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Extinction-based Processes for Enhancing the Effectiveness of Exposure Therapy

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Extinction-based Processes for Enhancing the Effectiveness of Exposure Therapy

A dissertation submitted in partial satisfaction of the requirements for the degree of Doctor of Philosophy in Psychology

by

Najwa Chowdhury Culver

2013

ABSTRACT OF THE DISSERTATION

Extinction-based Processes for Enhancing

the Effectiveness of Exposure Therapy

by

Najwa Chowdhury Culver

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2013

Professor Michelle G. Craske, Chair

The dissertation is a three-paper investigation of potential methods for improving exposure therapy outcomes for anxiety disorders. If proven effective, these methods can be utilized to modify exposure therapy for anxiety disorders in order to enhance treatment effects and reduce relapse rates.

Study 1 tested a prediction of the Rescorla-Wagner model: that presenting two fear-provoking stimuli simultaneously (compound extinction) would maximize learning during extinction. Participants were presented with single extinction trials only or single extinction trials followed by compound extinction trials. Additionally, participants were randomized to caffeine or placebo ingestion prior to extinction. Results indicated participants presented with compound trials demonstrated significantly less fear responding at spontaneous recovery whereas ingestion of caffeine provided limited

protection (only on valence ratings). At reinstatement, only compound extinction trials predicted attenuated fear responding.

Study 2 investigated whether occasional reinforced trials during extinction enhances learning, as has been demonstrated in recent literature in animal models. Participants were randomly assigned to typical exposure procedures (i.e., no CS-US pairings) or occasional CS-US pairings during extinction. Results indicated participants presented with occasional reinforced trials maintained elevated fear responding throughout exposure but demonstrated attenuated spontaneous recovery and rapid reacquisition effects at follow-up testing.

Study 3 was based on recent evidence in animal models suggesting that sustained arousal and enhanced fear responding throughout extinction predicts better long-term outcomes, which is contrary to traditional exposure therapy in which reduction of fear responding is used as an index of learning. Participants completed exposure with or without the presence of additional excitatory stimuli which were intended to enhance arousal and fear responding throughout exposure. A set of regression analyses investigating whether any exposure process measures predicted outcome indicated that sustained arousal throughout exposure and variability in subjective fear responding throughout exposure predicted lower levels of fear at follow-up testing.

In sum, these studies indicate that exposure therapy will be most effective when exposure sessions are unpredictable, variable, include multiple fear-provoking stimuli, and include some aversive events (e.g., social rejection, panic attack). Although fear responding may remain elevated throughout exposure, such procedures may predict better treatment outcomes and reduce the likelihood of relapse.

The dissertation of Najwa Chowdhury Culver is approved.

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2013

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VITA

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INTRODUCTION

There is extensive evidence for the effectiveness of exposure therapy for phobias and anxiety disorders (e.g., Norton & Price, 2007). Exposure therapy involves systematic repeated exposure to anxiety-provoking stimuli. Through such repeated exposure, the individual learns to no longer fear the stimulus. One mechanism believed to account for the acquisition and maintenance of fears and phobias is Pavlovian conditioning and extinction is believed to be at least one mechanism responsible for the effects of exposure therapy (Eelen & Vervliet, 2006). However, following successful exposure therapy, the fear response can return. In fact, numerous animal and human studies have shown instances where presentation of a fear-provoking stimulus (the conditional stimulus or CS) following exposure (i.e., extinction) re-elicits the fear response (the CR): if tested in a different context than the extinction context (renewal; Bouton 1993), if tested after time has passed since extinction (spontaneous recovery; Baum, 1988), upon re-exposure to the US after extinction (reinstatement; Rescorla & Heth, 1975), or if the CS and US occur together again (rapid reacquisition; Kehoe & Macrae, 1997).

This suggests that extinction is *not* unlearning of the previously learned CS-US association. Instead of erasing the CS-US memory, extinction is hypothesized to involve new inhibitory learning (e.g., Bouton, 1993) that then competes with the CS-US memory. Retrievability of the CS-US memory may explain return of fear following exposure therapy. Clearly, this subsequent return of fear (e.g., Rachman, 1989) presents a challenge for both researchers and clinicians. One way to reduce this return of fear is to enhance learning during extinction (i.e., facilitate learning of the CS-no US association). Investigations using animal models to explore methods for enhancing extinction learning

may provide insight into methods for increasing the effectiveness of exposure therapy (Hermans, Craske, Mineka, & Lovibond, 2006). Across a set of three studies, methods which have been effective in enhancing extinction learning in animals were investigated in humans.

