as a covariate to more fully equate groups on baseline levels of the outcome, minimizes the variance in the outcomes (thus increasing power), and is not subject to potential problems with "regression to the mean" (Tabachnick & Fidell, 2007). Participants who have completed at least Visit 1 (i.e., fear acquisition, attention bias modification training, and fear extinction) were included in the final analysis.

RESULTS

Results: Baseline Measures

For BDI, there was a statistically significant difference between ABMT groups, $F(2,305) = 7.38$, $p<0.01$. A Tukey post-hoc test revealed that BDI was significantly higher in TS, t=3.5, SE=0.73, p<0.01, and CON, t=3.08, SE=0.67, p<0.01 compared to TD.

For STAI-Trait Version, there was no statistical different between ABMT groups, F(2,305)=0.14, p>0.05.

For ASI, there was a statistically significant difference between ABMT groups, F(2,305)=16.45, p<0.01. A Tukey post-hoc test revealed that ASI was significantly lower in TS compared to CON, t=5.43, SE=3.42, p<0.01, and TD, t=4.07, SE=3.21, p<0.01.

For BIS, there was a statistically significant difference between ABMT groups, f(2,298)=4.13, p<0.05. A Tukey post-hoc test revealed that BIS was significantly higher in TS compared to CON, t=2.87, SE=0.58, p<0.05.

For MAAS, there was a statistically significant difference between ABMT groups, f(2,305)=9.34, p<0.01. A Tukey post-hoc test revealed that MAAS was significantly higher in TS compared to CON, t=3.32, SE=2.19, t<0.01, and TD, t=3.94, SE=2.06, p<0.01.

For the level of the muscle stimulation set by participants during the work-up procedure, there was a statistically significant difference between ABMT groups, f(2,305)=4.11, p<0.05. A Tukey post-hoc test revealed that the muscle stimulation level was significantly higher in TS compared to CON, t=2.87, SE=2.26, p<0.05.

For the subjective intensity of the level of muscle stimulation set by each participant during the work-up procedure, there was a statistically significant difference

between ABMT groups, $f(2,298)=8.73$, $p<0.01$. A Tukey post-hoc test revealed that the muscle stimulation level was experienced as significantly more intense in CON compared to TD, $t=3.39$, $SE=2.26$, $t<0.01$, and TS, $t=3.93$, $SE=0.25$, $p<0.01$. See Table 1 for mean and standard deviation of each measure for each ABMT Condition.

Baseline Measures	CON	TD	TS
US level	42.92(9.95)	46.67(12.93)	49.41(20.87)
US intensity	6.33(1.18)	5.47(1.83)	5.34(2.0)
BDI	8.5(4.14)	5.93(2.92)	8(6.72)
STAI	44.58(9.58)	44(6.50)	44.58(10.84)
ASI	70.67(25.93)	65.2(18.4)	52.12(26.81)
BIS	20.58(4.20)	21.47(3.94)	22.25(3.96)
MAAS	54.83(8.59)	54(21.41)	62.12(12.47)

Table 1.

Results: Attention Bias Modification Training

Pre-ABMT DPT

The ABMT Condition x Validity (valid or invalid) x Trial Type (CS_A/neutral, CS_C/neutral, or neutral/neutral) 3-way interaction was not significant, $\chi^2(4)=2.28$, $p>0.05$. The Validity x ABMT Condition interaction, $\chi^2(2)=0.25$, $p>0.05$, and Validity x Trial Type interaction, $\chi2(2)=0.29$, $p>0.05$, were also not significant, demonstrating that there was no difference in response time to probes presented on the same side of the CS and probes presented on the opposite side of the CS, when collapsed across ABMT conditions.

Although the Trial Type x ABMT Condition interaction was significant (see Appendix A for more detail), given that there should be no group differences at this point in the study, the subsequent analyses were conducted by collapsing across ABMT conditions (Figure 4). Namely, the main effect of Trial Type was significant, $\chi 2(2)=41.98$, $p<0.01$, such that Response Time to the CS_A/neutral trials was significantly *greater* compared to both CS_C/neutral trials, $z=6.16$, $SE=4.23$, $p<0.01$, and neutral/neutral trials, $z=3.26$, $SE=5.15$, $p<0.01$. Moreover, Response Time to CS_C/neutral trials was marginally *reduced* compared to neutral/neutral trials, $z=1.81$, $SE=5.15$, $p=0.071$. It is also important to note that despite that no differences in Response Time should be observed between ABMT conditions at this point, there was also a main effect of ABMT Condition, $\chi 2(2)=10.28$, $p<0.01$, such that CON had significantly greater Response Time to all Trial Types relative to TD, $z=2.91$, $SE=31.58$, $p<0.01$, and TS, $z=2.84$, $SE=33.88$, $p<0.01$.

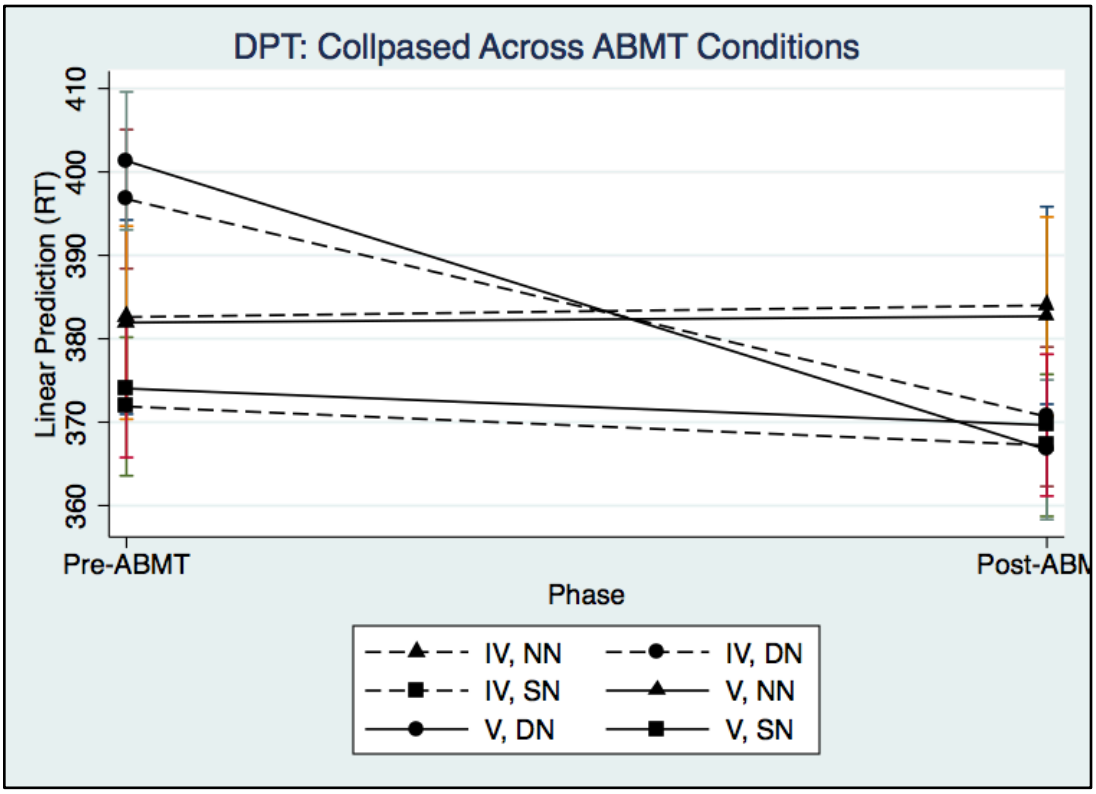


Figure 4.

Pre- to Post-ABMT DPT

The ABMT condition x Trial Type x Validity x Phase 4-way interaction was not significant, $\chi 2(4)=2.01$, $p>0.05$. All 3-way interactions were also not significant.

In TD, change in Response Time from Pre- to Post- ABMT for each Trial Type did not vary as a function of Validity. That being said, as expected, the decline in Response Time to CS_A/neutral trials (when collapsed across Validity) was significantly greater than CS_C/neutral trials, $z=4.22$, $SE=9.10$, $p<0.01$, and neutral/neutral trials, $z=2.56$, $SE=11.04$, $p<0.05$, suggesting that the participants did become faster in responding to the probes following CS_A /neutral trials as a consequence of attention training towards the CS_A. This decline in Response Time to CS_A /neutral trials was significantly greater than zero, $z=4.23$, $SE=10.82$, $p<0.01$ (Figure 5). But, it was not greater than the rate of decline in CON, $z=0.80$,

SE=18.65, p>0.05, but was greater when compared to TS, z=1.97, SE=18.66, p<0.05 (Figure 8). Since decline in RT to CS_A /neutral trials was not significantly different between TD and CON, we were not able to conclude successful attention training towards the CS+ in TD.

In TS, change in Response Time from Pre- to Post- ABMT for each Trial Type did not vary as a function of Validity. Furthermore, the expected reduction in Response Time on the CS-/neutral trials from Pre- to Post-ABMT was not significant, z=1.04, SE=16.01, p>0.05, and did not differ in comparison to TD, z=1.23, SE=21.28, p>0.05, and CON, z=1.64, SE=22.63, p>0.05 (Figure 8). Thus we were not able to conclude that successful attention training towards the CS- was established in TS. That being said, change in Response Time from Pre- to Post-ABMT was significantly different between CS_A/neutral trials and CS_C/neutral trials, z=2.26, SE=10.88, p<0.05, and neutral/neutral trials, z=2.72, SE=13.21, p<0.01, when collapsed across Validity. Namely, there did not appear to be an increase in Response Time for the CS_A/neutral trials, z=0.49, SE=15.60, p>0.05, compared to CS_C/neutral, z=1.63, SE=17.33, p>0.05, and neutral/neutral trials, z=1.09, SE=15.63, p>0.05, although none of the changes were significantly different from zero, but despite the differences being significantly different between Trial Types (Figure 6).

In CON, change in Response Time from Pre- to Post- ABMT for each Trial Type did not vary as a function of Validity. When collapsed across Validity, the decline in Response Time to CS_A/neutral trials relative to neutral/neutral trials was significantly different, z=2.10, SE=13.59, z<0.05, and this decline was marginally different from zero, z=1.88, SE=16.82, p=0.072 (Figure 7).

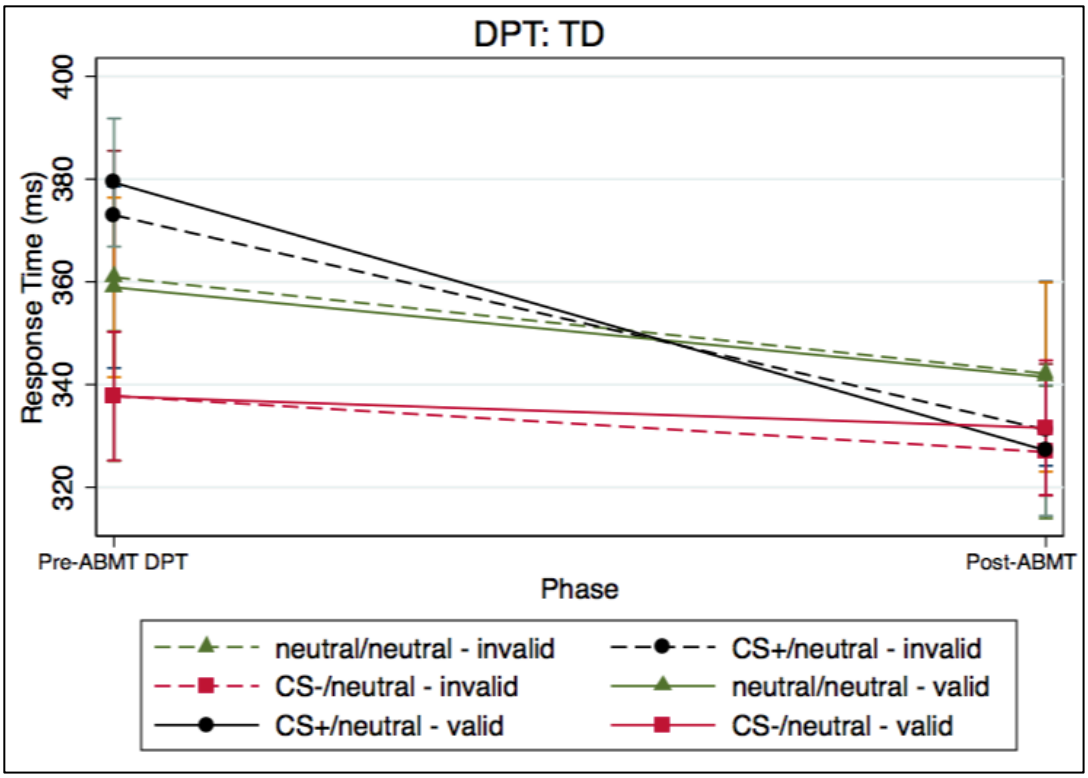


Figure 5.

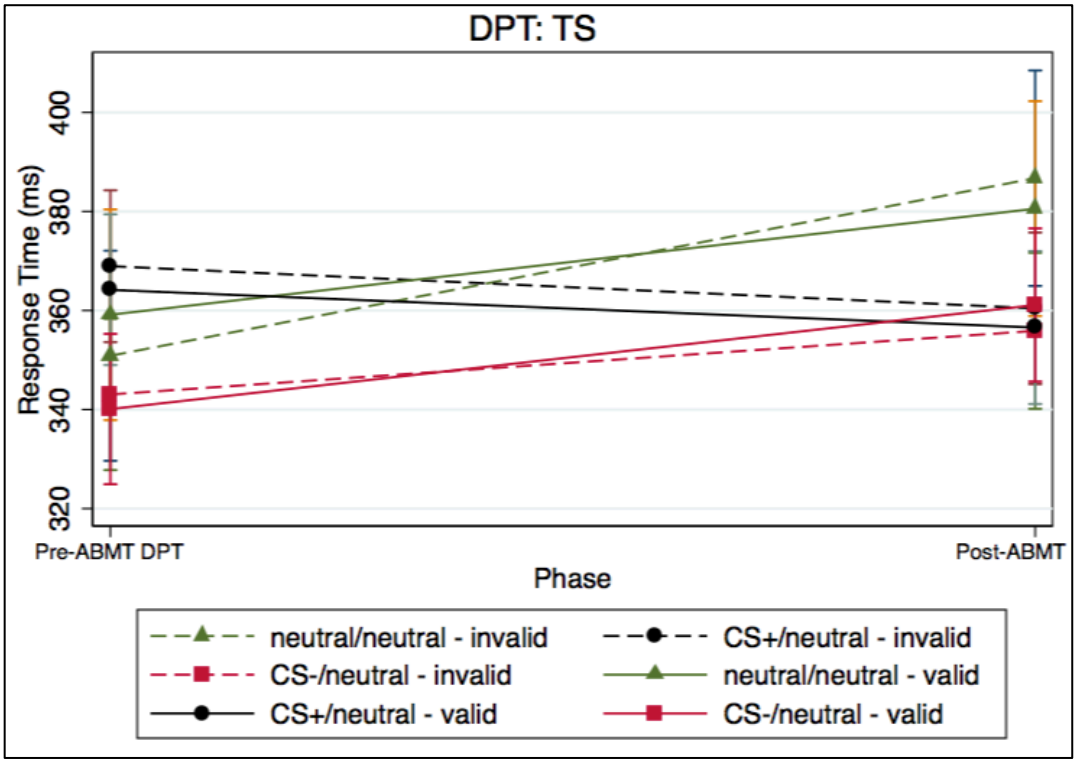


Figure 6.

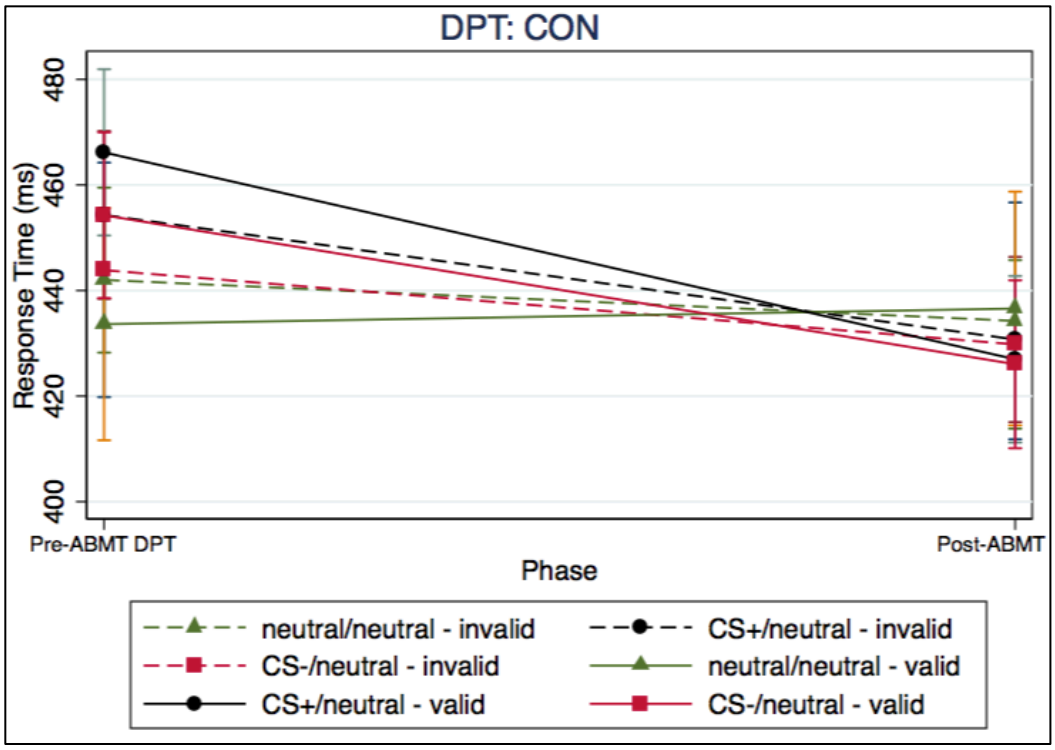


Figure 7.

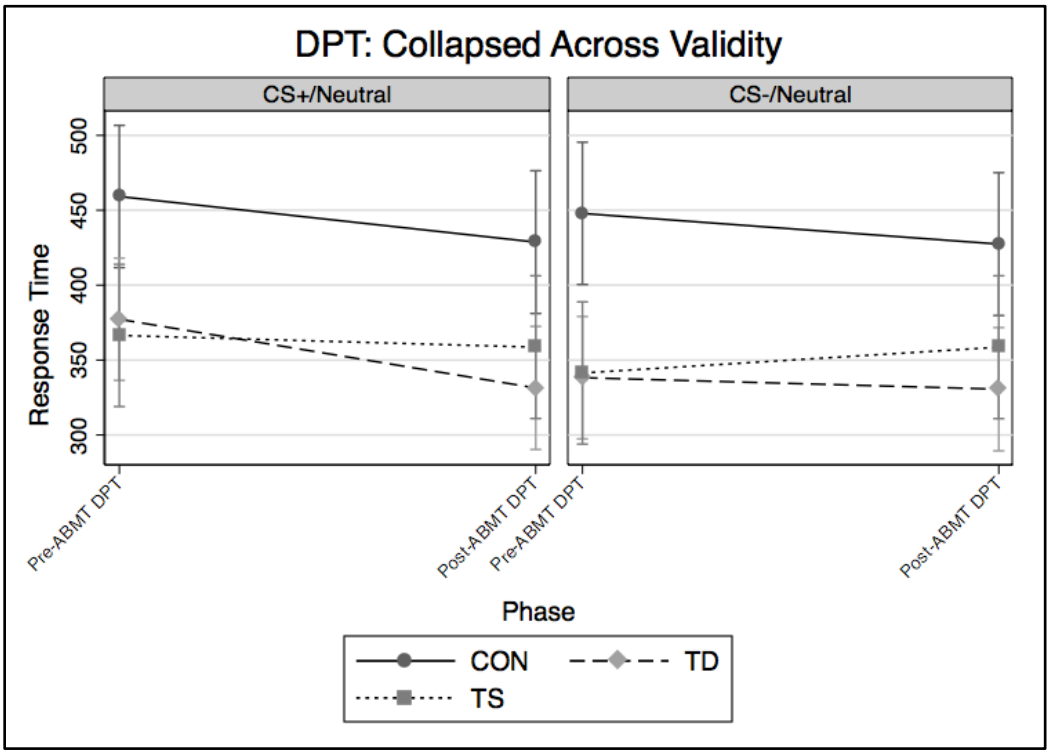


Figure 8.

Results: Fear Conditioning

Fear Acquisition

It was not expected that there would be between-group differences for Visit 1 Fear Acquisition as individuals in different groups were not treated differently until after the Fear Acquisition Phase.

US Expectancy Rating. The intercept represented the first trial of Acquisition. Since the Trial Type x ABMT Condition x Trial Order 3-way interaction was not significant, $\chi^2(42)=24.69$, $p>0.05$, further analyses were conducted by collapsing across ABMT Conditions. As expected, all four CS+ were not significantly different from one another at the first trial of Acquisition (Appendix B). Following Acquisition, at trial 8, EXT CS+ was not significantly different from ABMT CS+, but both were significantly greater than EXT CS- and ABMT CS-. Lastly, at trial 8, ABMT CS- was significantly greater than EXT CS- (Appendix C). Overall, differential fear acquisition was established (Figure 9).

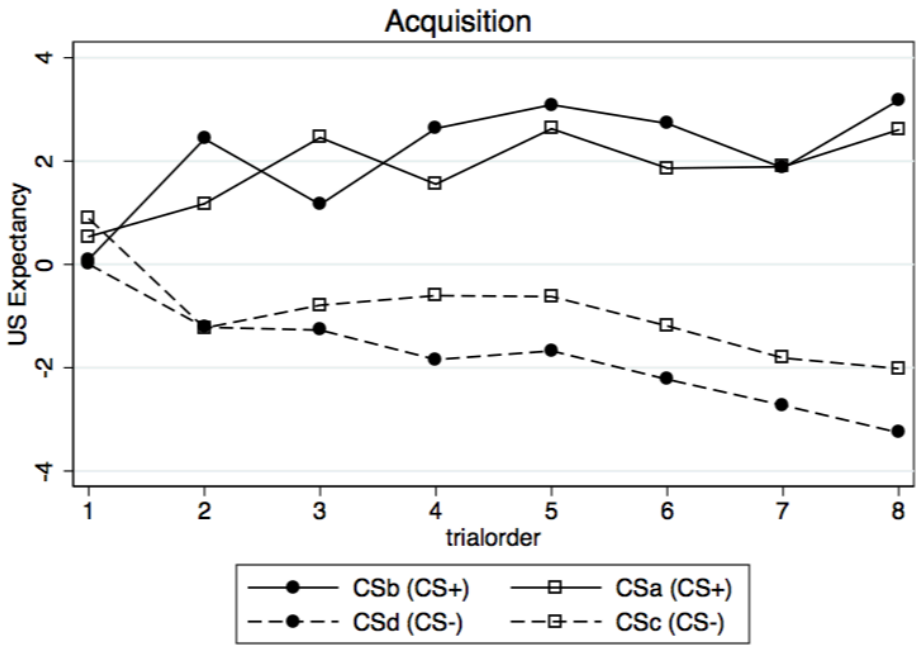


Figure 9.

Skin Conductance Response. The intercept represented the first block of Acquisition. Since the Trial Order x Trial Type x ABMT Condition 3-way interaction was not significant, $\chi^2(18)=12.88$, $p>0.05$, subsequent analyses were conducted with ABMT Conditions collapsed across one another. At the first block of Acquisition, the Trial Type x ABMT Condition interaction was also not significant, $\chi^2(6)=1.50$, $p>0.05$. Contrary to hypothesis, at the first block of Acquisition, CS_A and CS_B (CS+) were significantly greater than CS_C and CS_D (CS-). See Appendix D. That being said, by the second block of Acquisition, pre-existing differences between the different trial types were eliminated. At the last block of Acquisition, skin conductance response to CS_A and CS_B continue to be significantly greater than CS_C and CS_D. Moreover, CS_A and CS_B did not differ from each other, nor did CS_C and CS_D differ from each other (Appendix E). Overall, differential conditioning was established (Figure 10).

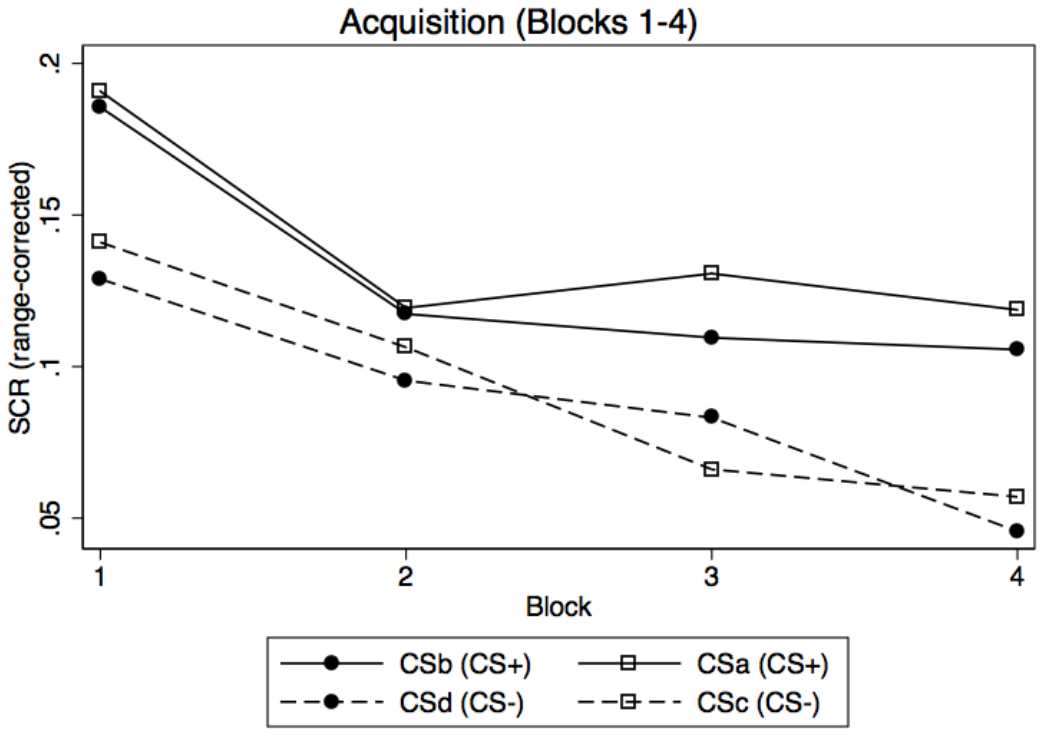


Figure 10.

Acoustic Startle Response. The intercept represented the first block of Acquisition. Since no significant main or interactive effects involving group (i.e., ABMT Condition) was found, further analyses were conducted by collapsing across ABMT Conditions. Contrary to hypothesis, defensiveness to CS_B was significantly greater compared to CS_A, z=3.32, SE=0.07, p<0.01, CS_C, z=4.74, SE=0.07, p<0.01, and CS_D, z=3.15, SE=0.07, p<0.01. At the last block of Acquisition, CS_B was significantly greater than CS_C, z=2.38, SE=0.07, p<0.05, but not CS_D, z=1.56, SE=0.07, p>0.05. CS_A was significantly greater than CS_D, z=2.00, SE=0.07, p<0.05, and CS_C, z=2.82, SE=0.07, p<0.01. As expected, CS_B did not differ from CS_A, z=0.44, SE=0.07, p>0.05, and CS_D also did not differ from CS_C, z=0.82, SE=0.07, p>0.05. Overall, fear acquisition was established to CS_A and CS_B (Figure 11).

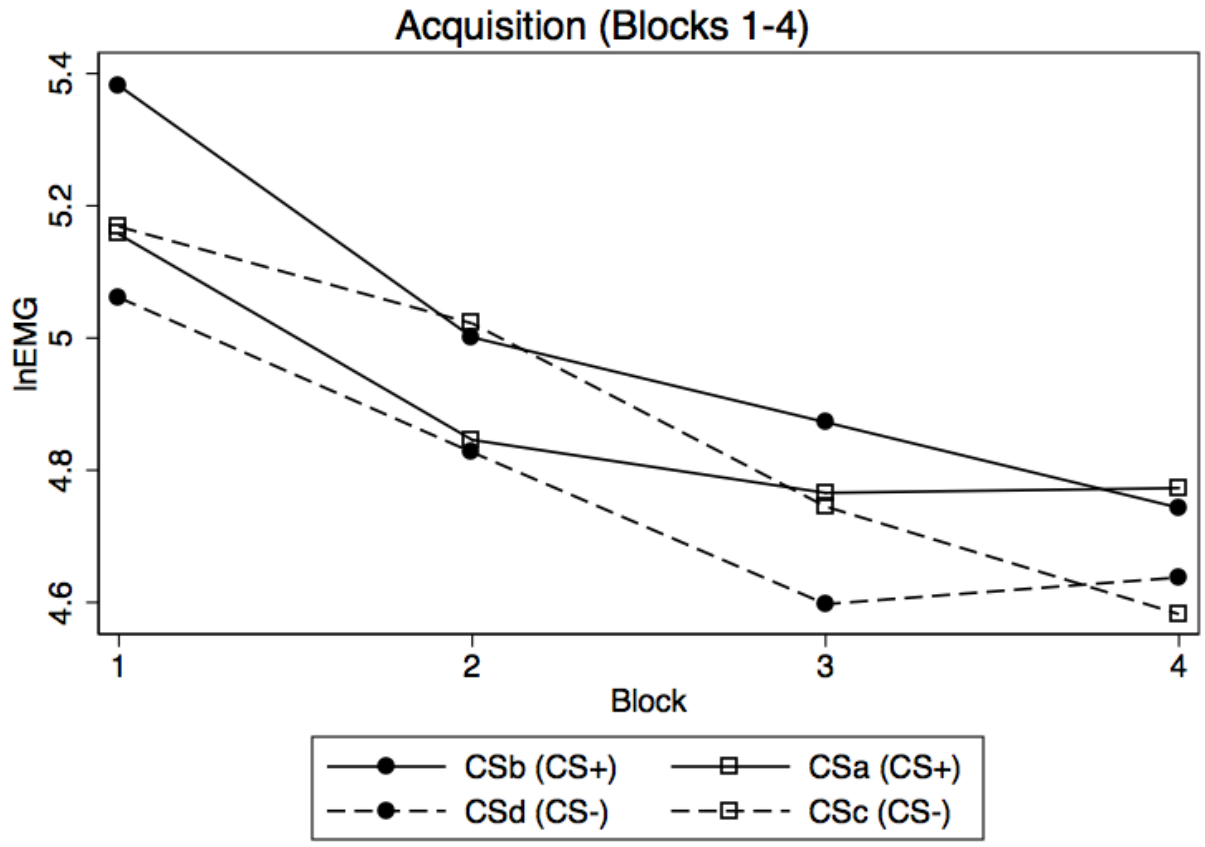


Figure 11.

CS-US Contingency Awareness. This measure was assessed following Fear Acquisition. Chi-square analysis was performed to determine whether or not CS-US Contingency Awareness (yes or no) differed as a function of ABMT Condition. Contrary to expectation, CS-US Contingency Awareness varied as a function of ABMT Condition, $\chi^2(2)=831.99$, $p<0.01$, such that TD, $z=28.15$, $SE=0.04$, $p<0.01$, and TS, $z=19.06$, $SE=0.04$, $p<0.01$, were less likely to be aware of the CS-US Contingency relative to CON.

US Level and Intensity. Contrary to expectation, the intensity of the US during Acquisition was significantly different across ABMT Conditions, $F(2,298)=8.73$, $p<0.01$, such that the US intensity was significantly higher in CON compared to TD, $t=3.39$, $SE=0.26$, $p<0.01$, and TS, $t=9.93$, $SE=0.25$, $p<0.01$. Similarly, the level of the US set during Acquisition also varied as a function of ABMT Condition, $F(2,305)=4.11$, $p<0.05$. Specifically, the US level was significantly higher in TS compared to CON, $t=2.87$, $SE=2.26$, $p<0.05$ (Table 1).

Extinction

The intercept represented the first trial of Extinction and the slope of each conditioning measure across the subsequent 7 trials of Extinction represented the rate of extinction performance. It was hypothesized that reactivity to the CS_B (CS+) will decrease across the 8 trials of Extinction performance for all participants. Furthermore, it was also hypothesized that there would be a significant group x trial order interaction such that attention training towards the CS+ would yield a greater extinction performance (i.e., steeper negative slope) relative to no attention training; and attention training towards the CS- would interfere with extinction performance to the CS_B.

US Expectancy Rating. As expected, there was a significant linear effect of Trial Order demonstrating extinction to the CS_B (CS+), $B=-0.92$, $SE=0.15$, $Z=-6.34$, $p<0.01$, and CS_D (CS-),

B=-0.25, SE=0.10, Z=-2.46, p<0.05. Although the ABMT Condition x Trial Order interaction was significant to the CS+, $\chi^2(2)=6.59$, p<0.05, contrary to my hypothesis, attention training towards the CS+ (TD) interfered with the rate of extinction performance compared to attention training towards the CS- (TS) , z=-2.34, SE=0.19, p<0.05, and the no training group (CON), z=2.06, SE=0.20, p<0.05. The hypothesis that attention training towards the CS- would interfere with the rate of extinction performance was not established, z=-0.15, SE=0.20, p>0.05. For the CS_D (CS-), US expectancy also decreased across trials, z-2.46, SE=0.10, p<0.014, but it did not vary by ABMT Condition, $\chi^2(2)=1.10$, p>0.05. Overall, extinction was established (Figure 12).

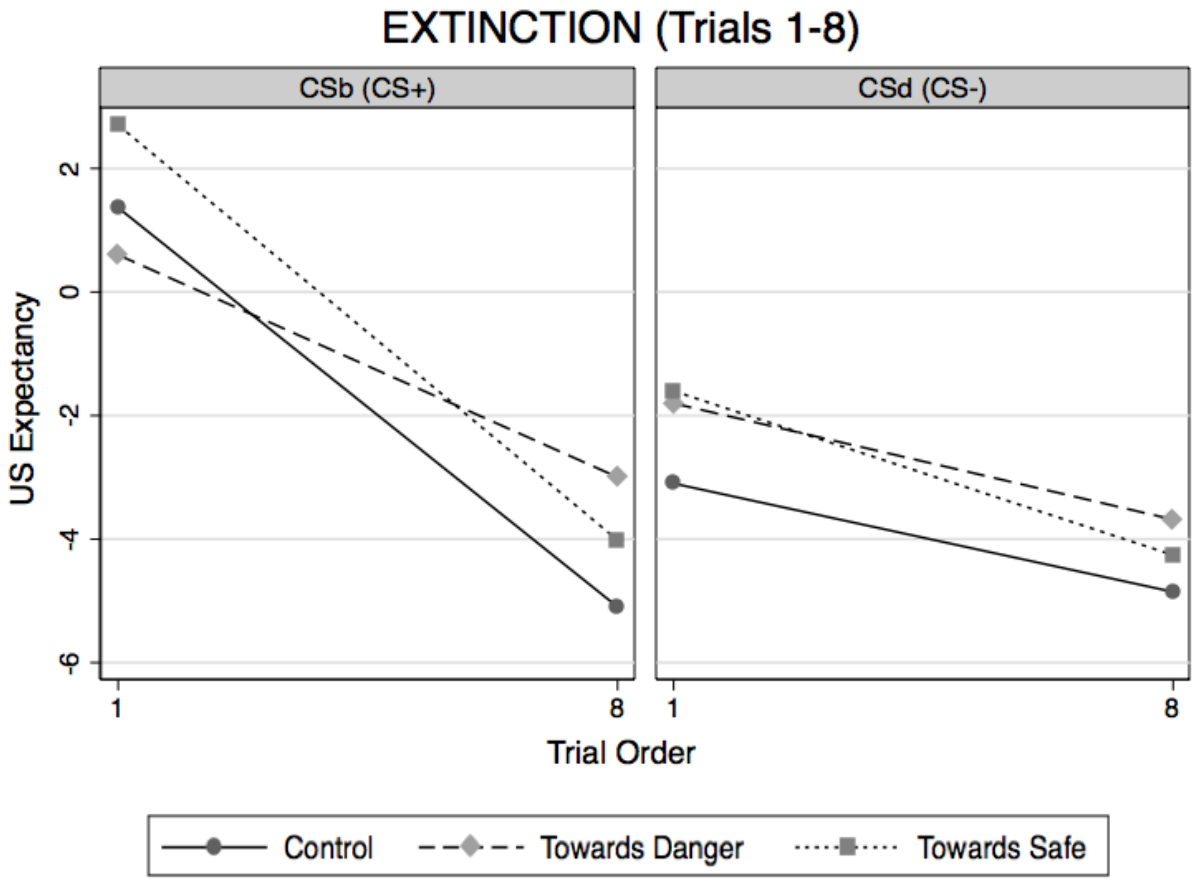


Figure 12.

Skin Conductance Response. As expected, the intercept was significantly to the CS_B (CS+) compared to the CS_D (CS-), z=-7.05, SE=0.018, p<0.01, demonstrating successful differential acquisition that did not differ as a function of ABMT Condition, z=-0.88, SE=0.43, p>0.05. As expected, there was a significant linear effect of Trial Order demonstrating extinction to the CS_B, z=-2.68, SE=0.014, p<0.01, and CS_D, z=-2.37, SE=0.014, p<0.05. Although it was hypothesized that the rate of extinction performance would vary as a function of attention training, it was not supported by a significant ABMT Condition x Trial Order interaction, $\chi^2(2)=2.56$, p>0.05. Given *a priori* hypothesis, pairwise comparisons across the three ABMT Conditions on rate of extinction were conducted regardless. Contrary to our hypothesis, not only was the decrease in arousal during extinction to the CS_B (CS+) facilitated in TS relative to CON, z=-2.16, SE=0.01, p<0.05, this facilitation was not observed between TD and CON, z=-1.16, SE=0.01, p>0.05. For the CS_D (CS-), decline in responding did not vary as a function of ABMT Condition. Overall, extinction was established in all three groups (Figure 13).

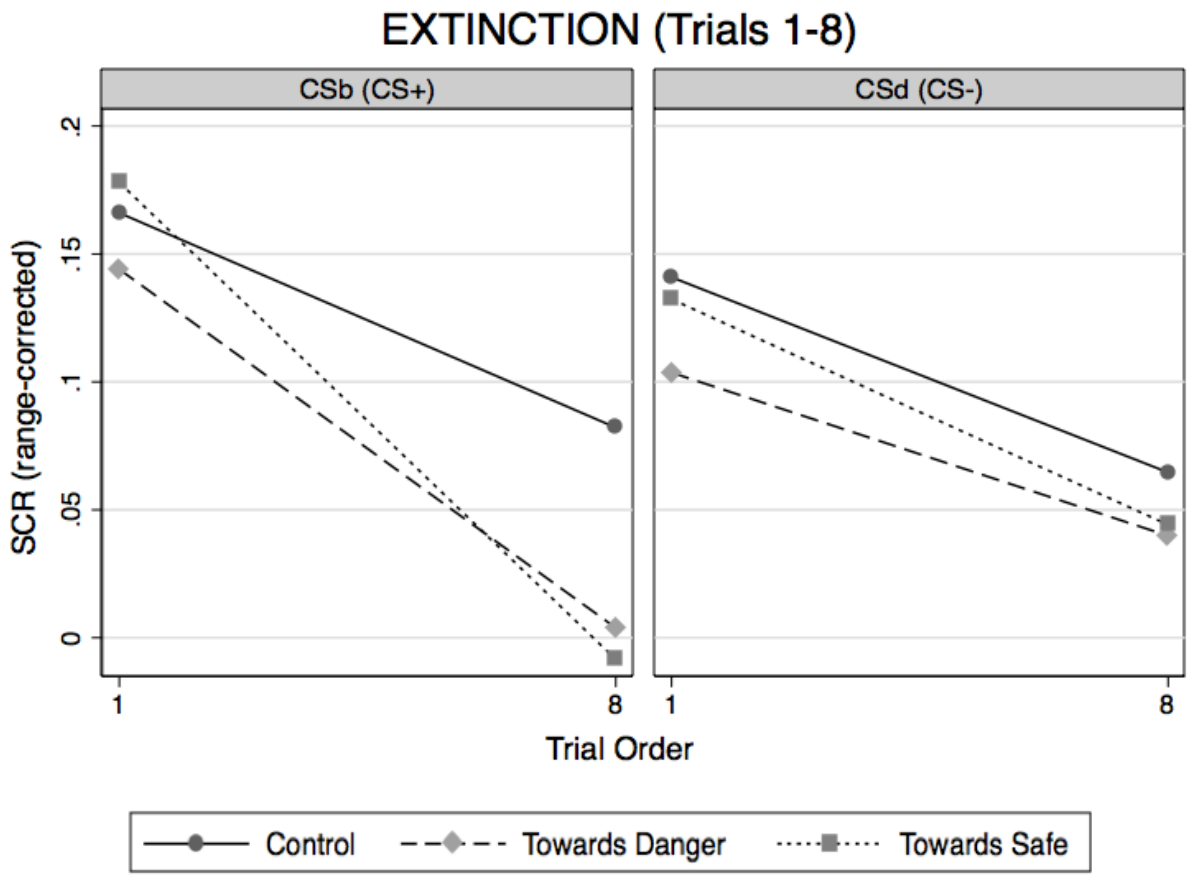


Figure 13.

Acoustic Startle Response. Contrary to hypothesis, the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=3.77$, $p>0.05$, despite a general decline in defensiveness to the CS_B (CS+) across Extinction, B=-0.07, SE=0.03, Z=-2.53, $p<0.05$. Also contrary to our hypothesis, extinction was not facilitated in TD, z=-1.57, SE=0.04, $p>0.05$; instead, extinction was marginally facilitated in TS relative to CON, z=-1.86, SE=0.04, p=0.064. For the CS_D (CS-), the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=3.30$, $p>0.05$, despite an overall decline in defensiveness when collapsed across ABMT Conditions, B=-0.08, SE=0.03, z=13.33, $p<0.01$. Contrary to hypothesis the

decline in defensiveness to the CS_D (CS-) was marginally greater in TS relative to CON, z=-1.80, SE=0.03, p=0.072. Overall, extinction was established (Figure 14).

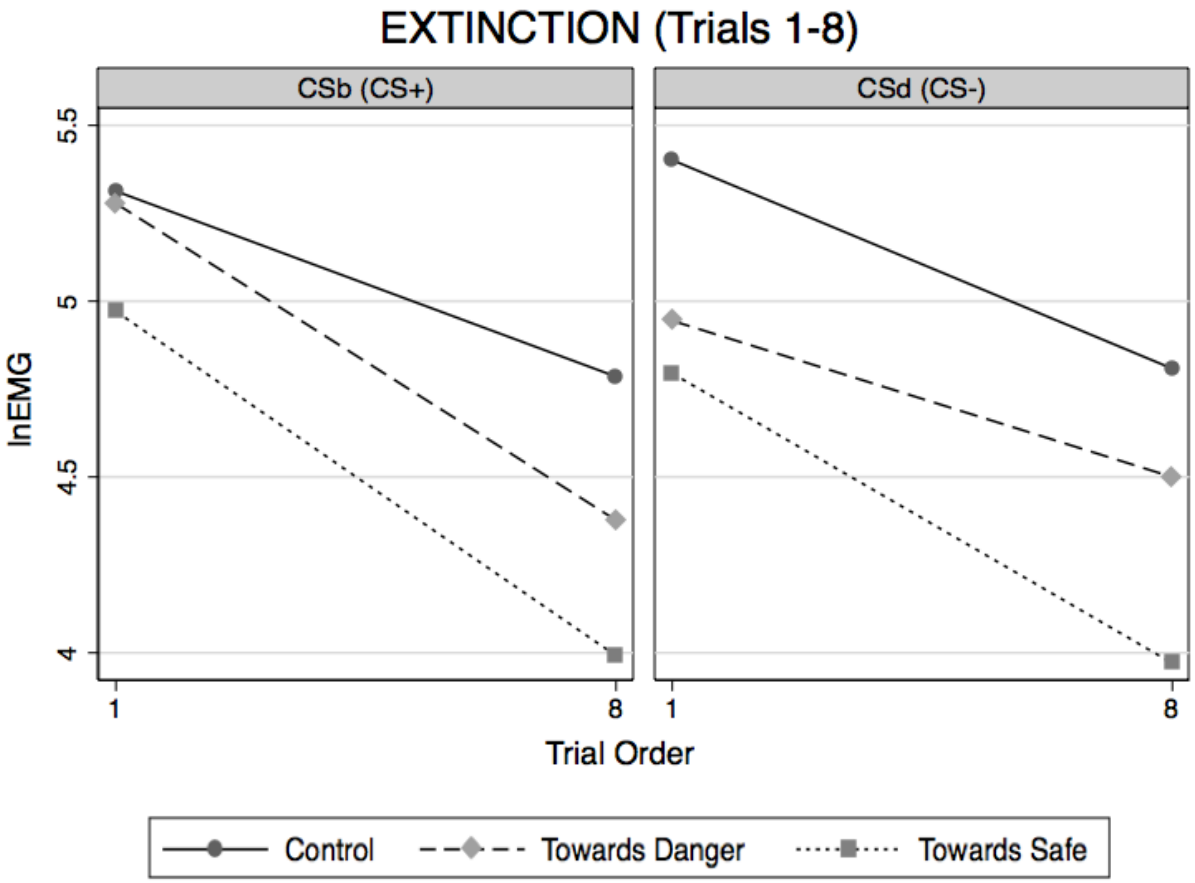


Figure 14.

Extinction Retest (Spontaneous Recovery Effect)

The spontaneous recovery effect represents the degree to which reactivity to a previously extinguished CS (CS+ and CS-) is recovered following the passage of time. Approximately 7 days passed in between Extinction (Visit 1) and Extinction Retest (Visit 2). More importantly, recovery of the conditioned response at Extinction Retest is often considered as a more accurate index of extinction *learning* (from Visit 1) than the performance during Extinction itself (Craske et al., 2008). The Extinction phase and Retest of Extinction phase were combined in the current model, so that the change in reactivity to

the CS from the last trial of Extinction (trial 8) to the first trial of Extinction Retest could be examined. It was hypothesized that the conditioned response to the CS_B (CS+) following extinction training at the first trial of Extinction Retest would vary as a function of ABMT Condition, such that greater extinction learning would be observed in TD relative to CON and TS.