Study 1: Compound Stimulus Presentations During Extinction

One way to enhance learning during exposure may be to present two fear-provoking stimuli simultaneously during extinction. The theoretical basis for this derives from the Rescorla-Wagner Model (Rescorla & Wagner, 1972) which makes specific predictions for ways in which learning can be optimized during extinction training. A basic tenet of this model is that learning (during acquisition and extinction) is determined by how surprised the organism is. Surprise is defined by the discrepancy between what is expected to occur and what actually occurs.

The Rescorla-Wagner model predicts the *predictive value* (or associative strength) of the CS. It does so by calculating how this predictive value changes over trials of CS presentation during conditioning, extinction, or other procedures. The predictive value of the CS is defined as V and the change in predictive value of the CS during a given trial is defined as ΔV. After each trial, the new value of V is equal to the old value of V plus the change in value (ΔV): $V_{new} = V_{old} + \Delta V$. The change in predictive value of the CS (ΔV) on any given trial is calculated using several parameters: $\Delta V = \alpha\beta(\lambda - V)$. In this equation, *alpha* (α) represents the salience of the CS; in other words, how much the CS stands out or how much attention the individual pays to the CS. It can vary between 0 and 1, where 0 indicates that the CS attracts no attention and 1 indicates that it attracts maximum attention. *Beta* (β) represents the salience of the US and can also vary between 0 and 1.

Lastly, *lambda (λ)* represents the asymptote of learning possible given the US being used. Another way to describe λ is as the intensity of the US. Therefore, the $(\lambda - V)$ term describes the surprisingness of the US; V is the predictive value of the CS or what the organisms *expects* will occur while λ is the intensity of the US or what *actually* occurs. The difference between λ and V represents how surprised the individual is by what occurs and, according to the model, the amount of learning on a given trial is directly proportional to this element of surprise.

To illustrate the predictions made by the Rescorla-Wagner model, the parameters (α, β, and λ) need to be assigned values. A moderately salient CS can be represented with an *alpha* of 0.3 ($\alpha = 0.3$). A US that is maximally salient can be represented with a *beta* of 1 ($\beta = 1.0$). *Lambda* can be set to 1 so that the maximum learning possible will be equal to 1. If the CS is a neutral stimulus prior to acquisition training, the value of V will start at 0. Given these values, the model predicts the following for the first trial of acquisition training:

$$\Delta V = (0.3)(1.0)(1 - 0) = 0.3$$

Following this trial, V = 0 + 0.3 = 0.3.

This indicates that the predictive value of the CS begins at 0. However, following Trial 1, the predictive value of the CS is equal to 0.3. This is to say that the individual has learned something about the previously neutral CS; the associative strength of the CS has increased by 30%. On Trial 2, the following occurs:

$$\Delta V = (0.3)(1.0)(1 - 0.3) = 0.21$$

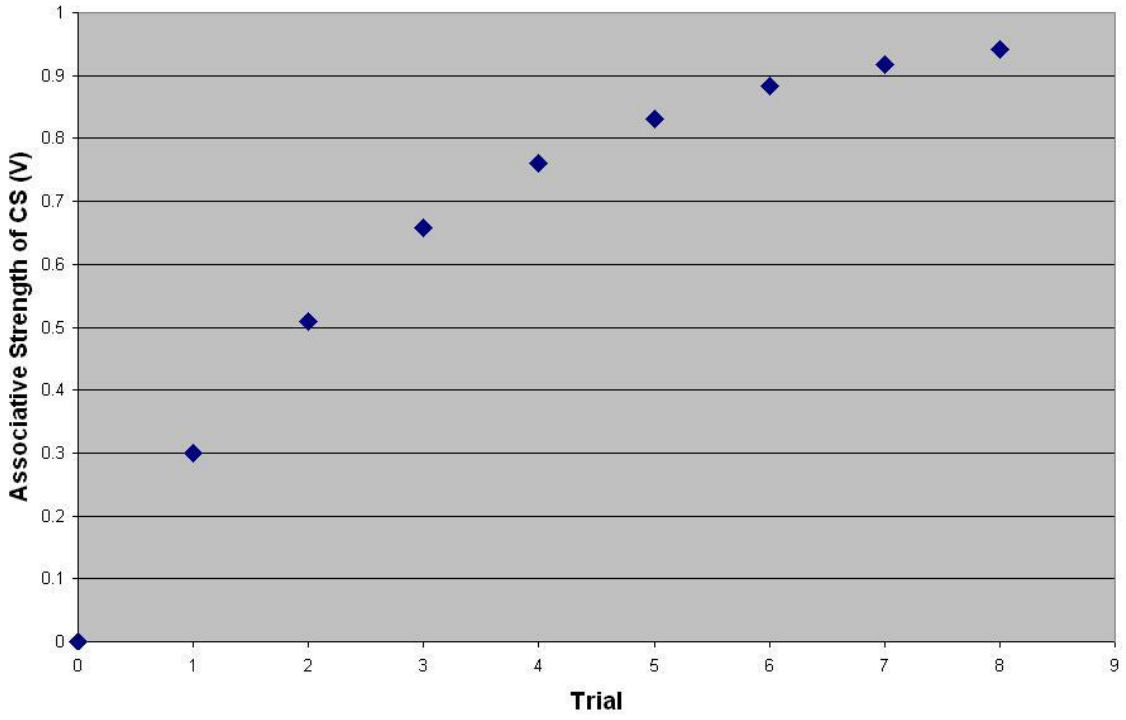
Following this trial, V = 0.3 + 0.21 = 0.51