US Expectancy Rating. As expected, US Expectancy to the CS_B (CS+), B=5.18, SE=1.05, Z=4.96, p<0.01, and to the CS_D (CS-), B=1.96, SE=0.69, Z=2.82, p<0.01, were recovered, and greater recovery was observed in the CS_B (CS+) relative to the CS_D (CS-), z=-3.35, SE=0.67, p<0.01. Although the ABMT Condition x Trial Order interaction did not reach statistical significance, $\chi^2(2)=4.85$, p=0.082, based on *a priori* hypothesis of the differential effect of attention training on Extinction Retest, pairwise comparisons across the three ABMT Conditions on Extinction Retest were conducted regardless. Partially consistent with our hypothesis, less US expectancy to the CS_B (CS+) was recovered in TD relative to CON, z=1.95, SE=1.42, p=0.052, and to TS, z=1.84, SE=1.33, p=0.065; whereas no such reduction was observed between TS and CON, z=0.21, SE=1.40, p>.05. On the other hand, Recovery of US Expectancy to the CS_D (CS-) did not differ as a function of ABMT Condition, $\chi^2(2)=0.03$, p>0.05. See Figure 15.

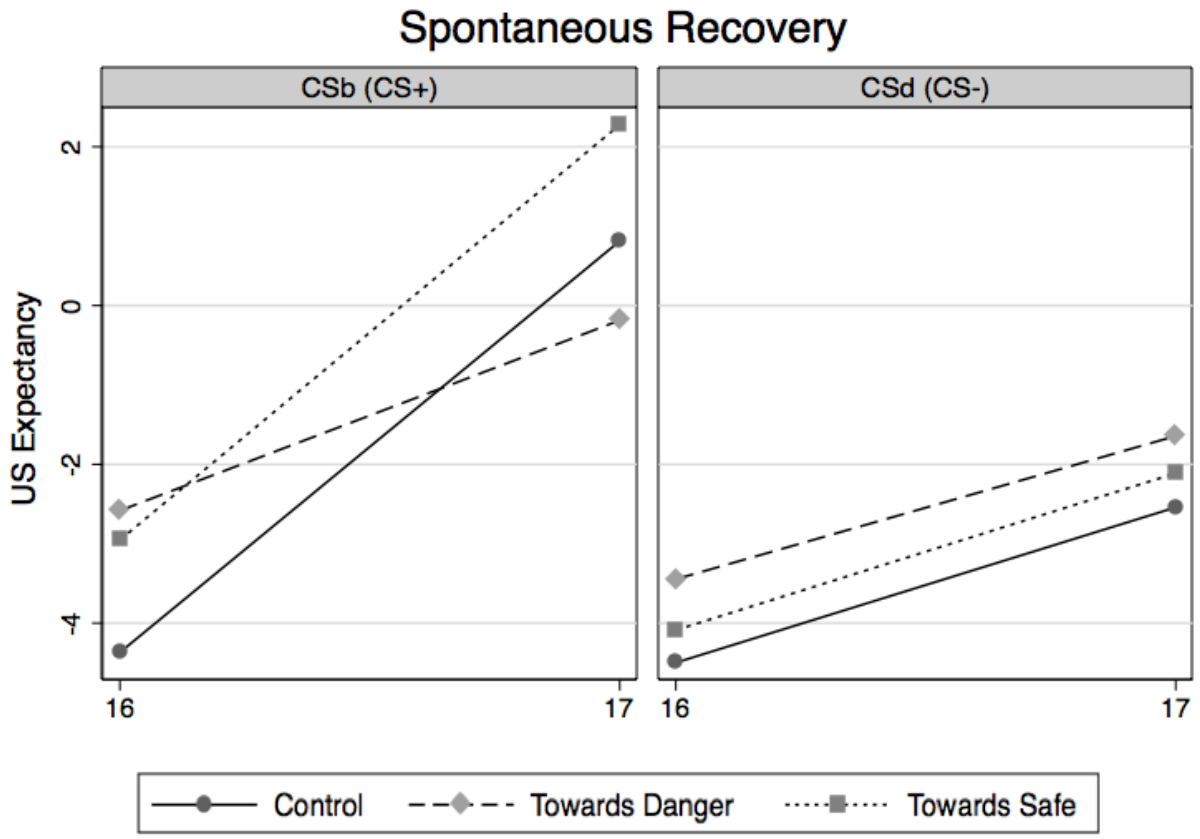


Figure 15. Note: 16=Last Trial of Extinction (Visit 1), 17=First Extinction Retest (Visit 2)

Skin Conductance Response. The ABMT Condition x Trial Type x Trial Order 3-way interaction was not significant, $\chi^2(2)=2.97$, $p>0.05$. The Trial Order x Trial Type interaction was not significant, $\chi^2(1)=0.54$, $p>0.05$. The ABMT Condition x Trial Order interaction, however, was significant, $\chi^2(2)=9.06$, $p<0.05$, such that there was greater arousal in TD, $z=13.43$, $SE=0.06$, $p<0.01$, and TS, $z=-2.16$, $SE=0.06$, $p<0.05$, compared to CON to the CS_B (CS+). For the CS_D (CS-), although the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=1.67$, $p>0.05$, given *a priori* hypotheses about the effect of attention training on extinction-related processes, pairwise comparisons across the three ABMT Conditions at Extinction Retest was conducted. Consistent with our hypothesis, greater

arousal to the CS_D (CS-) was trending in TS compared to CON, t=-1.75, SE=0.09, p=0.083. See Figure 16.

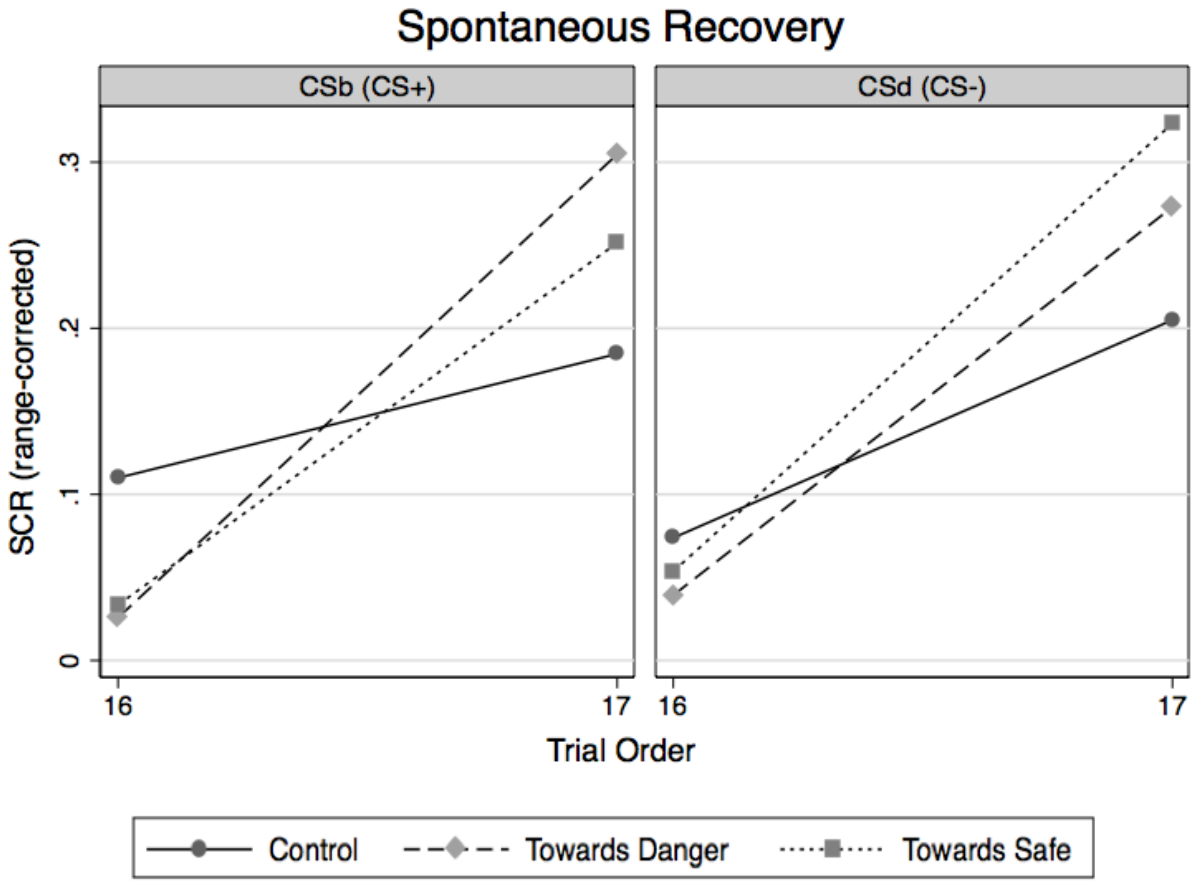


Figure 15. Note: 16=Last Trial of Extinction (Visit 1), 17=First Extinction Retest (Visit 2)

Acoustic Startle Response. The ABMT Condition x Trial Type x Trial Order 3-way interaction was not significant, $\chi^2(2)=0.05$, $p>0.05$. The Trial Order x Trial Type interaction was not significant, $\chi^2(1)=0.81$, $p>0.05$. The ABMT Condition x Trial Order interaction, however, was significant, $\chi^2(2)=7.96$, $p<0.05$. Contrary to expectation, there was greater defensiveness in TS relative to CON, z=-2.21, SE=0.42, $p<0.05$, and marginally greater defensiveness in TD relative to CON, z=-1.94, SE=0.41, p=0.053, to the CS_B (CS+). Similarly,

there was greater defensiveness in TS, z=-2.58, SE=0.35, p<0.05, and TD, z=-2.00, SE=0.34, p<0.05, relative to CON to the CS_D (CS-) as well. See Figure 17.

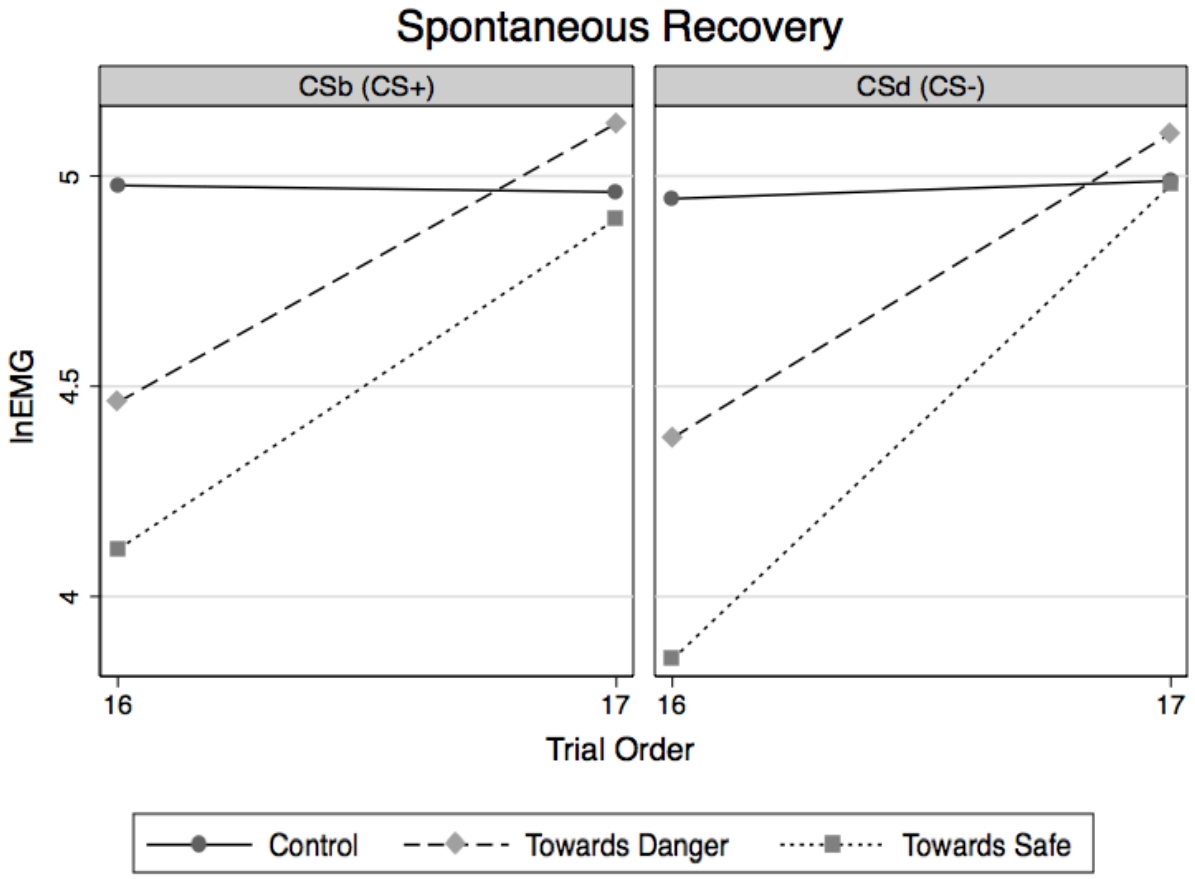


Figure 15. Note: 16=Last Trial of Extinction (Visit 1), 17=First Extinction Retest (Visit 2)

Extinction Retest (Reinstatement Effect)

It is operationalized as the reinstatement of reactivity to a previously extinguished CS (CS+ and CS-) following an unexpected delivery of the US (in between Trial 4 and Trial 5 during Extinction Retest) that is not paired with either the CS+ (CS_B) or the CS- (CS_D). It was hypothesized that reinstatement of the conditioned response to both CSs will vary as a function of attention training, such that reinstatement would be reduced in TD and facilitated in TS.

US Expectancy Rating. As expected, US Expectancy was marginally reinstated to the CS_B (CS+), z=1.92, SE=0.99, p=0.055, and to the CS_D (CS-), z=2.94, SE=0.81, p<0.05. For the CS_B (CS+), although the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=3.88$, p>0.05, based on *a priori* hypothesis of the differential effect of attention training on the reinstatement effect, post-hoc t tests showed that US Expectancy in TD showed an increasing trend, z=1.79, SE=1.28, p=0.073 , relative to TS. For the CS_D (CS-), although the reinstatement effect was significant when collapsed across all 3 attention training groups and did not yield a significant ABMT Condition x Trial Order interaction, $\chi^2(2)=3.33$, p>0.05, follow-up contrasts (reinstatement effect for each ABMT Condition) showed that US Expectancy in TD, $\chi^2(1)=1.41$, p>0.05, did not increase, whereas it did in TS, $\chi^2(1)=12.14$, p>0.01, and CON, $\chi^2(1)=8.67$, p>0.01. See Figure 18.

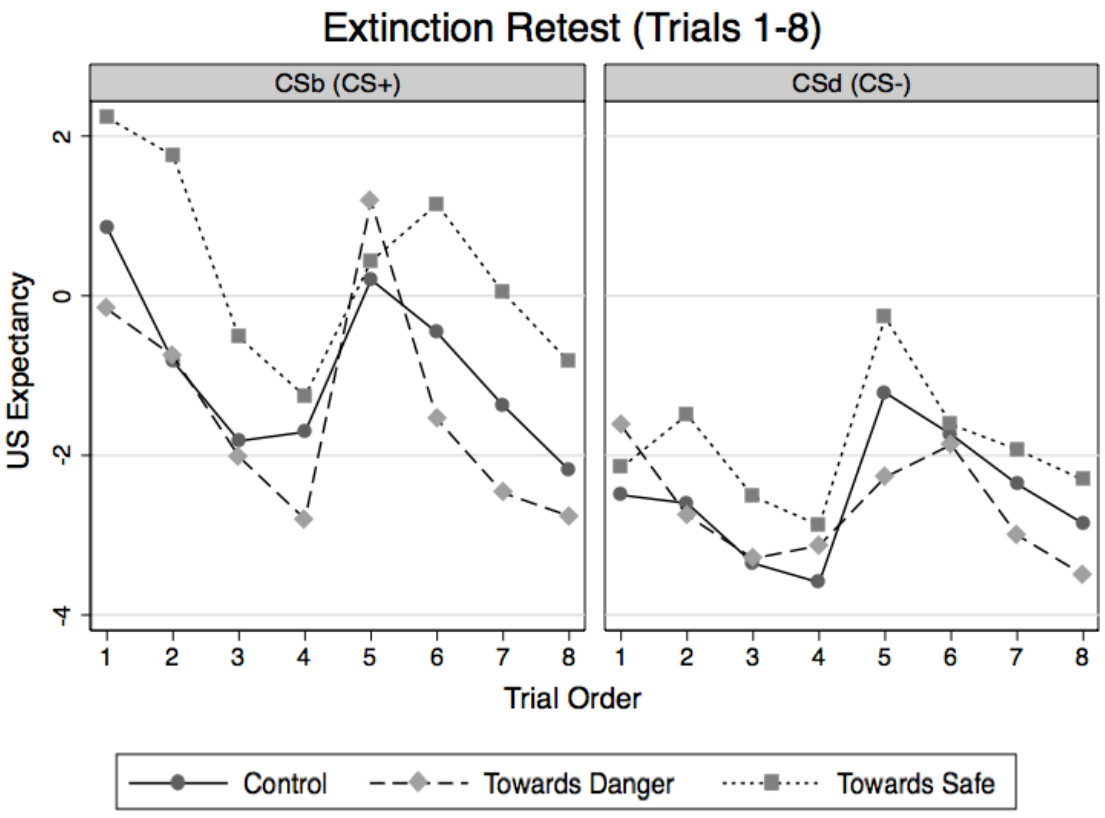


Figure 18. Note: Uncued shock delivered in between Trial 4 and Trial 5.

Skin Conductance Response. For the CS_B (CS+), as expected, the ABMT Condition x Trial Order interaction was significant, $\chi^2(2)=15.10$, $p<0.01$. Notably, arousal to the CS_B (CS+) was marginally greater in TS compared to TD, $z=1.77$, $SE=0.07$, $p=0.077$. For the CS_D (CS-), the ABMT Condition x Trial order interaction was not significant, $\chi^2(2)=4.06$, $p>0.05$. Furthermore, the reinstatement effect in TD and TS were not significantly different from each other on the CS_D (CS-), $z=-0.63$, $SE=0.05$, $p>0.05$. Pairwise comparisons to CON on the CS_B and CS_D were conducted, but it was determined that such comparisons would be invalid. For unknown reasons, aberrations occurred in CON such that they did not show any reinstatement of arousal to the CSs, despite clear reinstatement as evidenced by other measures. See Figure 19.

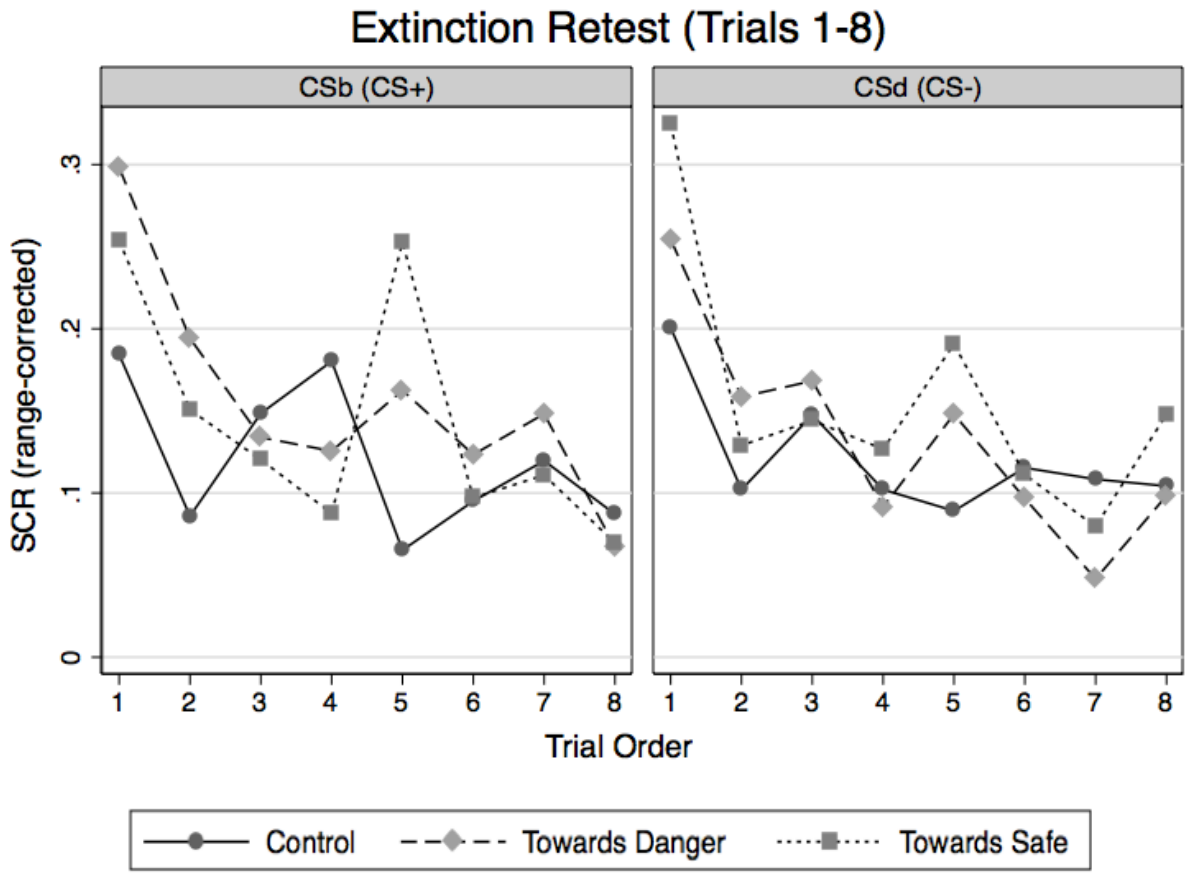


Figure 19. Note: Uncued shock delivered in between Trial 4 and Trial 5.

Acoustic Startle Response. Contrary to expectation, the ABMT Condition x Trial Order interaction was not significant for the CS_B (CS+), $\chi^2(2)=2.69$, $p>0.05$. Given *a priori* hypothesis about the differential effect of attention training on the reinstatement, effect, post hoc t tests were conducted. Contrary to our hypothesis, attention training neither enhanced nor impaired reinstatement of defensiveness to the CS_B (CS+) relative to no training (TD: z=-0.98, SE=0.26, p>0.05; TS: z=-1.64, SE=0.26, p>0.05). For the CS_D (CS-), the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=2.99$, $p>0.05$. Based on post hoc pairwise comparisons, there was a trend for a greater defensiveness to CS_D (CS-) in TD relative to CON, z=-1.73, SE=0.23, p=0.084. See Figure 20.

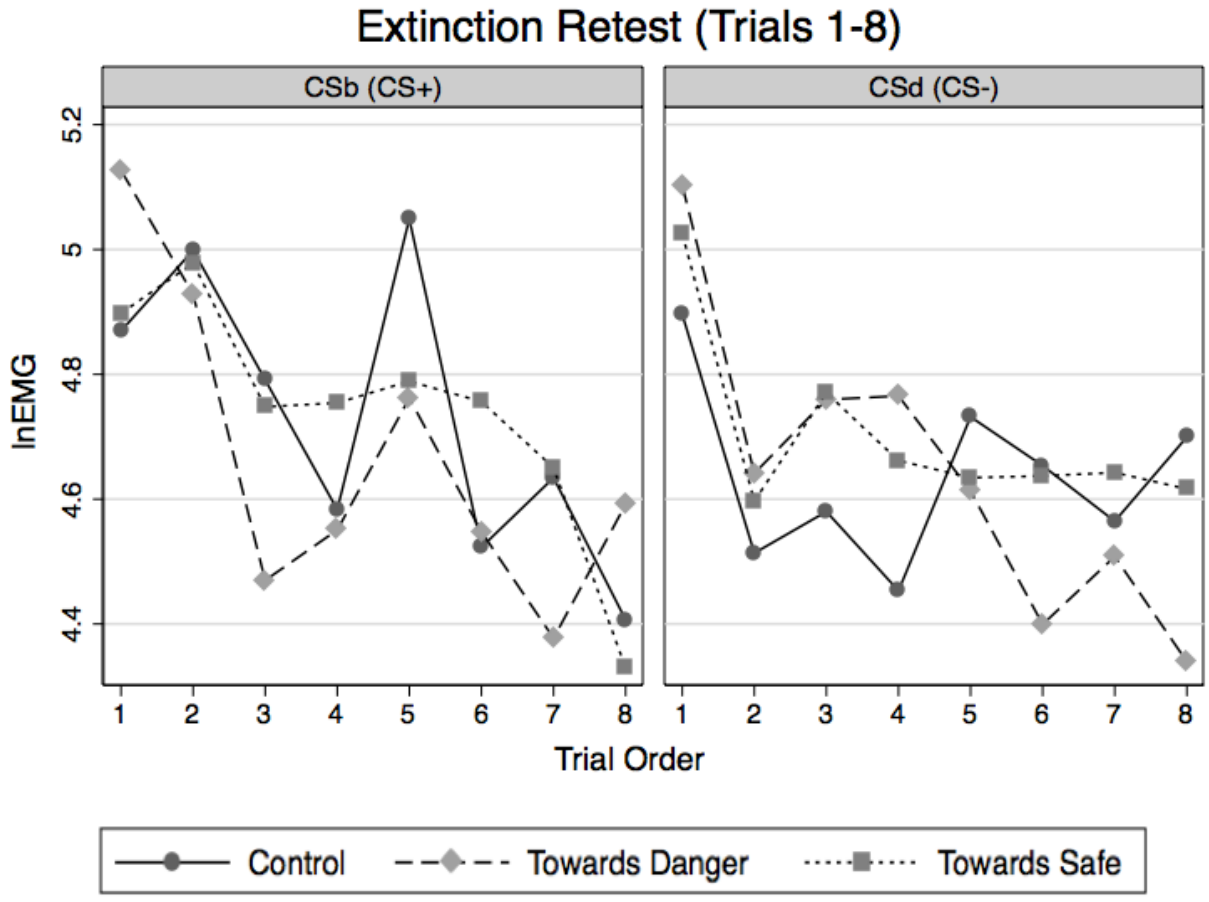


Figure 20. Note: Uncued shock delivered in between Trial 4 and Trial 5.

Extinction Retest (Re-Extinction Effect)

It is operationalized as the four trials following the delivery of the unpaired US (trials 5-8) during the Extinction Retest phase. The intercept is the first trial after US delivery and the slope represents the rate of re-extinction.

US Expectancy Rating. As expected, US Expectancy showed a linear decrease from Trial 5 to Trial 8 to the CS_B (CS+), z=-2.91, SE=0.28, p<0.01. Follow-up pairwise comparisons showed that the reduction to US Expectancy across trials was significantly smaller in TS relative to TD, z=2.20, SE=0.36, p<0.05, although this difference was not big enough to yield a significant ABMT Condition x Trial Order interaction, $\chi^2(2)=4.93$, p=0.08. To the CS_D (CS-), the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=0.38$, p>0.05. Collapsed across ABMT Conditions, US expectancy to the CS_D (CS-) continued to decline, B=-0.55, SE=0.21, Z=-2.7, p<0.01. See Figure 18.

Skin Conductance Response. For the CS_B (CS+), there was a significant ABMT Condition x Trial Order interaction, $\chi^2(2)=8.38$, p<0.05, such that the decline in arousal was greater in TS relative to CON, z=-2.89, SE=0.02, p<0.01. However, due to an unexplained aberration in reinstatement in CON, group comparisons against CON were difficult to interpret. Between TD and TS only, decline in arousal to the CS_B (CS+) did not differ, z=-1.48, SE=0.02, p>0.05. To the CS_D (CS-), the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=1.12$, p>0.05. See Figure 19.

Acoustic Startle Response. For the CS_B (CS+), the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=1.38$, p>0.05, despite an overall decline in defensiveness when collapsed across ABMT Conditions, B=-0.18, SE=0.08, z=-2.27, p<0.05. All pairwise comparisons of rate of re-extinction across ABMT Conditions were also not

significant. For the CS_D (CS-), the ABMT Condition x Trial Order interaction was also not significant, $\chi^2(2)=0.59$, $p>0.05$. Moreover, when collapsed across ABMT conditions, there was no decline in defensiveness on the re-extinction effect, B=-0.01, SE=0.07, z=-0.15, $p>0.05$. See Figure 20.

Results: Subject Units of Discomfort (SUD)

As expected, the ABMT Condition x Phase interaction was not significant, $\chi^2(12)=8.58$, $p>0.05$, indicating that SUD ratings did not vary as a function of attention training condition across the experiment. Pairwise comparisons at each phase to assess whether or not SUD ratings varied across ABMT conditions were conducted. As expected, at Baseline, ABMT groups did not differ in SUD; at Pre-Acquisition, SUD was significantly lower in TS relative to CON, z=2.58, SE=7.62, $p<0.05$, and TD, z=2.12, SE=7.17, $p<0.05$; at Post-Acquisition, SUD was marginally lower in TS compared to CON, z=1.80, SE=7.59, $p=0.072$. There was no ABMT group difference at Post-ABMT and Post-Extinction. At Pre-Reinstatement, SUD was significantly lower in TS compared to CON, p=2.04, SE=7.63, $p<0.05$. Similarly, at Post-Reinstatement, SUD continued to be significantly lower in TS compared to CON, z=2.38, SE=7.60, $p<0.05$ (Figure 21).

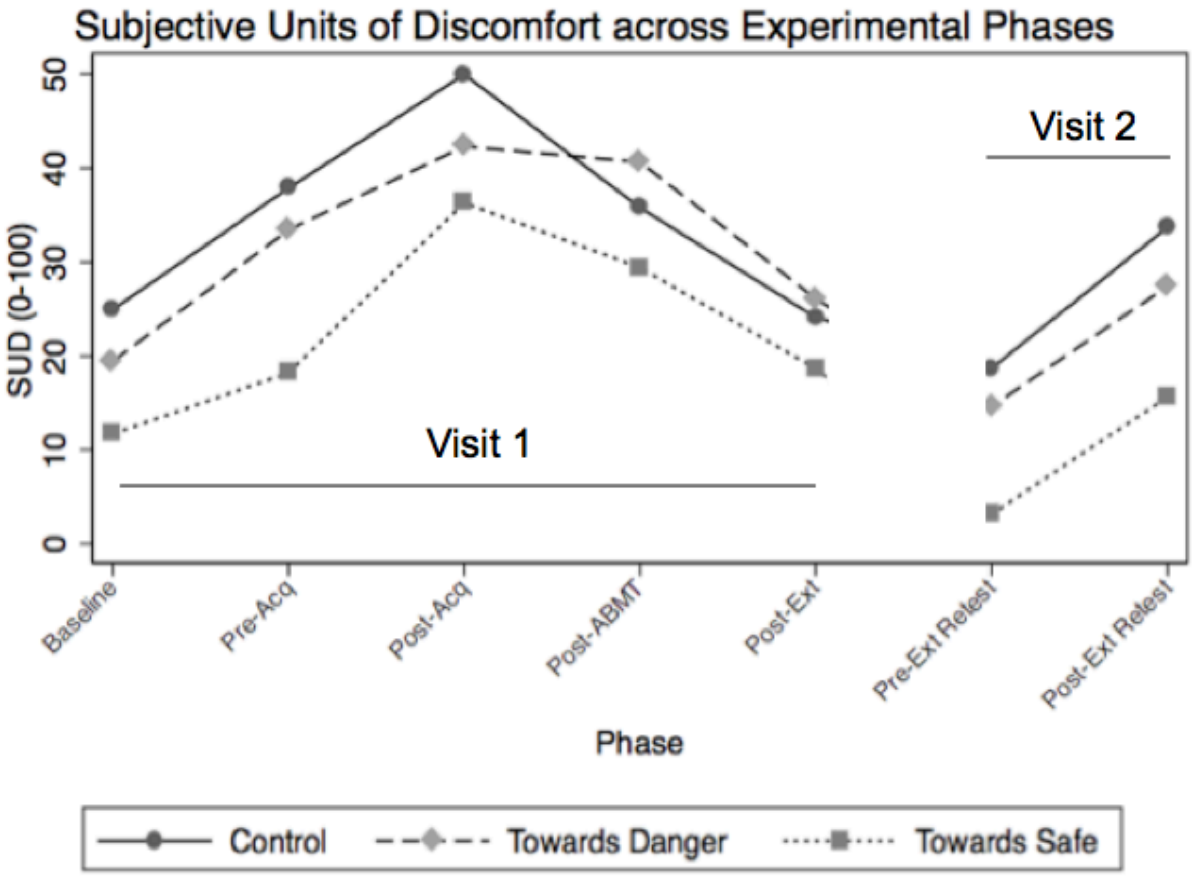


Figure 21.

Results: Self-Reported Valence and Arousal to CSs

For self-reported arousal and valence ratings, the crucial Phase x Trial Type interaction was significant, $\chi^2(15)=160.36$, $p<0.01$, and, $\chi^2(15)=143.52$, $p<0.01$, respectively. But, the Phase x Trial Type x ABMT Condition 3-way interaction was not significant, $\chi^2(30)=31.36$, $p>0.05$, and, $\chi^2(30)=35.74$, $p>0.05$, for arousal and valence, respectively, suggesting that changes in valence and arousal across experimental phases did not differ as a function of ABMT condition. However, given *a priori* hypotheses, specific pairwise comparisons were followed up at each phase.

Pre-Acquisition

It was not expected that there would be between group differences at Pre-Acquisition as individuals in different groups were not treated differently until after Acquisition. Consistent with this, no significant main or interactive effects involving group were found for self-reported arousal and valence ratings.

Arousal Rating. As expected, the ABMT Condition x Trial Type interaction was not significant, $\chi^2(6)=3.04$, $p>0.05$. In other words, all four CSs did not differ in self-reported arousal at the beginning of the experiment (Figure 22).

Valence Rating. As expected, the ABMT Condition x Trial Type interaction was not significant, $\chi^2(6)=3.04$, $p>0.05$. In other words, all four CSs did not differ in self-reported arousal at the beginning of the experiment. (Figure 23).

Post-Acquisition

It was not expected that there would be between group differences at Post-Acquisition as individuals in different groups were not treated differently until after Acquisition. The results were not consistent with this.

Arousal Rating. As expected, the ABMT Condition x Phase x Trial Type 3-way interaction was significant, $\chi^2(2)=8.84$, $p<0.05$. For CS+, the ABMT Condition x Phase interaction was significant, $\chi^2(2)=7.37$, $p<0.05$, suggesting that acquisition of self-reported arousal to the CS+ differed as a function of ABMT Condition, despite the fact that the attention training manipulation has not been introduced yet. Namely, arousal to the CS+ was significantly greater in CON compared to TD, $z=2.70$, $SE=0.96$, $p<0.05$. For the CS-, the ABMT Condition x Phase interaction was not significant, $\chi^2(2)=1.06$, $p>0.05$, suggesting that not only did arousal to the CS- not change from pre- to post-acquisition, $B=-0.75$, $SE=0.54$,

z=-1.38, p>0.05, it also did not differ as a function of ABMT Condition (Figure 22).

Valence Rating. As expected, the ABMT Condition x Phase x Trial Type 3-way interaction was significant, $\chi^2(2)=10.51$, p<0.01. For CS+, the ABMT Condition x Trial Order interaction was significant, $\chi^2(2)=11.94$, p<0.01, suggesting that acquisition of self-reported valence to the CS+ differed as a function of ABMT Condition, despite the fact that the attention training manipulation has not been introduced yet. Namely, the increase in valence was significant greater in TS, $\chi^2(2)=6.96$, p<0.01, and CON, $\chi^2(2)=3.22$, p<0.01, relative to TD. For the CS-, the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=0.67$, p>0.05, suggesting that not only did valence to the CS- not change from pre- to post-acquisition, B=-0.42, SE=0.57, z=-0.74, p>0.05, it also did not differ as a function of ABMT Condition (Figure 23).

Post-Attention Bias Modification Training (ABMT)

Arousal Rating. As expected, the ABMT Condition x Phase x Trial Type 3-way interaction was significant, $\chi^2(2)=8.13$, p<0.05. For CS+, the ABMT Condition x Trial Order interaction was marginally significant, $\chi^2(2)=5.32$, p=0.07, suggesting that self-reported arousal to the CS+ differed marginally as a function of ABMT Condition. Namely, the reduction in arousal to the CS+ was significantly greater in TS compared to TD, z=-2.3, SE=0.87, p<0.05. For the CS-, the ABMT Condition x Trial Order interaction was significant, $\chi^2(2)=7.21$, p<0.05, such that the decline in arousal to the CS- was greater in CON relative to TD, z=2.64, SE=0.54, p<0.01(Figure 22).

Valence Rating. As expected, the ABMT Condition x Phase x Trial Type 3-way interaction was significant, $\chi^2(2)=6.70$, p<0.05. For CS+, the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=2.39$, p>0.05, suggesting that self-reported valence to

the CS+ did not differ as a function of ABMT Condition. The overall decline in valence was significant, B=-3.42, SE=0.72, z=-4.73, p<0.01. For the CS-, the ABMT Condition x Trial Order interaction was marginally significant, $\chi^2(2)=5.20$, p=0.074. Namely, the decline in valence to the CS- was greater in TD relative to CON, z=2.70, SE=0.54, p<0.05 (Figure 23).

Post-Extinction

Arousal Rating. The ABMT Condition x Phase x Trial Type 3-way interaction was not significant, $\chi^2(2)=0.72$, p>0.05. For CS+, the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=0.46$, p>0.05, suggesting that the self-reported arousal to the CS+ did not differ as a function of ABMT Condition from pre- to post-Extinction. For the CS-, the ABMT Condition x Trial Order interaction was also not significant, $\chi^2(2)=3.55$, p>0.05, also suggesting that self-reported arousal to CS- also did not vary as a function of ABMT Condition (Figure 22).

Valence Rating. The ABMT Condition x Phase x Trial Type 3-way interaction was not significant, $\chi^2(2)=0.17$, p>0.05. For the CS+, the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=3.01$, p>0.05. For the CS-, the ABMT Condition x Trial Order interaction was significant, $\chi^2(2)=9.36$, p<0.01, suggesting that self-reported valence to the CS- differed as a function of ABMT Condition (Figure 23). Namely, self-reported valence was greater to the CS- in TD relative to TS.

Pre-Extinction Retest

Arousal Rating. The ABMT Condition x Phase x Trial Type 3-way interaction was not significant, $\chi^2(2)=0.41$, p>0.05. For CS+, the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=3.13$, p>0.05, suggesting that self-reported arousal to the CS+ did not differ as a function of ABMT Condition from the post-Extinction to pre-Extinction Retest.

For the CS-, the ABMT Condition x Trial Order interaction was also not significant, $\chi^2(2)=1.86$, $p>0.05$, also suggesting that self-reported arousal to CS- also did not vary as a function of ABMT Condition (Figure 22).

Valence Rating. The ABMT Condition x Phase x Trial Type 3-way interaction was not significant, $\chi^2(2)=0.35$, $p>0.05$. For the CS+, the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=1.45$, $p>0.05$, suggesting that self-reported valence did not vary across ABMT Conditions. For the CS-, the ABMT Condition x Trial Order interaction was marginally significant, $\chi^2(2)=5.46$, $p=0.065$, such that the decline in self-reported valence was greater in TD relative to TS, $z=-2.24$, $SE=0.62$, $p<0.05$ (Figure 23).

Post-Extinction Retest

Arousal Rating. The ABMT Condition x Phase x Trial Type 3-way interaction was not significant, $\chi^2(2)=2.75$, $p>0.05$. For CS+, the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=3.78$, $p>0.05$, suggesting that the self-reported arousal to the CS+ did not differ as a function of ABMT Condition. For the CS-, the ABMT Condition x Trial Order interaction was also not significant, $\chi^2(2)=0.19$, $p>0.05$, also suggesting that self-reported arousal to CS- also did not vary as a function of ABMT Condition (Figure 22).

Valence Rating. The ABMT Condition x Phase x Trial Type 3-way interaction was not significant, $\chi^2(2)=1.62$, $p>0.05$. For the CS+, the ABMT Condition x Trial Order interaction was not significant, $\chi^2(2)=2.03$, $p>0.05$, suggesting that self-reported valence did not vary across ABMT Conditions. For the CS-, the ABMT Condition x Trial Order interaction was also not significant, $\chi^2(2)=0.13$, $p>0.05$, suggesting that self-reported valence did not vary across ABMT Conditions (Figure 23).

Summary

Despite randomization, we observed an unexpected baseline difference (at Post-Acquisition) prior to the introduction of ABMT manipulation. Namely, arousal to the CS+ was significantly greater in CON compared to TD, and the increase in valence was significantly greater in TS and CON compared to TD. Following ABMT, decrease in arousal to the CS_B (CS+) in TS was trending towards significance relative to TD, which was an unexpected finding. Equally unexpected, the decline in arousal to the CS_D (CS-) was greater in CON relative to TD. On the other hand, as expected, the decline in valence to the CS_D (CS-) was greater in TD relative to CON. Although we hypothesized that TD would facilitate the extinction of valence and arousal to the CS_B (CS+) following Extinction compared to CON and TS, the results did not observe any group differences. An unexpected finding was that self-reported valence was greater to the CS_D (CS-) in TD relative to TS. Despite similar hypotheses about reactivity on Visit 2 at Extinction Retest, we continued to *not* observe any groups differences on self-reported valence and arousal ratings for the most part. Although not specifically hypothesized, but still consistent with our general hypothesis, we did find that the decline in self-reported valence was greater in TD relative to TS for the CS_B (CS+). Following Extinction Retest, no group differences were observed in valence and arousal ratings. Overall, ABMT did not have a particularly strong effect on self-reported valance and arousal ratings to the CS_B (CS+) and CS_D (CS-) following each experimental phase across both visits.

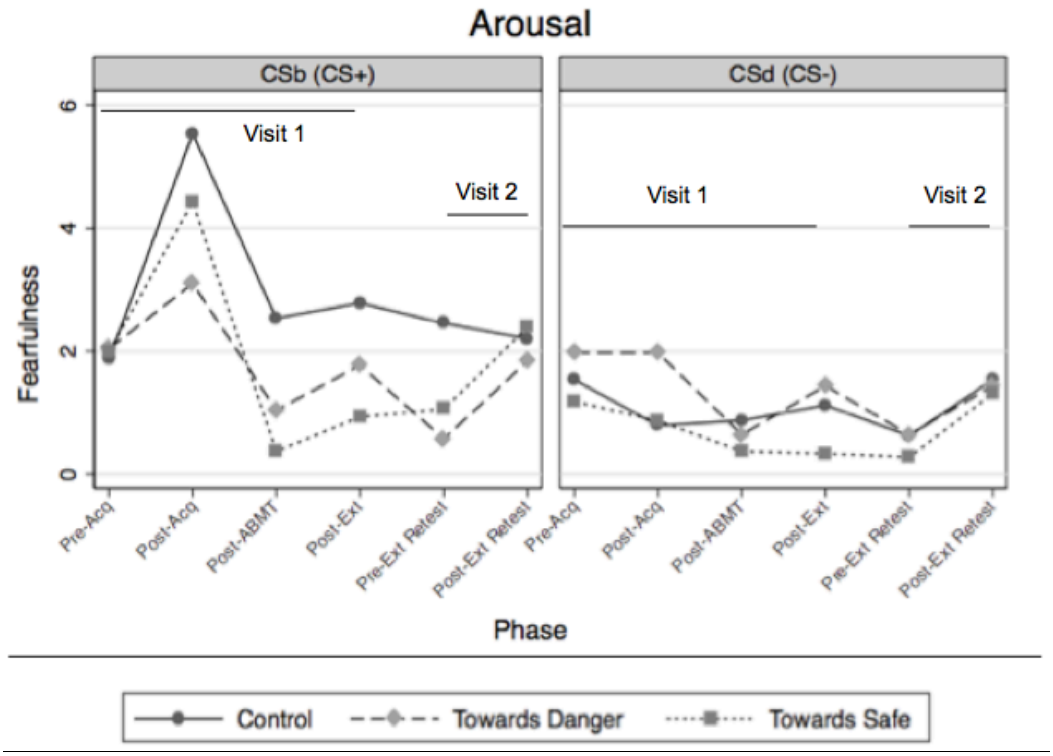


Figure 22.

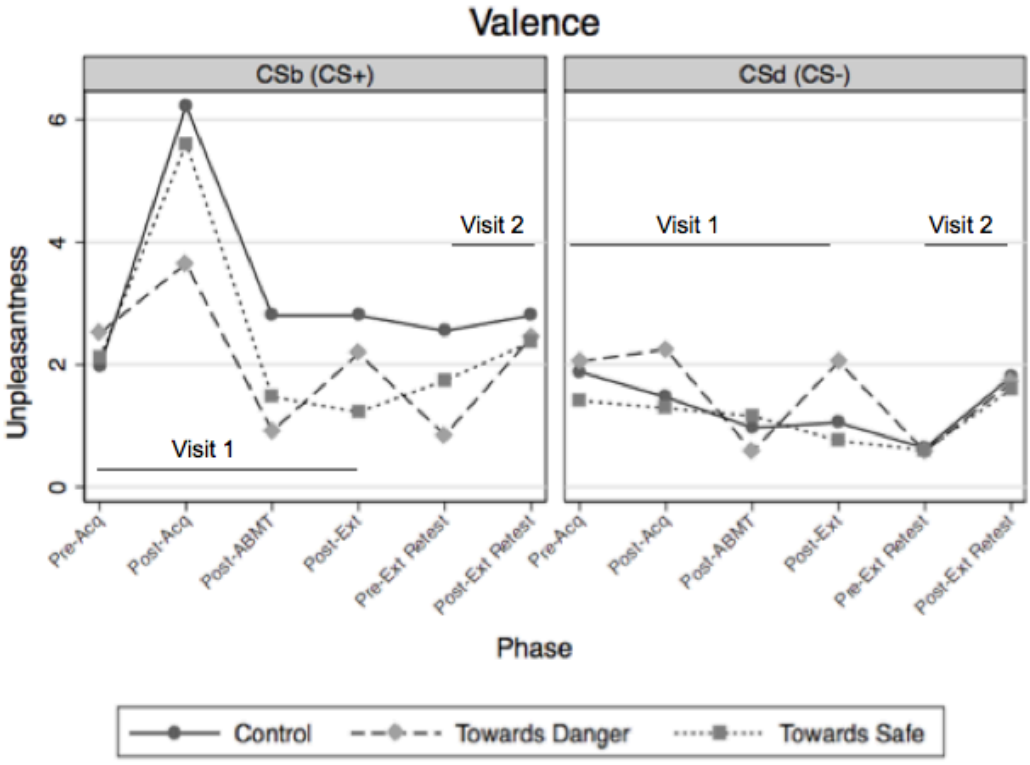


Figure 23.

DISCUSSION

Although it is well established that selective attention to evolutionarily prepared threat cues (e.g., spiders, snakes; Mogg et al., 2000) is observed across both healthy and clinically-anxious samples, and that disorder-specific stimuli can also command an attentional bias in disordered-samples (e.g., Mogg & Bradley, 2004), much less is known about the establishment of an attentional bias to stimuli that have *acquired* a signal value for threat through learning/experience (e.g., fear acquisition). Thus, the first aim of the study was to examine attention bias to acquired threat cues (CS+) in comparison with neutral cues and safety cues (CS-).