Thus, after Trial 2, the associative strength of the CS has increased to 0.51. On Trial 3, the model predicts the following:

$$\Delta V = (0.3)(1.0)(1 - 0.51) = 0.147$$

Following this trial, V = 0.51 + 0.147 = 0.657.

It is important to note that the rate at which the CS's associative strength is increasing is declining with each trial. Eventually, there will be very little increase in value across trials as the predictive value of the CS approaches its maximum value (*lambda*). Thus, the Rescorla-Wagner model predicts that the associative strength of the CS during acquisition will rise as a negative exponential function of trials toward an asymptotic value of *lambda*.



To illustrate the Rescorla-Wagner model's predictions for extinction, similar calculations are made. On the first trial of extinction, assuming that the CS was fully

conditioned during acquisition training, the associative strength of the CS (V) will be equal to the maximum learning possible given the US used (λ). In this example, $V = 1$. During extinction training, the individual is presented with the CS but the US does not occur; thus US intensity or the maximum learning possible given the US (λ) = 0. So, on the first trial of extinction learning, the individual will be surprised because expectation of the US is at 1.0 but what actually occurs is no US (0). Formally, the Rescorla-Wagner equation for Trial 1 of extinction training given these parameter values is as follows:

$$\Delta V = (0.3)(1.0)(0 - 1) = -0.3$$

Following this trial, V = 1 + (-0.3) = 0.7

Thus, on Trial 1 of extinction training, the predictive value of the CS decreases to 0.7. On Trial 2, the following occurs:

$$\Delta V = (0.3)(1.0)(0 - 0.7) = -0.21$$

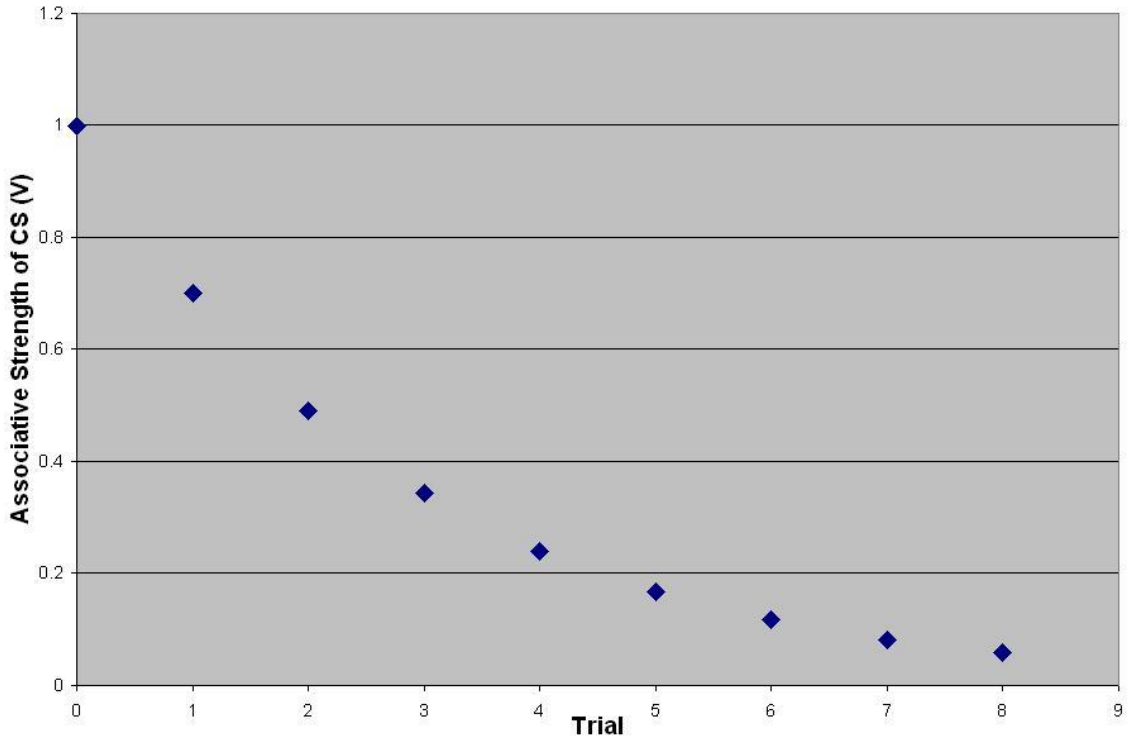
Following this trial, V = 0.7 + (-0.21) = 0.49