The second aim of the study was to examine the effect of attention training on fear extinction. To this end, several hypothesizes were tested in this study based on several theories of classical (fear) conditioning that posit that attention allocation is a prerequisite for learning (Mackintosh, 1975; Mitchell, De Houwer, & Lovibond, 2009; Wagner, 1981). Given that learning, in general, requires attention, it follows that the strength of fear extinction should also depend on the availability of attentional resources. In line with this logic, this study hypothesized that (1) attention allocation training towards the CS+ would enhance extinction performance; (2) it would also facilitate extinction learning and lead to less recovery of conditional fear responding at spontaneous recovery and reinstatement tests; and (3) attention allocation training towards the CS- functionally serves as avoidance and would be detrimental to fear extinction performance and learning as evidenced by greater recovery of the conditional fear at spontaneous recovery and reinstatement tests. Results were mixed in supporting the study hypotheses.

Efficacy of Fear Acquisition

Overall, we observed differential fear acquisition to both sets of the CS+ and the CS-. The effect was particularly robust as indexed by self-reported CS-US expectancy ratings. Although differential fear acquisition was also established based on physiological indices (ASR and SCR) by the last block of Acquisition, it was notable that there was an unexpected decline in the amplitude of the SCR with each subsequent block of Acquisition. The decline in skin conductance responding to the onset of the CS may be due to the fact that the startle probe, administered during the CS presentation to elicit the ASR, may have overshadowed the conditional responding to the geometric shapes themselves (CSs). This may be due to the idea of CS-US belongingness such that an auditory stimulus (i.e., startle probe) compared to a visual stimulus (i.e., geometric shape) may more easily acquire conditional responding to an aversive shock (US) due to their evolutionary relationship (Garcia & Koelling, 1966). Thus, more conditional responding to the CS+ may be observed in the ASR (elicited by the startle probe) rather than the SCR (elicited by the onset of the geometric shape). Regardless of this slight anomaly, it could be safely assumed that successful differential fear acquisition was established.

Effect of Differential Conditioning on Attention Allocation to CS+ and CS-

Greatly to our surprise, we did not find an attentional bias towards the CS+ as operationalized by quicker response time on valid CS+/neutral trials versus valid neutral/neutral trials in our study. Instead, we found the opposite effect. Namely, participants were *slower* to respond to probes in the vicinity of the CS+. This seemed to suggest that that the presence of the CS+ interfered with the subsequent task of identifying the probe (":" or ". ."). Furthermore, this pattern held true regardless of the location of the

probe (in the same or opposite location of the CS+). Due to the fact that prior literature is not well-established and only one other study to date has examined the impact of differential fear conditioning on attention bias as measured by the DPT (Pischek-Simpson et al., 2009), comparisons with prior studies were limited. Consistent with our finding, Pischek-Simpson and colleagues also did not find a difference in response time between valid and invalid trials. This would also suggest that the presence of the CS+ had a uniform effect on the subsequent probe task regardless of the location of the probe. That being said, Pischek-Simpson and colleagues still found that the CS+ *facilitated* the detection of the probe relative to the CS-. This was not observed in our study. Although the two studies were similar in methodology, our study consisted of high trait-anxious participants and the other study did not. The difference in response time observed could be due to the moderating effect of trait anxiety. There is some support for this based on a recent meta-analysis of threat-related attentional bias in anxious and non-anxious individuals (Bar-Haim et al., 2007), which found a significant difference between anxious and control participants in studies that were based on measures of trait anxiety.

In our review of findings relating attention bias (using the DPT) to fear/anxiety in general (not limited to *acquired* bias as a result of fear conditioning), we found several studies that also did *not* find an attentional bias for threat cues relative to neutral cues (e.g., Roberts et al., 2005; Livermore et al., 2007). However, only one study to date observed a *delay* in response time to threat cues as we did in the current study (Bar-Haim et al., 2010). Bar-Haim and colleagues suggested that the reverse effect observed in their study was due to the fact that their study consisted of a traumatized population who had gone through war-related stress. Namely, attentional threat *avoidance* may reduce the acute impact of

imminent threat. Although the our sample cannot be compared with the sample from Bar-Haim et al.'s study, the results together do challenge current thinking about the role of attention across diverse populations.

Our results, based on the DPT, were more akin to the pattern of results observed in high trait-anxious participants who have undergone the emotional stroop task. In the emotional stroop task, anxious participants are *slower* in responding to the color of a word when the semantic content of the word is related to threat compared to non-anxious participants. It is hypothesized that the attentional bias to word with content related to threat would *interfere* with one's ability to perform a concurrent task (i.e., naming the ink color of the word) because attentional resources have been disproportionately allocated to the content of the word instead of the ink color of the word. The interfering effect of the emotional stroop task does not, however, clarify the mechanism through which it operates or the components of attention on which the mechanisms operate. One likely mechanism is attentional control, which is responsible for orienting, maintaining, and shifting attention (cf. Posner, 1980; 1990) and it may modulate the degree to which emotional stimuli withdraw attention from task demands (Derryberry & Reed, 2002; Eysenck et al., 2007; Reinholdt-Dunne et al., 2009; Wyble et al., 2008). Disruption of this mechanism would explain the delayed effect to the CS+ (i.e., slowed reaction to the probe task following CS+/neutral trials) observed in our study.

Efficacy of ABMT on DPT

Efficacy of ABMT on attention allocation on CS at Post-ABMT Dot Probe Task was difficult to assess due to the lack of attentional bias to the CS+ at Pre-ABMT assessment. Instead, we observed a general interference effect of the CS+ resulting in slower response

time to the probe following CS+ relative to CS- and/or neutral trials at Pre-ABMT. From pre- to post-ABMT, decrease in RT to CS+/neutral trials was greater in TD than in TS. That being said, this rate was not different between TD and CON. In summary, we observed some support for the efficacy of TD, but it was not conclusive.

In TS, we observed a similar interference effect of the CS+ at Pre- relative to the CS. That being said, we did not observe a reduction in response time to the CS- as hypothesized. However, we continued to observe a reduction in response time to the CS+ such that by Post-, response times to both the CS+ and CS- were significantly faster than the neutral trials. In summary, ABMT towards the CS- was not successful.

In CON, we did not find an attentional bias towards the CS+ to begin with. By Post, response times to the CS+, CS-, and neutral trials also did not differentiate, but were faster in general. In summary, our ABMT manipulation was partly successful in TD, but not successful in TS.

Effect of ABMT on Extinction Performance

Contrary to hypothesis, ABMT Towards the CS+ did not facilitate greater extinction performance relative to No ABMT Control on all conditioning measures (ASR, SCR, US Expectancy Rating, and Valence and Arousal Ratings). In fact, ABMT Towards the CS- facilitated greater extinction performance (i.e., slope of extinction) to the CS+ on SCR compared to the No ABMT Control group. Although a similar decline in SCR was observed in ABMT Towards CS+, this was not significantly different from the No ABMT Control group. That being said, the skin conductance response at the last trial of Extinction was significantly smaller in the ABMT Towards CS+ group as well as the Towards CS- group in comparison with the control group, which suggested that a comparable degree of

facilitation in extinction performance was observed in both attention retraining groups, in spite of the fact that a statistically significant difference in the (negative) slope of the SCR was not observed between ABMT Towards CS+ and the no ABMT control.

A similar pattern was observed in the ASR, where ABMT Towards the CS- facilitated greater extinction performance to the CS+ and marginally to the CS- relative to the No ABMT Control group. Furthermore, ABMT Towards the CS+ interfered with extinction performance to the CS- on US expectancy rating.

With respect to valence and arousal ratings, ABMT Towards the CS+ resulted in increased subjective valence rating to the CS- from pre- to post- Extinction relative to both the ABMT Towards the CS- and No ABMT Control groups.

It is unclear why making the CS- more salient (TS group) would enhance extinction performance to the CS+ (evidenced by ASR and SCR) and CS- (evidenced by ASR). These results contradict with the universal fact that attention is required for (new) learning to occur and the lack of attention should be detrimental to learning, including learning the inhibitory association between the CS+ and no-US during Fear Extinction. It is possible that training participants to attend towards the CS- functionally served to distract participants from attending to the CS+ during Extinction, even though it is not likely to be the case because we did not observe a similar pattern in CON. But if it were the case, then it follows that the greater reduction in conditional responding to the CS+ did not actually reflect greater extinction, but rather greater decrease in reactivity due to being distracted by the CS-, and thus less conditional responding to the CS+ was processed and expressed. If this is true, it should also follow that despite the appearance of greater extinction performance to the CS+ in the ABMT Towards CS- group, extinction learning as measured

by recovery of the conditional response at spontaneous recovery and reinstatement (Visit 2) should be enhanced due to a lack of "true" learning during Extinction. This was confirmed in the subsequent extinction tests. Namely, we did not continue to observe a facilitating effect of ABMT Towards the CS- at Spontaneous Recovery and Reinstatement. It is also noteworthy that the influence of attention engagement versus distraction during fear extinction (or exposure therapy) is still a topic of debate (e.g., Johnstone & Page, 2004; Schmid-Leuz et al., 2007).

It is also unclear why US-expectancy ratings did not converge with objective measures, such as the skin conductance response and the acoustic startle response, in reflecting the facilitating effect of extinction performance when attention allocation was trained towards the CS-. There is some evidence that subjective US-expectancy ratings, skin conductance response, and acoustic startle reactivity may dissociate from each other (e.g., McAndrew et al., 2012; Sevenster et al., 2014). The desynchrony among various indices of conditional fear responding comes from a body of literature investigating whether Pavlovian conditioning in humans is a unitary system in which conscious awareness of contingencies is necessary (e.g., Hinchy et al., 1995) or whether it is driven by dual systems, one under conscious control and the other under automatic associative processes (McLaren et al., 1994). The results from this study is more in line with the latter theory, which allows for a dissociation between conscious awareness of contingencies (i.e., US expectancy) and a physiological CR, such as the acoustic startle response. A separate line of reasoning based on the nature of the attention training also suggest that it may impact automatic measures of conditioning (e.g., skin conductance and acoustic startle reactivity) more directly compared to more strategic components of conditioning (i.e., US

expectancy) because the dot probe task itself operates on a more unconscious level of cognitive processing (see review by Cisler et al., 2010).

Contrary to hypothesis, extinction performance to the CS+ was impaired in the ABMT Towards CS+ group relative to the ABMT Towards CS- group and the no ABMT Control group based on US expectancy ratings. Although a significant decline was observed in the ABMT Towards CS+ group, confirming successful extinction performance, the decline in conditional responding in the two other groups were significantly more steep. The results are not consistent with the cognitive accounts of classical conditioning and with Emotional Processing Theory (Foa & Kozak, 1986), which posits that in order for exposure therapy to work, the underlying fear structure must be activated and then change, which can only happen when the anxious individual attends to the threatening stimulus. The results are also at odds with the experiment conducted by Van Bockstaele and colleagues (2010), which found that training attention towards the CS+ during extinction promoted extinction performance on US expectancy ratings in comparison with training attention away from the CS+. That being said, the methodology in the Van Bockstaele study differed from the methodology in the current study in significant ways. First, Van Bockstaele and colleagues combined attention retraining manipulation with fear extinction concurrently. Second, by combining attention retraining and fear extinction in one phase, significantly fewer CS+ and CS- trials were presented compared to the typical attention bias modification training based on the original design proposed by MacLeod and colleagues (2002). Lastly, Van Bockstaele's study involved the same set of conditional stimuli being presented during both attention retraining and fear extinction; whereas the current study examined the impact of attention retraining on one set of conditional stimuli (CS+ and CS-)

on a different set of conditional stimuli during extinction. It should not be assumed that (attention) training to one set of stimuli would generalize to other stimuli. The implicit assumption regarding the transfer of learning from ABMT to everyday life was more carefully scrutinized in a more recent study by Van Bockstaele and colleagues (2012), which also did not find successful generalization of attention training to a separate set of stimuli presented during a subsequent emotional interference task. It is possible that the absence of a facilitating effect of attention retraining on fear extinction to a separate set of stimuli found in the current study merely adds to the growing body of evidence pointing to the idea that attentional training with the dot probe task is limited to spatial attention, and that the effects fail to generalize to other components of attention, such those involved during fear extinction learning.

Effect of ABMT on Spontaneous Recovery

In light of more recent advances in the literature suggesting that fear reduction during or at the end of extinction does not predict responding upon re-test (Bouton et al., 2006; Rescorla, 2006; Plendl, Wolfgang et al., 2010), we hypothesized that measures assessed at a later point in time, such as on a subsequent visit taking place 7 days following the Extinction phase, may be a better marker of the impact of ABMT on (fear extinction) learning. We hypothesized that ABMT Towards the CS+ would promote extinction learning as evidenced by reduced recovery of the conditional response to the CS+ at Spontaneous Recovery. The results partially supported this claim, but also provided contradictory evidence.

Impact of Any Attention Training

Contrary to our hypothesis, both attention retraining groups (ABMT Towards CS+ and CS-) resulted in enhanced spontaneous recovery of the skin conductance response to the CS+. Similarly, the acoustic startle response also provided further evidence for the negative impact of any attention retraining on the conditional response to the CS+. Moreover, although not specifically predicted in our hypothesis, a similar increase in the spontaneous recovery of the conditional responding was also observed towards the CS- on skin conductance. It is unclear why attention retraining procedures that increased the salience to both the CS+ and the CS- would equally contribute to greater spontaneous recovery of the CS+ relative to no attention retraining.

Upon closer examination, it was very curious that the spontaneous recovery effect was not observed in the CON group on both skin conductance response and acoustic startle response to the CS+ and CS- despite the effect being observed in the measure of US expectancy. The lack of a spontaneous recovery effect in CON could be a spurious finding or due to pre-existing group differences independent from fear conditioning or group differences during fear conditioning (acquisition and/or extinction). A baseline difference was found in CON relative to other ABMT conditions such that participants in CON reported greater intensity to the US they experienced during Acquisition. This should, however, predict greater spontaneous recovery rather than the lack of it. During Extinction, group difference was also observed in between CON and other ABMT groups. Namely, extinction of the CS+ was reduced in CON relative to TS. This, too, should predict greater subsequent spontaneous recovery in CON rather than less. We conclude that because it was not clear why spontaneous recovery was not observed in CON, ABMT group comparisons to CON

were difficult to interpret at best and misleading at worst. If constricting our comparisons to just between the two attention retraining groups, we conclude that both training impacted Spontaneous Recovery similarly as measured by the acoustic startle response and skin conductance response.

Impact of ABMT Towards CS+

Consistent with our hypothesis, training towards the CS+ resulted in a marginally significant reduction in spontaneous recovery to the CS+ on US expectancy rating in comparison with the control group. Other measures of conditioning did not replicate this finding.

Impact of ABMT Towards CS-

Although our study did not specifically test the spontaneous recovery of the conditional response to the CS-, a marginally significant increase in skin conductance response was observed in ABMT towards the CS- compared to no attention retraining. Other measures did not replicate this finding.

Effect of ABMT on Reinstatement

Reinstatement refers to the return of conditioned responding that is observed when an unpredictable US is presented after extinction (e.g., Bouton, 1984). It provides one line of evidence for the idea that conditional fear can be recovered even following successful extinction. Given its clinical implication (i.e., relapse following successful exposure therapy), we were interested in examining the effect of ABMT on Reinstatement. The study hypothesized that training greater attention allocation to the CS+ during extinction (TD) would reduce the degree of Reinstatement to the CS+ because the CS+- no US association during Extinction would be better learned and consequently carried over into

Reinstatement. By the same token, attention training to the CS- (TS) would interfere with extinction learning and result in greater conditional responding after Reinstatement relative to CON and TD.

Impact of Any Attention Training

Contrary to expectation, we found some evidence to support that both attention retraining towards the CS+ and CS- resulted in a reduction in acoustic startle responding to the CS+ on Reinstatement compared to no attention training group. Although the reduction in responding to the CS+ in the attention training towards CS+ group was expected, we did not expect a similar decrease in the attention training towards CS- group.

Impact of ABMT Towards CS+

Although unexpected but very interesting nonetheless, attention training towards CS+ resulted in greater differentiation between the CS+ and CS- on US expectancy rating compared to other ABMT conditions. The greater differentiation was a result of both heightened US expectancy to the CS+ and reduced US expectancy to the CS-. Moreover, the US expectancy was very quickly extinguished following Reinstatement with continued non-reinforcement of the CS+. This pattern was not observed in TS or CON. That being said, physiological measures did not provide corroborating evidence.

Impact of ABMT Towards CS-

Although we hypothesized that Training Towards the CS- would result in greater conditional responding at Reinstatement, our results did not support this.

Re-Extinction

Impact of Any Attention Training

No differences between the two attention retraining groups and the no attention training control group were found in any of the conditioning measures.

Impact of ABMT Towards CS+

Contrary to expectation, attention training Towards the CS+ resulted in a decline in conditional responding on all measures to the CS+, but this was not any different than what was observed in the no attention training control group. Despite the absence of an enhancement effect from training towards the CS+ in comparison with the no attention training control group as hypothesized, we did observe significantly greater decline in conditional responding of US expectancy in comparison with the attention retraining towards CS- group. Group differences were not observed for the CS-.

Impact of ABMT Towards CS-

Although we hypothesized that training towards the CS- would have a detrimental effect on the decline in conditional responding to the CS+ relative to the no attention control group, we did not observe such a group difference on any of the conditioning measures. This was the case for the CS- as well. Overall, re-extinction was established to both the CS+ and CS- in TS. But, the rate of re-extinction was significantly slower in TS compared to TD on US expectancy rating to the CS+; and at the last trial of Re-Extinction, US expectancy rating to the CS+ continued to be significantly higher in TS compared to TD.

Summary

The aim of the present study was to investigate whether attention bias modification training towards threat or towards safety would enhance or impair fear extinction. First,

the main finding was that facilitating attention towards the CS- enhanced extinction performance to the CS+ on both the skin conductance response and the acoustic startle response relative to the no attention retraining control group. This was unexpected and particularly interesting as extinction, which is based on the learning of a new CS-no US association, relies on attention allocation, presumably to the CS+, and attention training towards the CS- should be detrimental to extinction performance. That being said, our result appears to be in line with the body of work supporting the benefits of attention bias modification training in the treatment of disordered anxiety (see review by Van Bockstaele et al., 2014). The precise mechanisms through which it operates and its moderating factors are still very much under examination as mixed results and null findings are equally abundant.

Second, the facilitating effects of attention training towards the CS- observed during extinction performance were eliminated at Spontaneous Recovery and Reinstatement, which took place approximately 7 days following Extinction. One explanation could be that the effects of attention retraining were temporary, but this would not be consistent with the literature that has established that the effects of ABMT continue to have an effect weeks following administration on both self-reported and behavioral measures of anxiety. That being said, only three studies have actually measured the physiological impact of ABMT on a subsequent "stress" task (Baert et al., 2012; Heeran et al., 2012; Higgins & Hughes, 2012). No study to date other than the current study has examined the physiological effects of ABMT on a differential fear conditioning paradigm across 2 separate visits.

Third, as predicted, facilitating attention towards the CS+ reduced US expectancy to the CS+ at Spontaneous Recovery compared to the no attention retraining group. Fourth, an unexpected but interesting finding was the effect of attention retraining towards the CS+ on Reinstatement. Following an unpaired delivery of the US, training participants to attend towards the CS+ resulted in greater discrimination between the CS+ and CS- on US expectancy. Although reinstatement effects has traditionally been posited to occur exclusively to the extinguished CS (i.e., CS+) following extinction (Bouton, 1993), more recent theories and experiments have also observed non-differential return of the conditional response to both the CS+ and CS- (see review by Dirikx et al., 2008), which is more consistent with the pattern of results observed to the CS- in the current study (in TS and CON). It is possible that attention training towards the CS+ prior to extinction helped disrupt retrieval of the excitatory US-context association posited to be the mechanism through which reinstatement to the CS- could occur (Westbrook et al., 2002; Schmajuk et al., 2007), through training more focused attentional processing of the CS-US association instead of the US-context association. Moreover, despite marginally greater reinstatement to the CS+ relative to the no attention control group, an immediate re-correction of US expectancy was observed as soon as the participants learned that the CS+ was no longer reinforced by the US, such that by the end of Re-Extinction, US expectancy between the attention training towards CS+ and no attention training control group no longer differed. That being said, it is curious that physiological measures of reinstatement were not consistent with US expectancy ratings.

Finally, as expected, we found some evidence for the harmful effect that attention training towards the CS- may have on processing the CS+ during Re-Extinction. Namely,

following an unexpected US presentation, training towards the CS- did not lead to eventual extinction of the US expectancy response to the CS+ despite subsequent non-reinforced CS+ presentations; whereas such a decline in responding was observed in the attention training towards CS+ group and the no attention training control group. Using Westbrook et al's model for reinstatement, it is possible that focused attention processing of the CS- may have inadvertently distracted the participants from learning the (inhibitory) CS+ - no US association during extinction, so that when the context became associated with the US during reinstatement, it retrieved greater conditional responding in favor of the CS+ - US association (from Acquisition) than the weakened CS+ - no US association (from Extinction). That being said, we did not observe any evidence of a weakened CS+ - no US association during Extinction in our study; in fact, our results suggested otherwise. Namely, attention training towards the CS- enhanced extinction performance based on physiological measures of conditioning. It is curious that we did not observe this in US expectancy rating during Extinction and the increased reinstatement effect was observed only in US expectancy rating and not in physiological measures of skin conductance and the acoustic startle response.

Despite the somewhat mixed results observed in this study across various conditioning measures (SCR, ASR, US expectancy and valence/arousal ratings) and procedural measures (extinction performance, spontaneous recovery, reinstatement), perhaps the most consistent and interesting finding was that attention training towards the CS+ appeared to uniquely facilitate the "cognitive" model of extinction; namely, the measure of extinction that is based on consciously available contingency knowledge (i.e., US expectancy). This was particularly striking because attention, presumably one of many

unconscious, "low-level" processes during conditioning, directly impacted US expectancy, a conscious, "high-level" process during conditioning (Lovibond, 2004). Clearly, fear conditioning (including extinction) operates on multiple levels of processing. Although it should be no surprise that modification to one process can influence another process, the precise nature of this transaction is only beginning to be illuminated.

Limitations

This study has some limitations. First, pre-existing group differences were observed despite random assignment. Most importantly, groups differed on self-reported intensity of the US during Acquisition as well as CS-US contingency awareness when asked following Acquisition. This is important in light of (1) the long-standing debate on whether fear learning can occur without awareness of the CS-US relationship, and (2) its differential impact on skin conductance response and acoustic startle response. This might explain, in part, the inconsistent finding between skin conductance and acoustic startle response observed in the current study. Second, the efficacy of the attention bias modification training is questionable. Although many studies that have used the dot probe task to measure attention bias towards various threat cues also were not able to establish bias based on the difference between valid and invalid trials (for review see Van Bockstaele et al., 2013), and thus calls into question the sensitivity and reliability of the DPT as a measure of attentional bias, without a valid baseline (CON) against which comparisons could be made, it is difficult to assess the efficacy of our attention bias modification training. Third, because the set of CSs presented to the participants were different during ABMT and Extinction, and it was *presumed* that participants would transfer their acquired attentional bias as a result of ABMT to a new set of CSs during Extinction. We were not able to

conclusively confirm that participants were actually attending more to the CS+ (TD condition), more to the CS- (TS condition), or equally to both the CS+ and CS- (CON condition) without a separate assessment of attention bias on the set of CSs presented during Extinction. Although the participants should theoretically differ only on their exposure to the type of ABMT received, and thus subsequent group differences should be able to be attributed to ABMT, we did not include a separate manipulation check in the design of the experiment to verify this.

Clinical Implications

These limitations notwithstanding, our results may prove to be clinically relevant. Our results suggest that attending one's attention towards threatening information in the environment help facilitate detection of changes in the environment, not unlike changes in CS-US expectancy from acquisition to extinction. The ability to detect changes in contingency is particularly important in individuals who are anxious and has a tendency to anticipate threat in their environment when none exist. Exposure therapy, the primary treatment for disordered anxiety and fear, is built on the principle that the disconfirmation of expectancy of a negative outcome is critical for fear reduction.

It is undeniable that attention allocation is a prerequisite for learning to occur in the therapy context, but it is unclear what "paying attention" constitutes. Our study would suggest that by visually attending to the feared stimulus (e.g., spiders, needles) during exposure therapy, the return of exaggerated, overestimated appraisals of threat following the completion of therapy, and sometimes following an unexpected stressful event, may be eliminated, which would in turn, reduce sensitivity to future stressors.

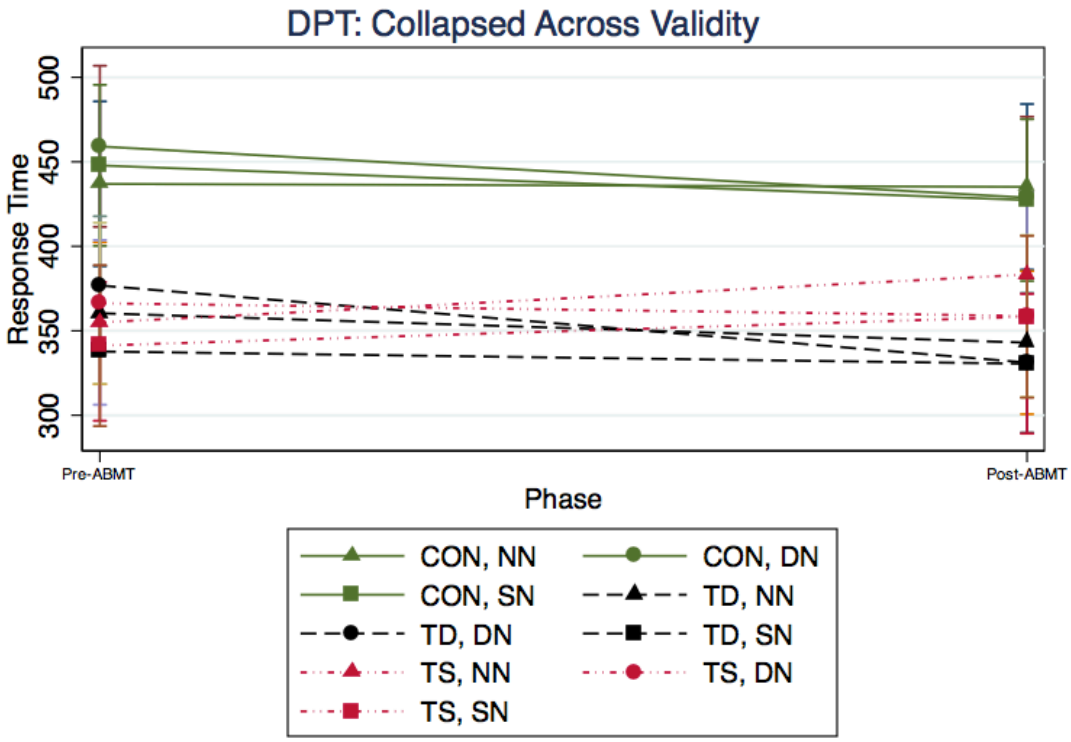
Results from our study also suggest that by directing attention towards a safety cue during exposure therapy (e.g., anti-anxiety medication) may lead to temporary relief from the physiological sensations of anxiety during exposure therapy, but it is not long-lasting. Moreover, there is some evidence to suggest that such training may impair one's ability to retrieve the learning of the discrepancy between the expected negative outcome and the actual neutral outcome during exposure therapy upon re-encountering a separate anxiety-provoking stimulus (e.g., car accident) following the completion of therapy.

Future Directions

It will be important for future studies to replicate and build on the design and results of the current study. A number of components were unique to this study and need to be further investigated. First, the impact of attention retraining on physiological indices of fear is not well documented in the literature. Thus, further examination of the intersection between attention modification and physiological markers of anxiety during fear learning (e.g., skin conductance, acoustic startle response, cardiac vagal tone) is warranted. Next, it'll also be important to further tease apart the similarities and differences between attention modification and distraction during exposure therapy. This could be done by examining patterns of attention allocation across different stages of information processing (i.e., orienting, disengaging, shifting, response inhibition, etc.) Lastly, knowing that attention constitutes one of many cognitive processes that underlie fear learning and extinction, it'll be important to continue to explore the impact of attention retraining on a whole gamut of cognitive processes (e.g., cognitive appraisal, mindfulness, attentional control) as potential mediators and moderators in the development, maintenance, and treatment of disordered anxiety and fear.

Appendix A

The Trial Type x ABMT Condition interaction was significant, $\chi^2(4)=12.89$, $p<0.05$. Namely, in CON, Response Time to CS+/neutral trials was significantly greater relative to neutral/neutral trials, suggesting slower response to CS+ relative to neutral trials following Fear Acquisition, $z=2.31$, $SE=9.60$, $p<0.05$). In TD, following Fear Acquisition, Response Time to CS+/neutral trials was significantly greater than neutral/neutral trials, $z=2.14$, $SE=7.70$, $p<0.05$, and CS-/neutral trials, $z=6.17$, $SE=6.33$, $p<0.01$. Moreover, Response Time to neutral/neutral trials was also significantly greater than CS-/neutral trials, $z=2.94$, $SE=7.71$, $p<0.01$. In TS, following Fear Acquisition, Response Time to CS+/neutral trials was significantly greater than CS-/neutral trials, $z=3.29$, $SE=7.64$, $p<0.01$. It is also apparent that there was a significant main effect of ABMT Condition such that Response Time, in general, was greater in CON across all Trial Types in comparison with TD and TS, $\chi^2(2)=10.29$, $p<0.01$.



Appendix B

Post-hoc pairwise comparisons between Trial Types (1=CSa; 2=CSb; 4=CSc; 5=CSd) at Trial 1 of Fear Acquisition on US expectancy.

	Contrast	Delta-method Std. Err.	Unadjusted z	Unadjusted P>|z|	Unadjusted [95% Conf.	Interval]
trialtype						
2 vs 1	.4102957	.5372094	0.76	0.445	-.6426154	1.463207
4 vs 1	-.0892396	.5372094	-0.17	0.868	-1.142151	.9636715
5 vs 1	.7668256	.5372094	1.43	0.153	-.2860855	1.819737
4 vs 2	-.4995353	.5372094	-0.93	0.352	-1.552446	.5533758
5 vs 2	.3565299	.5372094	0.66	0.507	-.6963813	1.409441
5 vs 4	.8560652	.5372094	1.59	0.111	-.1968459	1.908976

Appendix C

Post-hoc pairwise comparisons between Trial Types (1=CSa; 2=CSb; 4=CSc; 5=CSd) at Trial 8 of Fear Acquisition on US expectancy.

	Contrast	Delta-method Std. Err.	Unadjusted z	Unadjusted P>|z|	Unadjusted [95% Conf.	Interval]
trialtype						
2 vs 1	-.5318666	.5372094	-0.99	0.322	-1.584778	.5210445
4 vs 1	-6.041801	.5372094	-11.25	0.000	-7.094712	-4.988889
5 vs 1	-4.927219	.5372094	-9.17	0.000	-5.98013	-3.874308
4 vs 2	-5.509934	.5372094	-10.26	0.000	-6.562845	-4.457023
5 vs 2	-4.395353	.5372094	-8.18	0.000	-5.448264	-3.342441
5 vs 4	1.114581	.5372094	2.07	0.038	.0616702	2.167492

Appendix D

Post-hoc pairwise comparisons between Trial Types (1=CSa; 2=CSb; 4=CSc; 5=CSd) at Block 1 of Fear Acquisition on skin conductance response.

	Contrast	Delta-method Std. Err.	Unadjusted z	Unadjusted P>|z|	Unadjusted [95% Conf.	Interval]
trialtype						
2 vs 1	.0055526	.0149255	0.37	0.710	-.0237008	.034806
4 vs 1	-.0560119	.0148159	-3.78	0.000	-.0850505	-.0269733
5 vs 1	-.0440438	.0148159	-2.97	0.003	-.0730824	-.0150052
4 vs 2	-.0615645	.0149255	-4.12	0.000	-.0908179	-.0323111
5 vs 2	-.0495965	.0149255	-3.32	0.001	-.0788499	-.020343
5 vs 4	.011968	.0148159	0.81	0.419	-.0170706	.0410066

Appendix E

Post-hoc pairwise comparisons between Trial Types (1=CSa; 2=CSb; 4=CSc; 5=CSd) at Block 4 of Fear Acquisition on skin conductance response.

	Contrast	Delta-method Std. Err.	Unadjusted z	Unadjusted P>|z|	Unadjusted [95% Conf.	Interval]
trialtype						
2 vs 1	.0120568	.0148159	0.81	0.416	-.0169818	.0410954
4 vs 1	-.060316	.0148159	-4.07	0.000	-.0893546	-.0312774
5 vs 1	-.0501365	.0149244	-3.36	0.001	-.0793878	-.0208852
4 vs 2	-.0723727	.0148159	-4.88	0.000	-.1014113	-.0433341
5 vs 2	-.0621933	.0149244	-4.17	0.000	-.0914446	-.032942
5 vs 4	.0101795	.0149244	0.68	0.495	-.0190718	.0394308

Appendix F

Phase: Acquisition
Measure: US Expectancy

		Mean	Std. Error	z	p	[95% Conf.	Int]
CSa (CS+)	Trial 1	.0993898	.4573532	0.22	0.828	-.797006	.9957856
	2	2.429487	.4573532	5.31	0.000	1.533091	3.325883
	3	1.159177	.4573532	2.53	0.011	.2627808	2.055572
	4	2.632567	.4573532	5.76	0.000	1.736171	3.528963
	5	3.09052	.4573532	6.76	0.000	2.194124	3.986916
	6	2.728156	.4573532	5.97	0.000	1.83176	3.624552
	7	1.867651	.4573532	4.08	0.000	.9712555	2.764047
	8	3.178204	.4573532	6.95	0.000	2.281808	4.0746
CSb (CS+)	Trial 1	.539028	.4573532	1.18	0.239	-.3573678	1.435424
	2	1.174968	.4573532	2.57	0.010	.2785725	2.071364
	3	2.455562	.4573532	5.37	0.000	1.559166	3.351958
	4	1.553139	.4573532	3.40	0.001	.6567437	2.449535
	5	2.625455	.4573532	5.74	0.000	1.729059	3.521851
	6	1.862411	.4573532	4.07	0.000	.9660155	2.758807
	7	1.890032	.4573532	4.13	0.000	.993636	2.786428
	8	2.605913	.4573532	5.70	0.000	1.709517	3.502309
CSc (CS-)	Trial 1	.0037692	.4573532	0.01	0.993	-.8926266	.900165
	2	-1.216026	.4573532	-2.66	0.008	-2.112422	-.3196305
	3	-1.271619	.4573532	-2.78	0.005	-2.168015	-.375223
	4	-1.84245	.4573532	-4.03	0.000	-2.738846	-.9460546
	5	-1.674289	.4573532	-3.66	0.000	-2.570684	-.7778929
	6	-2.226214	.4573532	-4.87	0.000	-3.12261	-1.329819
	7	-2.728104	.4573532	-5.96	0.000	-3.624499	-1.831708
	8	-3.257034	.4573532	-7.12	0.000	-4.15343	-2.360639
CSd (CS-)	Trial 1	.8882287	.4573532	1.94	0.052	-.0081671	1.784624
	2	-1.230677	.4573532	-2.69	0.007	-2.127073	-.3342815
	3	-.7895307	.4573532	-1.73	0.084	-1.685926	.1068651
	4	-.6037406	.4573532	-1.32	0.187	-1.500136	.2926552
	5	-.6225704	.4573532	-1.36	0.173	-1.518966	.2738254
	6	-1.191397	.4573532	-2.60	0.009	-2.087793	-.2950014
	7	-1.811975	.4573532	-3.96	0.000	-2.708371	-.9155797
	8	-2.01727	.4573532	-4.41	0.000	-2.913666	-1.120874

Appendix G

Phase: Acquisition
Measure: Skin Conductance Response

		Mean	Std. Error	z	p	[95% Conf.	Interval]
CSa (CS+)	Block 1	.1855518	.0134041	13.84	0.000	.1592803	.2118233
	2	.1173687	.0134041	8.76	0.000	.0910972	.1436402
	3	.1095153	.0134041	8.17	0.000	.0832438	.1357868
	4	.1055844	.0134041	7.88	0.000	.0793129	.1318559
CSb (CS+)	Block 1	.1908279	.013522	14.11	0.000	.1643253	.2173306
	2	.1193461	.0134041	8.90	0.000	.0930746	.1456176
	3	.1306779	.0134041	9.75	0.000	.1044064	.1569494
	4	.1187013	.0134041	8.86	0.000	.0924298	.1449728
CSc (CS-)	Block 1	.128857	.0134041	9.61	0.000	.1025855	.1551285
	2	.0953632	.0134041	7.11	0.000	.0690917	.1216348
	3	.0831462	.0134041	6.20	0.000	.0568747	.1094177
	4	.0456144	.0134041	3.40	0.001	.0193429	.0718859
CSd (CS-)	Block 1	.1409747	.0134041	10.52	0.000	.1147032	.1672462
	2	.1063416	.0134041	7.93	0.000	.0800701	.1326131
	3	.0660629	.0134041	4.93	0.000	.0397914	.0923344
	4	.0570207	.013522	4.22	0.000	.030518	.0835234

Appendix H

Phase: Acquisition
Measure: Acoustic Startle Response

		Mean	Std. Error	z	p	[95% Conf.	Interval]
CSa (CS+)	Block 1	5.381695	.1436341	37.47	0.000	5.100178	5.663213
	2	5.156928	.1436341	35.90	0.000	4.87541	5.438445
	3	5.061025	.1436341	35.24	0.000	4.779507	5.342542
	4	5.168419	.1436341	35.98	0.000	4.886901	5.449937
CSb (CS+)	Block 1	5.00099	.1466952	34.09	0.000	4.713473	5.288507
	2	4.845844	.1466952	33.03	0.000	4.558326	5.133361
	3	4.827084	.1466952	32.91	0.000	4.539567	5.114601
	4	5.022053	.1466952	34.23	0.000	4.734536	5.30957
CSc (CS-)	Block 1	4.87264	.150963	32.28	0.000	4.576758	5.168522
	2	4.765761	.150963	31.57	0.000	4.469879	5.061643
	3	4.597448	.150963	30.45	0.000	4.301566	4.89333
	4	4.745073	.150963	31.43	0.000	4.449191	5.040955
CSd (CS-)	Block 1	4.743228	.1563388	30.34	0.000	4.43681	5.049646
	2	4.773014	.1563388	30.53	0.000	4.466596	5.079432
	3	4.637774	.1563388	29.66	0.000	4.331356	4.944193
	4	4.582365	.1563388	29.31	0.000	4.275947	4.888784

Appendix I

Phase: Extinction
Measure: US Expectancy

	Mean	Std. Error	z	p	[95% Conf.	Interval]
CSb (CS+) Trial 1 CON	2.046331	.7439909	2.75	0.006	.5881356	3.504526
TD	1.028285	.6885035	1.49	0.135	-.3211569	2.377727
TS	2.788059	.6778691	4.11	0.000	1.45946	4.116658
CSb (CS+) Trial 2 CON	.8341243	.7254794	1.15	0.250	-.5877892	2.256038
TD	.2387246	.6713576	0.36	0.722	-1.077112	1.554561
TS	2.350781	.6487475	3.62	0.000	1.07926	3.622303
CSb (CS+) Trial 3 CON	-1.150378	.7151735	-1.61	0.108	-2.552093	.2513362
TD	-.4720123	.6618118	-0.71	0.476	-1.76914	.8251149
TS	1.370468	.6395276	2.14	0.032	.1170173	2.623919
CSb (CS+) Trial 4 CON	-1.456806	.713429	-2.04	0.041	-2.855101	-.0585113
TD	-1.941631	.6601958	-2.94	0.003	-3.235591	-.6476713
TS	-1.046278	.6379668	-1.64	0.101	-2.29667	.2041145
CSb (CS+) Trial 5 CON	-3.399697	.7203079	-4.72	0.000	-4.811474	-1.987919
TD	-1.408563	.6665675	-2.11	0.035	-2.715011	-.102115
TS	-2.018731	.6441209	-3.13	0.002	-3.281184	-.7562768
CSb (CS+) Trial 6 CON	-3.579153	.7355685	-4.87	0.000	-5.020841	-2.137466
TD	-1.843321	.6807024	-2.71	0.007	-3.177474	-.5091691
TS	-2.509305	.6577733	-3.81	0.000	-3.798517	-1.220093
CSb (CS+) Trial 7 CON	-3.875989	.7587051	-5.11	0.000	-5.363024	-2.388955
TD	-2.577783	.7021319	-3.67	0.000	-3.953937	-1.20163
TS	-3.256733	.6784716	-4.80	0.000	-4.586513	-1.926953
CSb (CS+) Trial 8 CON	-4.349654	.7890251	-5.51	0.000	-5.896114	-2.803193
TD	-2.615507	.7382141	-3.58	0.000	-4.046701	-1.184314
TS	-2.932057	.7055959	-4.16	0.000	-4.315	-1.549115
CSd (CS-) Trial 1 CON	-1.871374	.7439909	-2.52	0.012	-3.329569	-.4131783
TD	-1.696107	.6885035	-2.46	0.014	-3.045549	-.3466648
TS	-1.342569	.6653082	-2.02	0.044	-2.646549	-.0385889
CSd (CS-) Trial 2 CON	-3.641055	.7254794	-5.02	0.000	-5.062969	-2.219142
TD	-2.105088	.6713576	-3.14	0.002	-3.420924	-.7892508
TS	-1.313906	.6487475	-2.03	0.043	-2.585428	-.0423847
CSd (CS-) Trial 3 CON	-4.26124	.7151735	-5.96	0.000	-5.662954	-2.859525
TD	-2.360206	.6618118	-3.57	0.000	-3.657334	-1.063079
TS	-3.246154	.6395276	-5.08	0.000	-4.499605	-1.992703
CSd (CS-) Trial 4 CON	-4.513569	.713429	-6.33	0.000	-5.911864	-3.115274
TD	-2.608885	.6601958	-3.95	0.000	-3.902845	-1.314925
TS	-3.149003	.6379668	-4.94	0.000	-4.399395	-1.898611
CSd (CS-) Trial 5 CON	-4.513568	.7203079	-6.27	0.000	-5.925346	-3.101791
TD	-2.898304	.6665675	-4.35	0.000	-4.204753	-1.591856
TS	-3.073509	.6441209	-4.77	0.000	-4.335963	-1.811055
CSd (CS-) Trial 6 CON	-4.448446	.7355685	-6.05	0.000	-5.890134	-3.006758
TD	-3.306142	.6807024	-4.86	0.000	-4.640294	-1.97199
TS	-3.551314	.6577733	-5.40	0.000	-4.840526	-2.262102

CSd (CS-) Trial 7 CON	-4.088712	.7587051	-5.39	0.000	-5.575747	-2.601678
TD	-3.495315	.7021319	-4.98	0.000	-4.871469	-2.119162
TS	-3.705094	.6784716	-5.46	0.000	-5.034873	-2.375314
CSd (CS-) Trial 8 CON	-4.479077	.7890251	-5.68	0.000	-6.025538	-2.932617
TD	-3.480187	.7302141	-4.77	0.000	-4.911381	-2.048994
TS	-4.067607	.7055959	-5.76	0.000	-5.45055	-2.684665

Appendix J

Phase: Extinction
Measure: Skin Conductance Response

	Mean	Std. Error	z	p	[95% Conf.	Interval]
CSb (CS+) Trial 1 CON	.2457386	.0313613	7.84	0.000	.1842716	.3072057
TD	.2701843	.0314888	8.58	0.000	.2084674	.3319013
TS	.2501621	.0353821	7.07	0.000	.1808144	.3195097
CSb (CS+) Trial 2 CON	.1317716	.0295087	4.47	0.000	.0739355	.1896077
TD	.0735322	.0285089	2.58	0.010	.0176558	.1294086
TS	.1415524	.0332923	4.25	0.000	.0763008	.206804
CSb (CS+) Trial 3 CON	.1067584	.028541	3.74	0.000	.0508191	.1626978
TD	.0643334	.0269594	2.39	0.017	.011494	.1171728
TS	.0806037	.0324107	2.49	0.013	.01708	.1441274
CSb (CS+) Trial 4 CON	.0773338	.0272526	2.84	0.005	.0239196	.130748
TD	.0492259	.0256935	1.92	0.055	-.0011325	.0995843
TS	.0455515	.0300048	1.52	0.129	-.0132568	.1043597
CSb (CS+) Trial 5 CON	.1004763	.026295	3.82	0.000	.048939	.1520136
TD	.0104591	.0260647	0.40	0.688	-.0406267	.0615449
TS	.0555476	.0298959	1.86	0.063	-.0030472	.1141425
CSb (CS+) Trial 6 CON	.0714797	.0257338	2.78	0.005	.0210425	.121917
TD	.0437112	.0263444	1.66	0.097	-.007923	.0953453
TS	.0426946	.0292568	1.46	0.144	-.0146477	.1000369
CSb (CS+) Trial 7 CON	.1425658	.0255588	5.58	0.000	.0924715	.19266
TD	.0741527	.0247157	3.00	0.003	.0257108	.1225946
TS	.0293745	.0290813	1.01	0.312	-.0276238	.0863727
CSb (CS+) Trial 8 CON	.1095883	.0250678	4.37	0.000	.0604563	.1587203
TD	.0322153	.0257217	1.25	0.210	-.0181984	.082629
TS	.0317776	.0293723	1.08	0.279	-.0257911	.0893463
CSd (CS-) Trial 1 CON	.1241285	.0326909	3.80	0.000	.0600555	.1882016
TD	.1100243	.0302986	3.63	0.000	.0506401	.1694084
TS	.1519807	.0353821	4.30	0.000	.0826331	.2213284
CSd (CS-) Trial 2 CON	.1481438	.0295087	5.02	0.000	.0903077	.2059799
TD	.0991762	.0285089	3.48	0.001	.0432998	.1550526
TS	.1590761	.0332923	4.78	0.000	.0938244	.2243277
CSd (CS-) Trial 3 CON	.1023753	.0279048	3.67	0.000	.0476829	.1570677
TD	.0778998	.0275343	2.83	0.005	.0239335	.1318661
TS	.0633881	.0314829	2.01	0.044	.0016828	.1250934
CSd (CS-) Trial 4 CON	.1411937	.0279868	5.05	0.000	.0863405	.1960469
TD	.075372	.0256935	2.93	0.003	.0250136	.1257304
TS	.0734949	.0300048	2.45	0.014	.0146867	.1323032
CSd (CS-) Trial 5 CON	.0873846	.0256229	3.41	0.001	.0371647	.1376046
TD	.0537895	.0253593	2.12	0.034	.0040863	.1034928
TS	.0407417	.0298851	1.36	0.173	-.017832	.0993154
CSd (CS-) Trial 6 CON						
TD						
TS						