Following Trial 2, the predictive value of the CS has decreased to 0.49. On Trial 3, the following occurs:

$$\Delta V = (0.3)(1.0)(0 - 0.49) = -0.147$$

Following this trial, V = 0.49 + (-0.147) = 0.343

Thus, following Trial 3, the predictive value of the CS has decreased to 0.343. The same phenomenon occurs here as during acquisition: the rate at which the predictive value of the CS is decreasing *declines* with each trial. Thus, the Rescorla-Wagner model predicts that the associative strength of the CS during extinction will decrease as a negative exponential function of trials toward an asymptotic value of *lambda*.



However, there is a problem with this view of extinction. According to the model, the predictive value of the CS will approach 0; thus, extinction is modeled as an erasure of the CS-US association. This cannot explain phenomena such as spontaneous recovery, reinstatement, renewal, and rapid reacquisition. Considering the role of the context may help illuminate the occurrence of such phenomena. According to Rescorla and Wagner (1972), the context and the CS compete for association with the US. This occurs because all stimuli present on a given trial, *including contextual stimuli*, determine the organism's expectancy of the US. Therefore, during the first CS-US pairing, both the CS and the context become associated with the US. However, the contextual cues remain present during the intertrial intervals and undergo extinction during these intervals since the US is not present. Thus, over multiple CS-US pairings, the organism learns that the CS is a more accurate predictor of the US than the context is. There is evidence for this

competition among stimuli (e.g., Fanselow, 1980). In this set of experiments, rats were given a choice between a context where they had received shocks as well as signals that indicated shock-free periods and another context in which they had received shocks only. Results indicated that rats avoided the context in which they had received shocks and signaled shock-free periods. One interpretation of these findings is that the signal became a conditioned inhibitor and therefore attenuated extinction of contextual fear during the intershock intervals. So, the contextual cues of the context in which there were signaled shock-free periods became more strongly associated with the shock (the US). Further evidence for this competition among stimuli can be found in experiments demonstrating that acquisition of contextual fear (US presentations in a given context) blocks subsequent acquisition of CS-US associations in that context (e.g., Baker, Mercier, Gabel, & Baker, 1981).

According to the Rescorla-Wagner Model, these same processes also operate during extinction training. In other words, both the CS as well as the extinction context are competing for association with the absence of the US. If this is the case, there are actually ***two*** equations to calculate during Trial 1 of extinction training:

$$\Delta V_{CS} = (0.3)(1.0)(0 - 1) = -0.3$$

and

$$\Delta V_{Context} = (0.3)(1.0)(0 - 1) = -0.3$$

V is equal to 1 in both equations because the presence of the CS causes the individual to expect the US to occur. Following Trial 1, the predictive value of the CS (V_{CS}) = 1 + (-0.3) = 0.7 and the predictive value of the context ($V_{Context}$) = 0 + (-0.3) = -0.3. The context does not have predictive value before this trial but it does have

inhibitory predictive value following the trial; thus, the context is becoming a conditioned inhibitor as is suggested by Rescorla and Cunningham (1978). On the following trial, expectation (V) will be equal to the summation of the predictive value of the CS (V_{CS}) and the predictive value of the context ($V_{Context}$); that is, $V = V_{CS} + V_{Context} = 0.7 + (-0.3) = 0.4$:

$$\Delta V_{CS} = (0.3)(1.0)(0 - 0.4) = -0.12$$

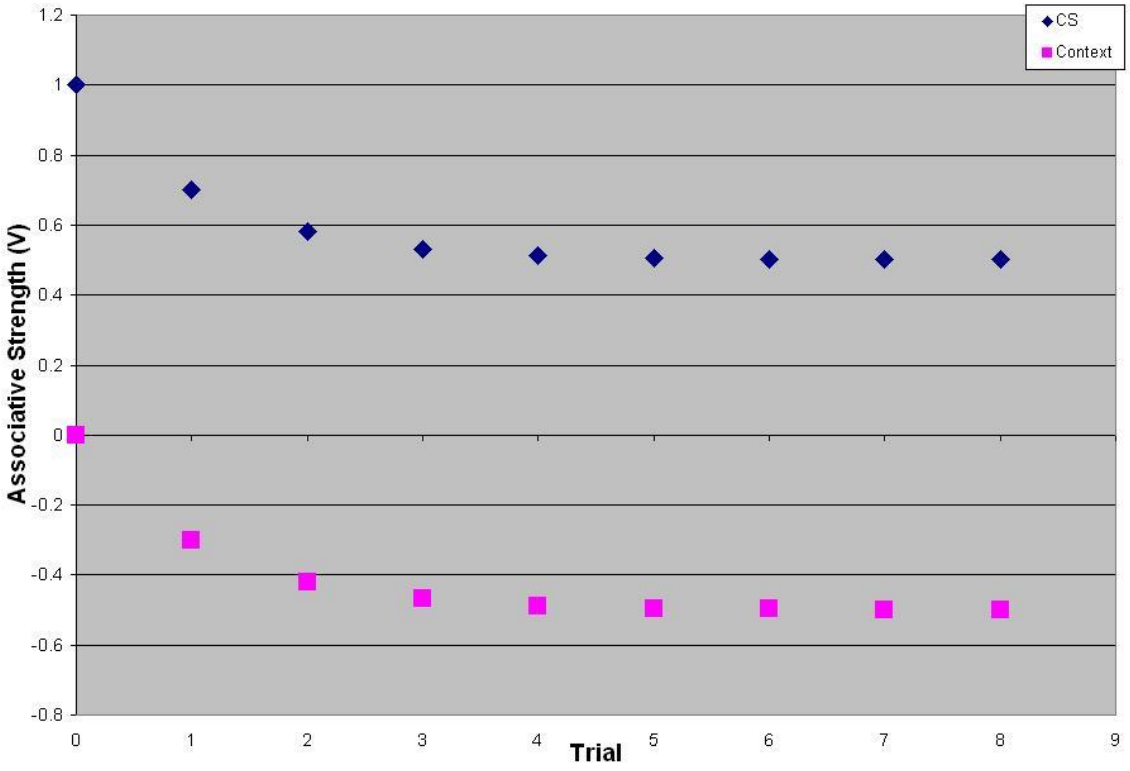
$$\Delta V_{Context} = (0.3)(1.0)(0 - 0.4) = -0.12$$

Thus, following Trial 2 of extinction, $V_{CS} = 0.7 + (-0.12) = 0.58$ and $V_{Context} = -0.3 + (-0.12) = -0.42$. At the third trial, expectation of the US will be equal to the summation of the predictive value of the CS (0.58) and the predictive value of the context (-0.42); in other words, 0.16:

$$\Delta V_{CS} = (0.3)(1.0)(0 - 0.16) = -0.048$$

$$\Delta V_{Context} = (0.3)(1.0)(0 - 0.16) = -0.048$$

So, following Trial 3 of extinction, $V_{CS} = 0.58 + (-0.048) = 0.532$ and $V_{Context} = -0.42 + (-0.048) = -0.468$. As can be seen in Figure 3, the prediction for extinction given that the context is acquiring inhibitory properties is that V_{CS} can never approach 0.