CSd (CS-) Trial 7 CON	.0572299	.0263724	2.17	0.030	.0055411	.1089188
TD	.0625789	.0254382	2.46	0.014	.0127209	.1124368
TS	.0840571	.0280272	3.00	0.003	.0291249	.1389894
CSd (CS-) Trial 8 CON	.0707019	.0266614	2.65	0.008	.0184465	.1229573
TD	.0433918	.0249792	1.74	0.082	-.0055665	.0923501
TS	.0532255	.0282825	1.88	0.060	-.0022073	.1086582

Appendix K

Phase: Extinction
Measure: Acoustic Startle Response

		Mean	Std. Error	z	p	[95% Conf.	Interval]
CSb (CS+) Trial 1	CON	5.426099	.3101157	17.50	0.000	4.818283	6.033914
	TD	5.432919	.258388	21.03	0.000	4.926488	5.93935
	TS	5.060379	.2613115	19.37	0.000	4.548218	5.57254
CSb (CS+) Trial 2	CON	5.108118	.3031212	16.85	0.000	4.514011	5.702224
	TD	5.033357	.259754	19.38	0.000	4.524249	5.542466
	TS	4.693011	.2602997	18.03	0.000	4.182833	5.20319
CSb (CS+) Trial 3	CON	5.164227	.3088123	16.72	0.000	4.558966	5.769488
	TD	5.134769	.2637059	19.47	0.000	4.617915	5.651623
	TS	4.615536	.2642513	17.47	0.000	4.097613	5.133459
CSb (CS+) Trial 4	CON	5.195942	.3073136	16.91	0.000	4.593619	5.798266
	TD	4.958738	.2633417	18.83	0.000	4.442598	5.474878
	TS	4.466507	.26388	16.93	0.000	3.949312	4.983703
CSb (CS+) Trial 5	CON	4.913416	.3098962	15.86	0.000	4.306031	5.520801
	TD	4.618356	.2655519	17.39	0.000	4.097884	5.138828
	TS	4.538659	.2660857	17.06	0.000	4.017141	5.060178
CSb (CS+) Trial 6	CON	4.734442	.316488	14.96	0.000	4.114137	5.354747
	TD	4.6408	.2702603	17.17	0.000	4.1111	5.170501
	TS	4.351303	.2685596	16.20	0.000	3.824936	4.87767
CSb (CS+) Trial 7	CON	4.855552	.3197075	15.19	0.000	4.228937	5.482167
	TD	4.612999	.2707709	17.04	0.000	4.082298	5.1437
	TS	3.812282	.2712944	14.05	0.000	3.280475	4.34393
CSb (CS+) Trial 8	CON	4.924786	.327689	15.03	0.000	4.282528	5.567045
	TD	4.445915	.2737644	16.24	0.000	3.909347	4.982484
	TS	4.120612	.2766157	14.90	0.000	3.578455	4.662769
CSd (CS-) Trial 1	CON	5.440794	.301525	18.04	0.000	4.849816	6.031772
	TD	5.108082	.258388	19.77	0.000	4.601651	5.614513
	TS	4.795796	.261261	18.36	0.000	4.283734	5.307858
CSd (CS-) Trial 2	CON	5.401881	.3031212	17.82	0.000	4.807774	5.995987
	TD	4.624292	.259754	17.80	0.000	4.115183	5.1334
	TS	4.552023	.2602997	17.49	0.000	4.041845	5.062201
CSd (CS-) Trial 3	CON	5.254087	.3050528	17.22	0.000	4.656195	5.85198
	TD	5.010154	.2614071	19.17	0.000	4.497806	5.522503
	TS	4.542471	.2619493	17.34	0.000	4.02906	5.055882
CSd (CS-) Trial 4	CON	4.925848	.3154611	15.61	0.000	4.307555	5.54414
	TD	4.813138	.2633417	18.28	0.000	4.296998	5.329278
	TS	4.455729	.2661024	16.74	0.000	3.934178	4.97728
CSd (CS-) Trial 5	CON	4.892984	.3098962	15.79	0.000	4.285599	5.50037
	TD	4.61816	.2655519	17.39	0.000	4.097688	5.138632
	TS	4.291893	.2660857	16.13	0.000	3.770374	4.813411
CSd (CS-) Trial 6	CON	4.996075	.3127927	15.97	0.000	4.383012	5.609137
	TD	4.653872	.270273	17.22	0.000	4.124146	5.183597
	TS	4.335102	.2685596	16.14	0.000	3.808735	4.861469

CSd (CS-) Trial 7 CON	4.8566	.3196606	15.19	0.000	4.230077	5.483124
TD	4.874891	.2730147	17.86	0.000	4.339792	5.40999
TS	4.032253	.2735926	14.74	0.000	3.496021	4.568484
CSd (CS-) Trial 8 CON	4.932894	.3194924	15.44	0.000	4.3067	5.559087
TD	4.361248	.2737644	15.93	0.000	3.82468	4.897817
TS	3.858586	.2766157	13.95	0.000	3.316429	4.400743

Appendix L

Phase: Extinction Retest
Measure: US Expectancy

	Mean	Std. Error	z	p	[95% Conf.	Interval]
CSb (CS+) Trial 1 CON	.8552787	.7695941	1.11	0.266	-.653098	2.363655
TD	-.1665922	.6881154	-0.24	0.809	-1.515274	1.182089
TS	2.233432	.7128599	3.13	0.002	.8362523	3.630612
CSb (CS+) Trial 2 CON	-.821548	.7622881	-1.08	0.281	-2.315605	.6725093
TD	-.7547856	.6815785	-1.11	0.268	-2.090655	.5810838
TS	1.754349	.7060993	2.48	0.013	.3704199	3.138278
CSb (CS+) Trial 3 CON	-1.81952	.763765	-2.38	0.017	-3.316472	-.3225684
TD	-2.817252	.6829	-2.95	0.003	-3.355712	-.6787929
TS	-.5223704	.7074659	-0.74	0.460	-1.908978	.8642374
CSb (CS+) Trial 4 CON	-1.707285	.7739745	-2.21	0.027	-3.224248	-.1903233
TD	-2.800787	.6920347	-4.05	0.000	-4.15715	-1.444424
TS	-1.259577	.7169134	-1.76	0.079	-2.664701	.1455477
CSb (CS+) Trial 5 CON	.2018269	.7925792	0.25	0.799	-1.3516	1.755254
TD	1.185783	.7086806	1.67	0.094	-.2032057	2.574771
TS	.4329368	.7341297	0.59	0.555	-1.005931	1.871805
CSb (CS+) Trial 6 CON	-.4631979	.8190872	-0.57	0.572	-2.068422	1.142027
TD	-1.546807	.7323257	-2.11	0.035	-2.982139	-.1114746
TS	1.147991	.7585861	1.51	0.130	-.3388109	2.634792
CSb (CS+) Trial 7 CON	-1.376665	.8525312	-1.61	0.106	-3.047595	.2942654
TD	-2.471361	.762319	-3.24	0.001	-3.965478	-.9772429
TS	.0390712	.7896103	0.05	0.961	-1.508536	1.586679
CSb (CS+) Trial 8 CON	-2.186568	.8923518	-2.45	0.014	-3.935545	-.4375907
TD	-2.76165	.797945	-3.46	0.001	-4.325593	-1.197706
TS	-.8304086	.8264627	-1.00	0.315	-2.450246	.7894286
CSd (CS-) Trial 1 CON	-2.497837	.7695941	-3.25	0.001	-4.006214	-.9894608
TD	-1.624191	.6881154	-2.36	0.018	-2.972872	-.2755094
TS	-2.156221	.7128599	-3.02	0.002	-3.553401	-.7590418
CSd (CS-) Trial 2 CON	-2.600235	.7622881	-3.41	0.001	-4.094293	-1.106178
TD	-2.753824	.6815785	-4.04	0.000	-4.089694	-1.417955
TS	-1.493169	.7060993	-2.11	0.034	-2.877098	-.1092399
CSd (CS-) Trial 3 CON	-3.353098	.763765	-4.39	0.000	-4.85005	-1.856146
TD	-3.293491	.6829	-4.82	0.000	-4.63195	-1.955032
TS	-2.515313	.7074659	-3.56	0.000	-3.90192	-1.128705
CSd (CS-) Trial 4 CON	-3.593613	.7739745	-4.64	0.000	-5.110576	-2.076651
TD	-3.13143	.6920347	-4.52	0.000	-4.487793	-1.775067
TS	-2.876978	.7169134	-4.01	0.000	-4.282103	-1.471854
CSd (CS-) Trial 5 CON	-1.215471	.7925792	-1.53	0.125	-2.768898	.3379555
TD	-2.274365	.7086806	-3.21	0.001	-3.663354	-.8853766
TS	-.271202	.7341297	-0.37	0.712	-1.71007	1.167666
CSd (CS-) Trial 6 CON	-1.739424	.8190872	-2.12	0.034	-3.344649	-.1341999
TD	-1.867871	.7323257	-2.55	0.011	-3.303203	-.4325392
TS	-1.613214	.7585861	-2.13	0.033	-3.100016	-.1264126

CSd (CS-) Trial 7 CON	−2.361676	.8525312	−2.77	0.006	−4.032606	−.6907457
TD	−2.999079	.762319	−3.93	0.000	−4.493196	−1.504961
TS	−1.92839	.7896103	−2.44	0.015	−3.475997	−.3807819
CSd (CS-) Trial 8 CON	−2.855596	.8923518	−3.20	0.001	−4.604574	−1.106619
TD	−3.496898	.797945	−4.38	0.000	−5.060842	−1.932955
TS	−2.309197	.8264627	−2.79	0.005	−3.929034	−.6893597

Appendix M

Phase: Extinction Retest
Measure: Skin Conductance Response

	Mean	Std. Error	z	p	[95% Conf.	Interval]
CSb (CS+) Trial 1 CON	.1842396	.0392806	4.69	0.000	.1072511	.2612281
TD	.2981076	.037942	7.86	0.000	.2237426	.3724725
TS	.2536937	.0443206	5.72	0.000	.1668269	.3405606
CSb (CS+) Trial 2 CON	.0859163	.0385019	2.23	0.026	.010454	.1613786
TD	.1942253	.0362344	5.36	0.000	.1232072	.2652433
TS	.1506474	.0437232	3.45	0.001	.0649515	.2363433
CSb (CS+) Trial 3 CON	.148476	.0380421	3.90	0.000	.0739149	.2230371
TD	.1338879	.035638	3.76	0.000	.0640387	.203737
TS	.1203175	.0406303	2.96	0.003	.0406836	.1999513
CSb (CS+) Trial 4 CON	.1801904	.0348014	5.18	0.000	.1119808	.2483999
TD	.1255094	.0336139	3.73	0.000	.0596274	.1913914
TS	.0876125	.0423609	2.07	0.039	.0045867	.1706383
CSb (CS+) Trial 5 CON	.0651807	.0373239	1.75	0.081	-.0079728	.1383343
TD	.1625469	.0346495	4.69	0.000	.0946351	.2304587
TS	.2524807	.0397376	6.35	0.000	.1745964	.330365
CSb (CS+) Trial 6 CON	.0950466	.0333983	2.85	0.004	.0295871	.1605061
TD	.1233586	.0331634	3.72	0.000	.0583594	.1883577
TS	.0970487	.0391772	2.48	0.013	.0202628	.1738347
CSb (CS+) Trial 7 CON	.1196542	.0354609	3.37	0.001	.0501522	.1891562
TD	.1481824	.0321103	4.61	0.000	.0852474	.2111174
TS	.1108784	.0375132	2.96	0.003	.0373539	.1844028
CSb (CS+) Trial 8 CON	.0870592	.0357718	2.43	0.015	.0169477	.1571707
TD	.0670194	.0323264	2.07	0.038	.0036609	.1303778
TS	.0695225	.0377654	1.84	0.066	-.0044962	.1435413
CSd (CS-) Trial 1 CON	.2006589	.0402754	4.98	0.000	.1217206	.2795972
TD	.2544058	.037942	6.71	0.000	.1800409	.3287708
TS	.3247203	.0443206	7.33	0.000	.2378535	.4115871
CSd (CS-) Trial 2 CON	.1020344	.0385019	2.65	0.008	.0265721	.1774967
TD	.1585016	.0362344	4.37	0.000	.0874835	.2295197
TS	.1289316	.0423272	3.05	0.002	.0459718	.2118914
CSd (CS-) Trial 3 CON	.1475649	.0369683	3.99	0.000	.0751085	.2200214
TD	.1682578	.0347807	4.84	0.000	.1000889	.2364268
TS	.1441739	.0406303	3.55	0.000	.0645401	.2238078
CSd (CS-) Trial 4 CON	.1020549	.0348014	2.93	0.003	.0338454	.1702644
TD	.0914649	.0336139	2.72	0.007	.0255829	.1573469
TS	.1267279	.0406841	3.11	0.002	.0469885	.2064673
CSd (CS-) Trial 5 CON	.0891057	.0349143	2.55	0.011	.0206749	.1575364
TD	.1479581	.0346524	4.27	0.000	.0800406	.2158756
TS	.1906649	.0397083	4.80	0.000	.112838	.2684917
CSd (CS-) Trial 6 CON	.1153588	.0368629	3.13	0.002	.0431089	.1876088
TD	.0966714	.032258	3.00	0.003	.0334468	.159896
TS	.1111132	.0376856	2.95	0.003	.0372508	.1849757

CSd (CS-) Trial 7 CON	.1083634	.0342775	3.16	0.002	.0411807	.175546
TD	.0476672	.0330385	1.44	0.149	-.0170871	.1124216
TS	.079569	.0375132	2.12	0.034	.0060445	.1530935
CSd (CS-) Trial 8 CON	.1042171	.037141	2.81	0.005	.031422	.1770122
TD	.0983756	.0343549	2.86	0.004	.0310413	.1657099
TS	.1479367	.0377654	3.92	0.000	.0739179	.2219554

Appendix N

Phase: Extinction Retest
Measure: Acoustic Startle Reflex

	Mean	Std. Error	z	p	[95% Conf.	Interval]
CSb (CS+) Trial 1 CON	4.869382	.2996169	16.25	0.000	4.282143	5.45662
TD	5.126884	.2448363	20.94	0.000	4.647014	5.606755
TS	4.897817	.2558854	19.14	0.000	4.396291	5.399343
CSb (CS+) Trial 2 CON	4.998762	.3005172	16.63	0.000	4.409759	5.587765
TD	4.928249	.2501012	19.71	0.000	4.43806	5.418438
TS	4.978064	.254152	19.59	0.000	4.479935	5.476192
CSb (CS+) Trial 3 CON	4.791091	.302216	15.85	0.000	4.198759	5.383424
TD	4.469888	.2490462	17.95	0.000	3.981767	4.95801
TS	4.747878	.2578767	18.41	0.000	4.242449	5.253307
CSb (CS+) Trial 4 CON	4.583217	.3046997	15.04	0.000	3.986016	5.180417
TD	4.550656	.2489831	18.28	0.000	4.062658	5.038654
TS	4.75421	.2576845	18.45	0.000	4.249158	5.259263
CSb (CS+) Trial 5 CON	5.048656	.3079495	16.39	0.000	4.445087	5.652226
TD	4.761741	.2516344	18.92	0.000	4.268547	5.254935
TS	4.788834	.2604293	18.39	0.000	4.278402	5.299267
CSb (CS+) Trial 6 CON	4.522945	.3119413	14.50	0.000	3.911551	5.134338
TD	4.547652	.2548913	17.84	0.000	4.048074	5.04723
TS	4.756369	.2638009	18.03	0.000	4.239329	5.27341
CSb (CS+) Trial 7 CON	4.634189	.3166472	14.64	0.000	4.013572	5.254806
TD	4.377164	.2587307	16.92	0.000	3.870061	4.884267
TS	4.650381	.2677757	17.37	0.000	4.12555	5.175212
CSb (CS+) Trial 8 CON	4.406656	.3220358	13.68	0.000	3.775478	5.037835
TD	4.59166	.2631273	17.45	0.000	4.07594	5.10738
TS	4.329275	.2723272	15.90	0.000	3.795524	4.863027
CSd (CS-) Trial 1 CON	4.895961	.2996169	16.34	0.000	4.308723	5.4832
TD	5.102937	.2448363	20.84	0.000	4.623067	5.582807
TS	5.025465	.2533915	19.83	0.000	4.528827	5.522103
CSd (CS-) Trial 2 CON	4.513587	.3005172	15.02	0.000	3.924585	5.10259
TD	4.641365	.2477234	18.74	0.000	4.155836	5.126894
TS	4.596996	.254152	18.09	0.000	4.098867	5.095124
CSd (CS-) Trial 3 CON	4.580102	.302216	15.16	0.000	3.987769	5.172434
TD	4.759496	.2469567	19.27	0.000	4.275469	5.243522
TS	4.770548	.2578767	18.50	0.000	4.265119	5.275977
CSd (CS-) Trial 4 CON	4.453311	.3085443	14.43	0.000	3.848575	5.058047
TD	4.765385	.2489831	19.14	0.000	4.277387	5.253383
TS	4.66169	.2576845	18.09	0.000	4.156638	5.166742
CSd (CS-) Trial 5 CON	4.731849	.3079495	15.37	0.000	4.128279	5.335419
TD	4.612879	.2516344	18.33	0.000	4.119684	5.106073
TS	4.634342	.2626841	17.64	0.000	4.119491	5.149194
CSd (CS-) Trial 6 CON	4.653624	.3119413	14.92	0.000	4.04223	5.265017
TD	4.399383	.2548913	17.26	0.000	3.899805	4.898896
TS	4.637208	.2660504	17.43	0.000	4.115759	5.158657

CSd (CS-) Trial 7	CON	4.564632	.3205007	14.24	0.000	3.936462	5.192802
	TD	4.508052	.2607628	17.29	0.000	3.996967	5.019138
	TS	4.642602	.2677757	17.34	0.000	4.117772	5.167433
CSd (CS-) Trial 8	CON	4.699615	.3220358	14.59	0.000	4.068436	5.330793
	TD	4.339628	.2631273	16.49	0.000	3.823908	4.855349
	TS	4.616318	.2723272	16.95	0.000	4.082566	5.150069

Appendix O

Subjective Units of Discomfort (SUD) across experimental phase.

		Mean	Std. Error	z	p	[95% Conf.	Interval]
CON	Baseline	24.96169	5.775436	4.32	0.000	13.64205	36.28134
	Pre-Acq	37.87836	5.751722	6.59	0.000	26.60519	49.15153
	Post-Acq	49.96169	5.7281	8.72	0.000	38.73482	61.18856
	Post-ABMT	35.79503	5.704573	6.27	0.000	24.61427	46.97578
	Post-EXT	24.12836	5.68114	4.25	0.000	12.99353	35.26319
	Pre-Ext Retest	18.71169	5.657804	3.31	0.001	7.622601	29.80078
	Post-Ext Retest	33.71169	5.634565	5.98	0.000	22.66815	44.75524
TD	Baseline	19.43783	5.379847	3.61	0.000	8.89352	29.98213
	Pre-Acq	33.44313	5.141858	6.50	0.000	23.36527	43.52099
	Post-Acq	42.3098	5.12072	8.26	0.000	32.27337	52.34622
	Post-ABMT	40.64313	5.099665	7.97	0.000	30.64797	50.63829
	Post-EXT	26.1098	5.078695	5.14	0.000	16.15574	36.06386
	Pre-Ext Retest	14.64313	5.057811	2.90	0.004	4.730003	24.55626
	Post-Ext Retest	27.44313	5.037015	5.45	0.000	17.57076	37.3155
TS	Baseline	11.73229	5.005633	2.34	0.019	1.921426	21.54315
	Pre-Acq	18.23229	4.985112	3.66	0.000	8.461646	28.00293
	Post-Acq	36.29479	4.964672	7.31	0.000	26.56421	46.02536
	Post-ABMT	29.41979	4.944313	5.95	0.000	19.72911	39.11046
	Post-EXT	18.66979	4.924036	3.79	0.000	9.018853	28.32072
	Pre-Ext Retest	3.145941	5.106652	0.62	0.538	-6.862914	13.1548
	Post-Ext Retest	15.6508	5.087033	3.08	0.002	5.680396	25.6212

Appendix P

Self-reported arousal (fearfulness) to CS across experimental phases.

	Mean	Std. Error	z	p	[95% Conf.	Interval]
CSb Pre-Acq CON	1.870125	.567784	3.29	0.001	.7572892	2.982961
Pre-Acq TD	2.037896	.5076811	4.01	0.000	1.042859	3.032932
Pre-Acq TS	1.990917	.4919401	4.05	0.000	1.026732	2.955102
Post-Acq CON	5.536792	.5460186	10.14	0.000	4.466615	6.606969
Post-Acq TD	3.104562	.4882071	6.36	0.000	2.147694	4.061431
Post-Acq TS	4.428417	.4730996	9.36	0.000	3.501158	5.355675
Post-ABMT CON	2.536792	.5303065	4.78	0.000	1.49741	3.576174
Post-ABMT TD	1.037896	.4741489	2.19	0.029	.1085809	1.96721
Post-ABMT TS	.3659167	.4594995	0.80	0.426	−.5346858	1.266519
Post-Ext CON	2.786792	.5211955	5.35	0.000	1.765268	3.808317
Post-Ext TD	1.771229	.4659968	3.80	0.000	.8578921	2.684566
Post-Ext TS	.9294717	.4622836	2.01	0.044	.0234124	1.835531
Pre-Ext Retest CON	2.453459	.5190334	4.73	0.000	1.436172	3.470745
Pre-Ext Retest TD	.571229	.4640622	1.23	0.218	−.3383161	1.480774
Pre-Ext Retest TS	1.056301	.4739656	2.23	0.026	.1273453	1.985256
Post-Ext Retest CON	2.203459	.5239061	4.21	0.000	1.176622	3.230296
Post-Ext Retest TD	1.837896	.4684221	3.92	0.000	.9198053	2.755986
Post-Ext Retest TS	2.387843	.4938704	4.83	0.000	1.419874	3.355811
CSd Pre-Acq CON	1.536792	.567784	2.71	0.007	.4239559	2.649628
Pre-Acq TD	1.971229	.5076811	3.88	0.000	.9761924	2.966266
Pre-Acq TS	1.178417	.4919401	2.40	0.017	.2142319	2.142602
Post-Acq CON	.786792	.5460186	1.44	0.150	−.2833847	1.856969
Post-Acq TD	1.971229	.4882071	4.04	0.000	1.014361	2.928097
Post-Acq TS	.8659167	.4730996	1.83	0.067	−.0613416	1.793175
Post-ABMT CON	.8701254	.5303065	1.64	0.101	−.1692563	1.909507
Post-ABMT TD	.6378957	.4741489	1.35	0.179	−.2914191	1.56721
Post-ABMT TS	.3659167	.4594995	0.80	0.426	−.5346858	1.266519
Post-Ext CON	1.120125	.5211955	2.15	0.032	.0986009	2.14165
Post-Ext TD	1.437896	.4659968	3.09	0.002	.5245588	2.351233
Post-Ext TS	.3294717	.4622836	0.71	0.476	−.5765876	1.235531
Pre-Ext Retest CON	.6201254	.5190334	1.19	0.232	−.3971614	1.637412
Pre-Ext Retest TD	.6378957	.4640622	1.37	0.169	−.2716494	1.547441
Pre-Ext Retest TS	.2705864	.4739656	0.57	0.568	−.658369	1.199542
Post-Ext Retest CON	1.536792	.5239061	2.93	0.003	.509955	2.563629
Post-Ext Retest TD	1.437896	.4684221	3.07	0.002	.5198053	2.355986
Post-Ext Retest TS	1.310919	.4938704	2.65	0.008	.3429512	2.278888

Appendix Q

Self-reported valence (unpleasantness) to CS across experimental phases.

	Mean	Std. Error	z	p	[95% Conf.	Interval]
CSb Pre-Acq CON	1.968632	.6038148	3.26	0.001	.7851769	3.152087
Pre-Acq TD	2.513942	.5398437	4.66	0.000	1.455868	3.572017
Pre-Acq TS	2.098502	.5232338	4.01	0.000	1.072982	3.124021
Post-Acq CON	6.218632	.5968519	10.42	0.000	5.048824	7.38844
Post-Acq TD	3.647276	.5336132	6.84	0.000	2.601413	4.693138
Post-Acq TS	5.598502	.5172074	10.82	0.000	4.584794	6.61221
Post-ABMT CON	2.801966	.5979887	4.69	0.000	1.629929	3.974002
Post-ABMT TD	.9139423	.5346305	1.71	0.087	−.1339141	1.961799
Post-ABMT TS	1.473502	.5181913	2.84	0.004	.4578654	2.489138
Post-Ext CON	2.801966	.6071797	4.61	0.000	1.611915	3.992016
Post-Ext TD	2.180609	.5428546	4.02	0.000	1.116633	3.244585
Post-Ext TS	1.221815	.5373085	2.27	0.023	.1687093	2.27492
Pre-Ext Retest CON	2.551966	.6240693	4.09	0.000	1.328812	3.775119
Pre-Ext Retest TD	.8472756	.5579672	1.52	0.129	−.2463199	1.940871
Pre-Ext Retest TS	1.74254	.5659157	3.08	0.002	.6333656	2.851714
Post-Ext Retest CON	2.801966	.6480557	4.32	0.000	1.5318	4.072131
Post-Ext Retest TD	2.447276	.5794293	4.22	0.000	1.311615	3.582936
Post-Ext Retest TS	2.369425	.6026702	3.93	0.000	1.188214	3.550637
CSd Pre-Acq CON	1.885299	.6038148	3.12	0.002	.7018436	3.068754
Pre-Acq TD	2.047276	.5398437	3.79	0.000	.9892014	3.10535
Pre-Acq TS	1.411002	.5232338	2.70	0.007	.3854823	2.436521
Post-Acq CON	1.468632	.5968519	2.46	0.014	.298824	2.63844
Post-Acq TD	2.247276	.5336132	4.21	0.000	1.201413	3.293138
Post-Acq TS	1.286002	.5172074	2.49	0.013	.2722939	2.29971
Post-ABMT CON	.9686322	.5979887	1.62	0.105	−.2034041	2.140668
Post-ABMT TD	.580609	.5346305	1.09	0.277	−.4672475	1.628465
Post-ABMT TS	1.161002	.5181913	2.24	0.025	.1453654	2.176638
Post-Ext CON	1.051966	.6071797	1.73	0.083	−.1380849	2.242016
Post-Ext TD	2.047276	.5428546	3.77	0.000	.9833001	3.111251
Post-Ext TS	.7551479	.5373085	1.41	0.160	−.2979574	1.808253
Pre-Ext Retest CON	.6352988	.6240693	1.02	0.309	−.5878545	1.858452
Pre-Ext Retest TD	.580609	.5579672	1.04	0.298	−.5129866	1.674205
Pre-Ext Retest TS	.5996829	.5659157	1.06	0.289	−.5094915	1.708857
Post-Ext Retest CON	1.801966	.6480557	2.78	0.005	.5317996	3.072131
Post-Ext Retest TD	1.713942	.5794293	2.96	0.003	.5782817	2.849603
Post-Ext Retest TS	1.600195	.6026702	2.66	0.008	.4189827	2.781407

References

Alberts, H., & Thewissen, R. (2011). The effect of a brief mindfulness intervention on memory for positively and negatively valenced stimuli. Mindfulness, 2, 73–77.

Arch, J. J., & Craske, M. G. (2006). Mechanisms of mindfulness: Emotion regulation following a focused breathing induction. Behaviour Research and Therapy, 44, 1849–1858.

Baer, R. A., Smith, G. T., Hopkins, J., Krietemeyer, J., & Toney, L. (2006). Using self-report assessment methods to explore facets of mindfulness. Assessment, 13, 27–45.

Baer, R. A., Smith, G. T., Lykins, E., Button, D., Krietemeyer, J., Sauer, S., et al. (2008). Construct validity of the Five Facet Mindfulness Questionnaire in meditating and non meditating samples. Assessment, 15, 329–342.

Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, & van IJzendoorn, M. H. (2007). Threat-related attentional bias in anxious and non-anxious individuals: A meta-analytic study. Psychological Bulletin, 133, 1–24.

Barlow, D. H., Raffa, S. D., & Cohen, E. M. (2002). Psychosocial treatments for panic disorders, phobias, and generalized anxiety disorder. In: P.C. Nathan & J.M. Gorman (Eds.), A guide to treatments that work (pp. 301-335). London: Oxford University Press.

Barrett, A. & Armony, J.L. (2009). Influence of trait anxiety on brain activity during the acquisition and extinction of aversive conditioning. Psychological Medicine, 39:255-265.

Beck AT, Ward CH, Mendelson M, Mock J, & Erbaugh J (1961) An inventory for measuring depression. Archives of General Psychiatry, 4, 561-571.

Beck, A. T. (1976). *Cognitive therapy and emotional disorders.* New York: International Universities Press.

Bishop, S. R., Lau, M., Shapiro, S., Carlson, L., Anderson, N. D., Carmody, J., et al. (2004). Mindfulness: A proposed operational definition. Clinical Psychology: Science and Practice, 11, 230–241.

Bishop, S., J.Duncan,M. Brett, et al. (2004). Prefrontal cortical function and anxiety: controlling attention to threat related stimuli. Nat. Neurosci,.7: 184–188.

Bishop, S.J., J. Duncan & A.D. Lawrence. (2004). State anxiety modulation of the amygdala response to unattended threat-related stimuli. J. Neurosci. 24: 10364–10368.

Bishop, S.J., J.D. Cohen, J. Fossella, et al.(2006). COMT genotype influences prefrontal response to emotional distraction. Cogn. Affect. Behav. Neurosci. 6: 62–70.

Bishop, S.J., R. Jenkins, A.D. Lawrence. (2007). Neural processing of fearful faces: Effects of anxiety are gated by perceptual capacity limitations. Cereb Cortex 17: 1595– 1603.

Bradley, B. P., Mogg, K., White, J., Groom, C., & de Bono, J. (1999). Attentional bias for emotional faces in generalized anxiety disorder. British Journal of Clinical Psychology, 38, 267–278.

Bremner, J.D. et al. (2005) Positron emission tomographic imaging of neural correlates of a fear acquisition and extinction paradigm in women with childhood sexual- abuse-related post-traumatic stress disorder. Psychol. Med. 35, 791–806

Brown, K. W., & Ryan, R. M. (2003). The benefits of being present: Mindfulness

and its role in psychological well-being. Journal of Personality and Social Psychology, 84, 822–848.

Brown, K.W. & Ryan, R.M. (2004). Perils and promise in defining and measuring mindfulness: Observations from experience. Clinical Psychology: Science and Practice, 11, pp 242-24.

Buckley, T. C., Blanchard, E. B., & Neill, W. T. (2000). Information processing and PTSD: A review of the empirical literature. Clinical Psychology Review, 20, 1041–1065

Campbell-Sills, L., Barlow, D. H., Brown, T. A., & Hofmann, S. G. (2006). Effects of suppression and acceptance on emotional responses of individuals with anxiety and mood disorders. Behaviour Research and Therapy, 44, 1251–1263.

Carlsson, K., Andersson, J., Petrovic, P., Petersson, K.M., Ohman, A. & Ingvar, M. (2006) Predictability modulates the affective and sensory-discriminative neural processing of pain. Neuroimage, 32, 1804–1814.

Carmody, J., Baer, R. A., Lykins, E. L. B., & Olendzki, N. (2009). An empirical study of the mechanisms of mindfulness in a mindfulness-based stress reduction program.
Journal of Clinical Psychology, 65, 613–626.

Carver CS & White TL (1994) Behavioral inhibition, behavior activation, and affective responses to impending reward and punishment: The BIS/BAS scales. Journal of Personality and Social Psychology, 67, 319-333.

Chambless, D. L., & Hollon, S. D. (1998). Defining empirically supported therapies. Journal of Consulting and Clinical Psychology, 66, 7-18.

Clark, D. M., & McManus, F. (2002). Information processing in social phobia. Biological Psychiatry, 51, 92–100.

Craske, M. G. (1999). Anxiety disorders: psychological approaches to theory and treatment. Boulder, CO: Westview Press.

Davis, M., Falls, W. A., & Gewirtz, J. (2000). Neural systems involved in fear inhibition: extinction and conditioned inhibition. In M. Myslobodsky & I. Weiner (Eds.), Contemporary issues in modeling psychopathology (pp. 113–142).

Dawson, M.E. and Reardon, P. (1973). Construct validity of recall and recognition-post-conditioning measures of awareness. J. Exp. Psychol. 98: 308 -315.

Delizonna, Laura L., Ryan P. Williams, and Ellen J. Langer. 2009. The effect of mindfulness on heart rate control. Journal of Adult Development 16(2): 61-65.

Dickie EW, Armony JL. (2008). Amygdala responses to unattended fearful faces: interaction between sex and trait anxiety. Psychiatry Res., 162:51–57.

Ehrenreich, J. T., & Gross, A. M. (2002). Biased attentional behavior in childhood anxiety: A review of theory and current empirical investigation. Clinical Psychology Review, 22, 991–1008.

Eifert, G. H., & Heffner, M. (2003). The effects of acceptance versus control contexts on avoidance of panic-related symptoms. Journal of Behavior Therapy and Experimental Psychiatry, 34, 293–312.

Erisman, S. M., & Roemer, L. (2010). A preliminary investigation of the effects of experimentally induced mindfulness on emotional responding to film clips. Emotion, 10, 72– 82.

Etkin A, Klemenhagen KC, Dudman JT, Rogan MT, Hen R, Kandel ER, Hirsch J: Individual differences in trait anxiety predict the response of the basolateral amygdala to unconsciously processed fearful faces. Neuron 2004, 44:1043-1055.

Eysenck, H. J. (1979). The conditioning model of neurosis. Behavioral and Brain Sciences, 2, 155–199.

Eysenck, H. J. (1992). Four ways five factors are *not* basic. *Personality and Individual Differences, 13,* 667-673.

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. Emotion, 7, 409–434.

Grillon, C. (2002). Associative learning deficits increase symptoms of anxiety in Humans. Biological 851–858. Psychiatry, 51,

Hakamata Y., Lissek S., Bar-Haim Y., Britton J. C., Fox N. A., Leibenluft E., Ernst M., Pine D. S. (2010). Attention bias modification treatment: a meta-analysis toward the establishment of novel treatment for anxiety. *Biol. Psychiatry* 68, 982–990.

Hallion LS, Ruscio AM *et al* (2011) A meta-analysis of the effect of cognitive bias modification on anxiety and depression. *Psychol Bull*. 137. 940-958.

Hargus, E., Crane, C., Barnhofer, T., & Williams, J. M. G. (2010). Effects of mindfulness on meta-awareness and specificity of describing prodromal symptoms in suicidal depression. Emotion, 1, 34–42.

Hayes, A. M., & Feldman, G. (2004). Clarifying the construct of mindfulness in the context of emotion regulation and the process of change in therapy. Clinical Psychology: Science and Practice, 11, 255–262.

Hayes, S. C. (1994). Content, context, and the types of psychological

acceptance.

In S. C. Hayes, N. S. Jacobson, V. M. Follette, & M. J. Dougher (Eds.), Acceptance and change: Content and context in psychotherapy (pp. 13–32). Reno, NV: Context Press.

Hayes, S. C., Luoma, J. B., Bond, F. W., Masuda, A., & Lillis, J. (2006). Acceptance and commitment therapy: Model, processes, and outcomes. Behaviour Research and Therapy, 44, 1–25.

Heinrichs, N., & Hofman, S. G. (2001). Information processing in social review. Clinical Psychology Review, 21, 751–770.

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

Herndon, F. (2008). Testing mindfulness with perceptual and cognitive factors: External vs. internal encoding, and the cognitive failures questionnaire. Personality and Individual Differences, 44, 32–41.

Hodgins, H. S., & Adair, K. C. (2010). Attentional processes and meditation. Consciousness and Cognition, 19, 872–878.

Hull, A.M. (2002). Neuroimaging findings in post-traumatic stress disorder. Systematic review. Br. J. Psychiatry , 181, 102-110

Jain, S., Shapiro, S. L., Swanick, S., Roesch, S. C., Mills, P. J., Bell, I., et al. (2007). A randomized controlled trial of mindfulness meditation versus relaxation training: Effects on distress, positive states of mind, rumination, and distraction. Annals of Behavioral Medicine, 33, 11–21.

Jha, A. P., Stanley, E. A., Kiyonaga, A., Wong, L., & Gelfand, L. (2010).

Examining the protective effects of mindfulness training on working memory capacity and affective experience. Emotion, 10, 54–64.

Josefsson, T., Larsman, P., Broberg, A., & Lundh, L. -G. (2011). Self-reported mindfulness mediates the relation between meditation experience and psychological well-being. Mindfulness, 2, 49–58.

Kabat-Zinn, J. (1982). An outpatient program in behavioral medicine for chronic pain patients based on the practice of mindfulness meditation: Theoretical considerations and preliminary results. General Hospital Psychiatry, 4, 33–47.

Kabat-Zinn, J. (1990). Full catastrophe living: How to cope with stress, pain and illness using mindfulness meditation. New York: NY: Bantam Dell.

Keng, S.; Smoski, M.; Robins, C. Effects of mindfulness on psychological health: A review of empirical studies. Clin. Psychol. Rev. 2011, 31, 1041–1056.

Koster EHW, Crombez G, Verschuere B, De Houwer J. Attention to threat in anxiety-prone individuals: mechanisms underlying attentional bias. *Cognitive Therapy and Research.*2006b;30:635–643.

Koster, E.H.W., Crombez, G., Van Damme, S., Verschuere, B., De Houwer., J. (2005). Signals for threat modulate attentional capture and holding: fear-conditioning and extinction during the exogenous cueing task. Cognition and Emotion, 19, 771–780.

LaBar, K.S. et al. (1998) Human amygdala activation during conditioned fear acquisition and extinction: a mixed-trial fMRI study. Neuron 20, 937–945

LeDoux JE. (2000). Emotion circuits in the brain. Annu Rev Neurosci 23: 155-184.

Levitt, J. T., Brown, T. A., Orsillo, S. M., & Barlow, D. H. (2004). The effects of

acceptance versus suppression of emotion on subjective and psychophysiological response to carbon dioxide challenge in patients with panic disorder. Behavior Therapy, 35, 747–766.

Lewicki, P. Internal and external encoding style and social motivation. J.P. Forgas, K.D. Williams, S.M. Laham (Eds.), Social Motivation: Conscious and Unconscious Processes, Cambridge University Press, Cambridge, MA (2005), pp. 194–209.

Linehan, M. (1993). Cognitive-behavioral treatment of borderline personality disorder. New York: Guilford Press.

Lipp, O. V., Sheridan, J., and Siddle, A. T. (1994). Human blink startle during aversive and nonaversive Pavlovian conditioning. Journal of Experimental Psychology: Animal Behavior Processes 20, 380-389.

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(11), 1391–1424.

Lykins, E., & Baer, R. A. (2009). Psychological functioning in a sample of long-term practitioners of mindfulness meditation. Journal of Cognitive Psychotherapy: An International Quarterly, 23, 226–241.

Mackintosh, N.J. (1975). A theory of attention: Variations in the associability of stimuli with reinforcement. Psychological Review, 82, 276-298.

MacLeod C, Mathews A, Tata P. (1986). Attentional bias in the emotional disorders. Journal of Abnormal Psychology. 95:15–20.

MacLeod C, Rutherford E, Campbell L, Ebsworthy G, Holker L. (2002). Selective attention and emotional vulnerability: assessing the causal basis of their

association through the experimental manipulation of attentional bias. J. Abnorm. Psychol. 111:107– 2.

Maren S, Quirk GJ. (2004). Neuronal signaling of fear memory. Nat Rev Neurosci 5: 844–852.

Mathews, A. (1990). Why worry? The cognitive function of anxiety. Behaviour Research and Therapy, 28, 455-468

Mathews, A., & Mackintosh, B. (1998). A cognitive model of selective processing in anxiety. Cognitive Therapy and Research, 22, 539–560.

Mathews, A., & MacLeod, C. (2002). Induced processing biases have causal effects on anxiety. Cognition and Emotion, 16, 310–315.

McNally, R. J. (1999). Panic and phobias. In T. Dalgleish & M. J. Power (Eds.), Handbook of cognition and emotion (pp. 479 – 496). New York: Wile.

Milad MR, Wright CI, Orr SP, Pitman RK, Quirk GJ, Rauch SL. (2007). Recall of fear extinction in humans activates the ventromedial prefrontal cortex and hippocampus in concert. Biol Psychiatry 62: 446–454.

Miller, Ralph R.; Barnet, Robert C.; Grahame, Nicholas J. (1995). "Assessment of the Rescorla-Wagner Model". Psychological Bulletin (American Psychological Association) 117 (3): 363–386.

Mineka, S., & Zinbarg, R. (1996). Conditioning and ethological models of anxiety disorders: stress-in-dynamic-context anxiety models. In D. A. Hope (Ed.), Nebraska symposium on motivation: Vol. 43. Perspectives on anxiety, panic, and fear. Lincoln: University of Nebraska Press.

Mogg K, Bradley BP. Some methodological issues in assessing attentional

biases for threatening faces in anxiety: a replication study using a modified version of the probe detection task. Behaviour Research and Therapy. 1999;37:595–604.

Mogg, K., & Bradley, B. (1998). A cognitive-motivational analysis of anxiety. Behaviour Research and Therapy, 36, 809-848

Mogg, K., & Bradley, B. P. (2005). Attentional bias in generalized anxiety disorder versus depressive disorder. Cognitive Therapy and Research, 29, 29 – 45.

Mor, N., & Winquist, J. (2002). Self-focused attention and negative affect: A meta- analysis. Psychological Bulletin, 128, 638-662.

Musa, C. Z., & Lepine, J. P. (2000). Cognitive aspects of social phobia: A review of theories and experimental research. European Psychiatry, 15, 59 – 66

Ortner, C. N. M., Kilner, S. J., & Zelazo, P. D. (2007). Mindfulness meditation and reduced emotional interference on a cognitive task. Motivation and Emotion, 31, 271–283.

Pearce J.M. and Hall G. (1980) A model for Pavlovian learning: Variations in the effectiveness of conditioned but not of unconditioned stimuli. Psychological Review, 87:532- 552.

Phelps EA, Delgado MR, Nearing KI, et al. (2004). Extinction learning in humans: role of the amygdala and vmPFC. Neuron, 43:897–905.

Phelps, E.A., O'Connor, K.J., Gatenby, J.C., Grillon, C., Gore, J.C. & Davis, M. (2001). Activation of the human amygdala to a cognitive representation of fear. Nature Neuroscience, 4, 437-441.

Pischek-Simpson LK, Boschen MJ, Neumann DL, et al. The development of an attentional bias for angry faces following Pavlovian fear conditioning. *Behav Res*

Ther 2009;47:322–330.

Powers, M. B., Zum Vorde Sive Vording, M. B., & Emmelkamp, P. M. G. (2009). Acceptance and commitment therapy: A meta-analytic review. Psychotherapy and Psychosomatics, 78, 73–80.

Prenoveau, J.M. (2011a). Stability of and Structural Relations Among Anxiety, Depression, Neuroticism, and Extraversion During Adolescence: Results from a 3-Year Longitudinal. BiblioBazaar, USA.

Prenoveau, J.M., Craske, M.G., Liao, B., & Ornitz., E.M. (2011b). Associative Conditioning Deficits: Caused by or Cause of Anxiety? Journal of Experimental Psychopathology, 2, 432-448.

Rachman, S. (1989). The return of fear: Review and prospect. Clinical Psychology Review, 9, 147-168.

Ramel, W., Goldin, P. R., Carmona, P. E., & McQuaid, J. R. (2004). The effects of mindfulness meditation on cognitive processes and affect in patients with past depression. Cognitive Therapy and Research, 28, 433–455.

Rescorla, R.A. & Wagner, A.R. (1972) A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement, Classical Conditioning II, A.H. Black & W.F. Prokasy, Eds., pp. 64–99.

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). Mindfulness-based cognitive therapy for depression: A new approach to preventing relapse. New York, NY: Guilford Press.

Sehlmeyer C, Dannlowski U, Schöning S, Kugel H, Pyka M, Pfleiderer B, Zwitserlood P, Schiffbauer H, Heindel W, Arolt V, Konrad C. (2010). Neural correlates of

trait anxiety in fear extinction. Psychol Med. 41(4):789-98.

Seligman, M. E. (1971). Phobias and preparedness. Behavior Therapy, 2, 307-320.

Shapiro, S. L., Carlson, L. E., Astin, J. A., & Freedman, B. (2006). Mechanisms of mindfulness. Journal of Clinical Psychology, 62, 373–386.

Shin LM, Liberzon I. (2010). The Neurocircuitry of fear, stress, and anxiety disorders. Neuropsychopharmacology, 35:169–191.

Shin LM, Whalen PJ, Pitman RK, Bush G, Macklin ML, Lasko NB, Orr SP, McInerney SC, and Rauch SL. (2001). An fMRI study of anterior cingulate function in posttraumatic stress disorder. Biol Psychiatry 50: 932–942.

Sotres-Bayon F, Diaz-Mataix L, Bush DE, LeDoux JE. 2009. Dissociable roles for the ventromedial prefrontal cortex and amygdala in fear extinction: NR2B contribution. Cereb Cortex 19: 474–482.

Spielberger CD, Gorsuch RL, & Lushene RE (1970) Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press.

Summerfeldt, L. J., & Endler, N. S. (1998). Examining the evidence for anxiety- related cognitive biases in obsessive– compulsive disorder. Journal of Anxiety Disorders, 12, 579 –598.

Thayer, J.F. et al. (2012) A meta-analysis of heart rate variability and neuroimaging studies: implications for heart rate variability as a marker of stress and health. Neurosci. Biobehav. Rev. 36, 747–756.

Teasdale, J. D., Moore, R. G., Hayhurst, H., Pope, M., Williams, S., & Segal, Z. V. (2002). Metacognitive awareness and prevention of relapse in depression: Empirical

evidence. Journal of Consulting and Clinical Psychology, 70, 275–287.

Venables PH, Christie MJ (1980): Electrodermal activity. In Techniques in Psychophysiology, ed. I Martin, PH Venables, pp. 2-67, John Wiley, New York.

Waters, A. M., Henry, J., Mogg, K., Bradley, B. P., & Pine, D. S. (2010). Attentional bias towards angry faces in childhood anxiety disorders. Journal of Behavior Therapy and Experimental Psychiatry, 41, 158–164.

Watson, D., & Pennebaker, J.W. (1989). Health complaints, stress, and distress: Exploring the central role of Negative Affectivity. Psychological Review, 96, 234- 254.

Watson, J.B., Rayner, R. (1920). Conditioned emotional reactions. Journal of Experimental Psychology, 3 (1920), pp. 1–14.

Williams, J. M., Watts, F. N., MacLeod, C., & Mathews, A. (1988). Cognitive psychology and emotional disorders. Chichester, U.K.: John Wiley & Son.