It is important to reiterate that the individual's expectancy of the US during presentation of the CS is always calculated by summating the predictive value of the CS and the predictive value of the context (i.e., $V_{CS} + V_{Context}$). Therefore, after 8 trials of extinction, whenever the CS is presented in this extinction context, prediction of the US is 0.50032768 + (-0.4996723) = 0.00065536 ≈ 0. In other words, because the individual no longer expects the US, conditional responding will be extinguished. However, the predictive value of the CS has not decreased to 0. Given the parameter values in this particular example, the predictive value or associative strength of the CS remains at approximately 0.5. This is because learning has reached its asymptote due to the fact that the individual is no longer surprised by the outcome. As demonstrated above, after 8 trials, US prediction is approximately 0; therefore, $V = \lambda = 0$. So, no further learning regarding the CS or the context will occur according to the model:

$$\Delta V_{CS} = (0.3)(1.0)(0 - 0) = 0$$

$$\Delta V_{Context} = (0.3)(1.0)(0 - 0) = 0$$

Thus, when the individual is presented with the CS in a context different than the extinction context, US-expectancy will be approximately 0.5 (given the parameter values in this example) and the individual will demonstrate recovery of the fear response. It is important to note that contexts are not limited to physical settings. For example, there is evidence from animals and humans that a change in internal context (i.e., drug state) from extinction to test can enhance return of fear (Bouton, Kenney, & Rosengard, 1990; Mystkowski, Mineka, Vernon, & Zinbarg, 2003). Because predictive value of the CS does not asymptote to 0 given the prediction that the extinction context is also acquiring inhibitory properties, phenomena such as renewal, spontaneous recovery, reinstatement, and rapid reacquisition can be accounted for.

Although this assumption that the extinction context becomes a conditioned inhibitor of the US allows the Rescorla-Wagner model to explain recovery of fear following extinction, data has not always supported this view of extinction (e.g., Bouton & King, 1983). Summation and retardation tests can be used to assess the inhibitory properties of a context; for example if an extinction context has truly acquired inhibitory properties, fear responding to any fearful stimulus, including a CS that has not undergone extinction training, should be attenuated. Studies that have conducted such tests sometimes find support for the context as a conditioned inhibitor (Polack, Laborda, & Miller, 2012) while others do not (Bouton & King, 1983). A more likely scenario is that the extinction context functions as a negative occasion setter and is able to modulate the

CS-US relationship without actually forming direct inhibitory context-US associations (Holland & Lamarre, 1984; Holland, 1989; Bouton & Swartzentruber, 1986).

Whether the extinction context functions as a conditioned inhibitor or a negative occasion setter, the problem of recovery of fear to the CS remains and methods for enhancing extinction learning are needed. The Rescorla-Wagner model can be used to predict ways in which extinction training can be enhanced. Some of the parameters are fixed and cannot be modified, such as the salience of the CS and the salience of the US. One parameter that may be available for modification is the individual's *expectancy* (i.e., *V*). Increasing expectancy during extinction will enhance the discrepancy between expectation and reality, thereby maximizing learning. One potential method for increasing US-expectancy is to present two CSs simultaneously (i.e., compound extinction). According to the Rescorla-Wagner model, when two CSs are presented simultaneously, expectation of the US (*V*) will be equal to the predictive value of one CS (V_{CSA}) plus the predictive value of the second CS (V_{CSB}). Given the parameter values used thus far, if two fully conditioned CSs are presented together during extinction, US-expectancy = $V = V_{CSA} + V_{CSB} = 1 + 1 = 2$. During Trial 1 of extinction, the following is predicted to occur:

$$\Delta V_{CSA} = (0.3)(1.0)(0 - 2) = -0.6$$

$$\Delta V_{CSB} = (0.3)(1.0)(0 - 2) = -0.6$$

$$\Delta V_{Context} = (0.3)(1.0)(0 - 2) = -0.6$$

Thus, following Trial 1, $V_{CSA} = 1.0 + (-0.6) = 0.4$, $V_{CSB} = 1.0 + (-0.6) = 0.4$, and $V_{Context} = 0 + (-0.6) = -0.6$. On the following trial, expectation (*V*) will be equal to the summation of the predictive value of CSA (V_{CSA}), the predictive value of CSB (V_{CSB}), and

the predictive value of the context ($V_{Context}$); that is, $V = V_{CSA} + V_{CSB} + V_{Context} = 0.4 + 0.4 + (-0.6) = 0.2$:

$$\Delta V_{CSA} = (0.3)(1.0)(0 - 0.2) = -0.06$$

$$\Delta V_{CSB} = (0.3)(1.0)(0 - 0.2) = -0.06$$

$$\Delta V_{Context} = (0.3)(1.0)(0 - 0.2) = -0.06$$

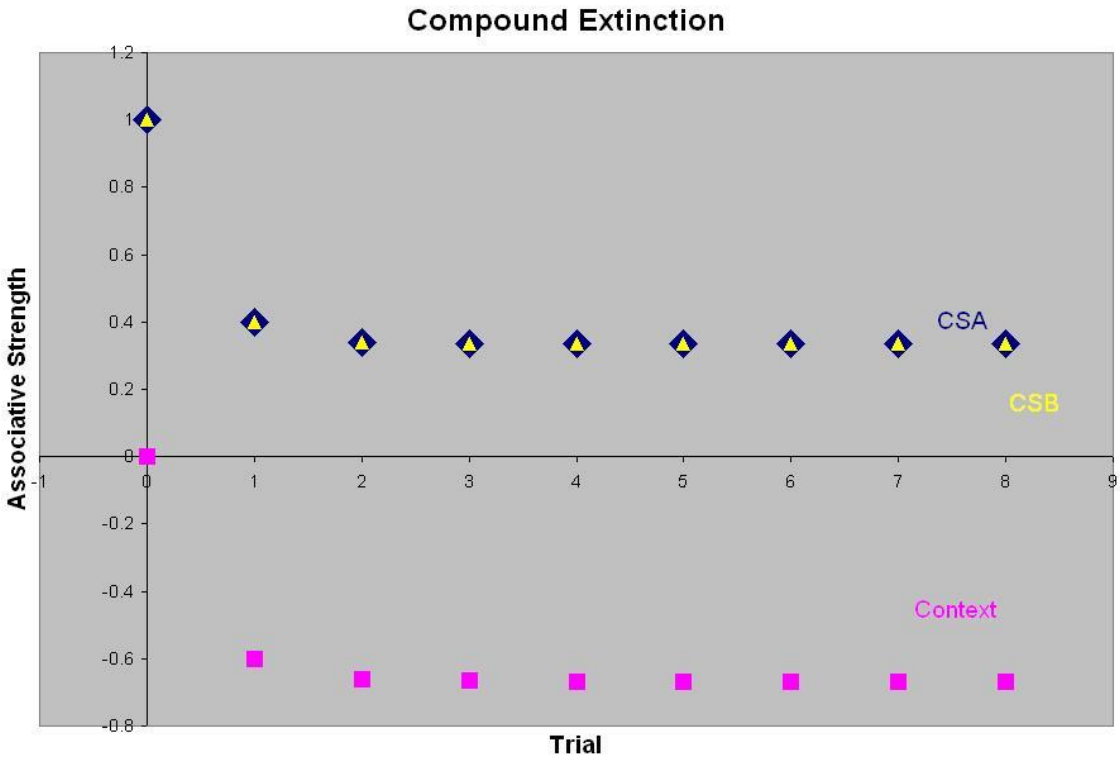
Thus, following Trial 2 of extinction, $V_{CSA} = 0.4 + (-0.06) = 0.34$, $V_{CSB} = 0.4 + (-0.06) = 0.34$, and $V_{Context} = -0.6 + (-0.06) = -0.66$. On the following trial, expectation (V) $= V_{CSA} + V_{CSB} + V_{Context} = 0.34 + 0.34 + (-0.66) = 0.02$:

$$\Delta V_{CSA} = (0.3)(1.0)(0 - 0.02) = -0.006$$

$$\Delta V_{CSB} = (0.3)(1.0)(0 - 0.02) = -0.006$$

$$\Delta V_{Context} = (0.3)(1.0)(0 - 0.02) = -0.006$$

So, following Trial 3 of extinction, $V_{CSA} = 0.34 + (-0.006) = 0.334$, $V_{CSB} = 0.34 + (-0.006) = 0.334$, and $V_{Context} = -0.66 + (-0.006) = -0.666$. As shown in Figure 4, the prediction for extinction given compound CS presentations is that learning will be enhanced (i.e., the predictive value of the CS approaches a lower asymptote than when the CS is presented on its own during extinction). Given the parameter values discussed here, the predictive value of the CS will approach 0.3 given compound CS presentations (whereas the asymptote given single CS presentations was 0.5). Thus, the Rescorla-Wagner model predicts that extinction learning will be enhanced if two CSs are presented in compound.



However, data has not supported this prediction of the model with animals (Pineno, Zilski, & Schachtman, 2007) or humans (Vervliet, Vansteenwegen, Hermans, & Eelen, 2007). These results are difficult to explain with elemental models of associative learning (e.g., the Rescorla-Wagner model) but it may be possible to explain such results with configural models of associative learning (e.g., Pearce, 1987); the latter predict that a compound CS presentation during extinction will be perceived as one consolidated stimulus and, therefore, any new learning that occurs during extinction will be in regards to this compound stimulus rather than to each of the individual stimuli which comprise the compound. (See Introduction of Study 1 for more details). One possible solution to this problem may be to present each CS on its own several times during extinction training before pairing two CSs together in order to facilitate elemental processing of each individual CS comprising the compound.

The Rescorla-Wagner model predicts enhanced extinction learning given this paradigm as well. If each CS is presented several times on its own, the predictive value of each will have reached asymptote. Given the parameters described previously, on the eighth trial of extinction, the following is predicted to occur:

$$\Delta V_{CS} = (0.3)(1.0)(0 - 0.0016) = -0.00049$$

$$\Delta V_{Context} = (0.3)(1.0)(0 - 0.0016) = -0.00049$$

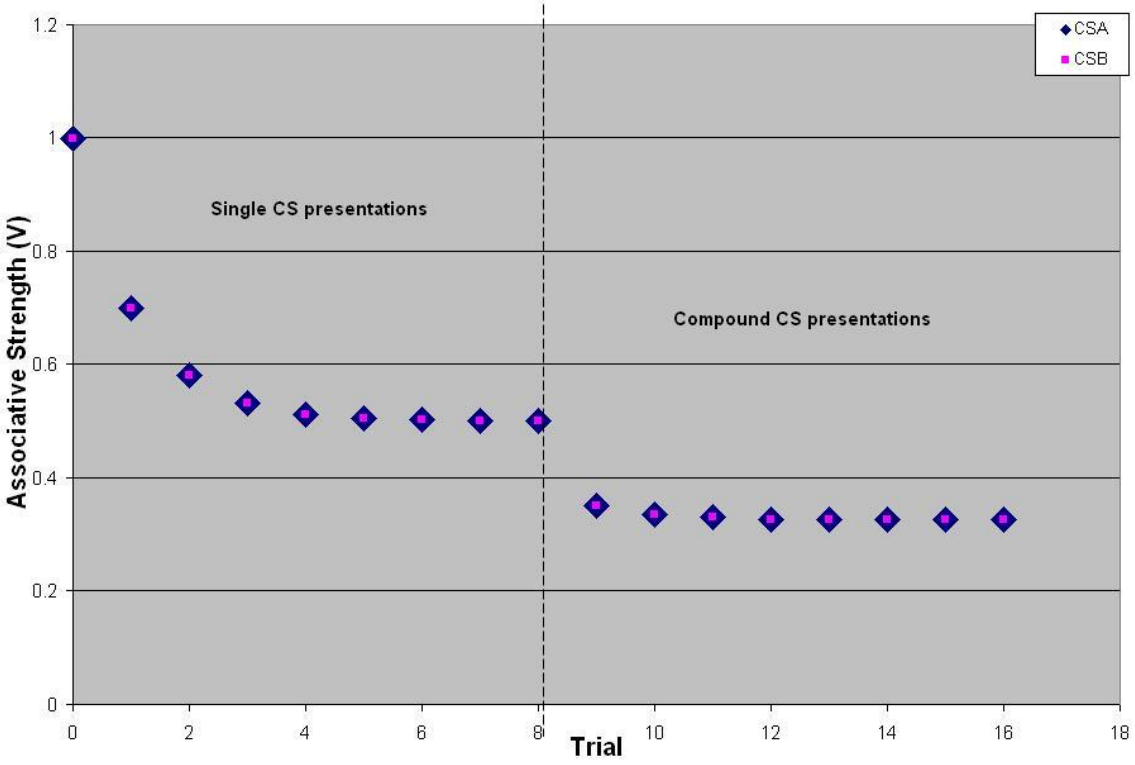
Following this trial, $V_{CS} = 0.5008 + (-0.00049) = 0.5003$ and $V_{Context} = -0.4992 + (-0.00049) = -0.49967$. If two CSs undergo extinction training until this point and are then presented in compound, the organism's expectation of the US will be equal to the predictive value of each CS and the context: $V = V_{CSA} + V_{CSB} + V_{Context} = 0.5003 + 0.5003 + (-0.49967) = 0.50093$. So, US-expectancy will increase during compound CS presentations and this increase in US-expectancy is predicted to drive learning. That is, because US-expectancy has increased, the discrepancy between what the organism *expects* and what actually *occurs* (no US) is greater. Thus, the error term has increased and the individual is more surprised by what happens; this increase in surprise drives learning. Even though predictive value of each CS had reached asymptote during presentations of each CS on its own, presenting the CSs in compound will cause predictive value of each CS to decrease further (i.e., extinction learning will be enhanced).

$$\Delta V_{CSA} = (0.3)(1.0)(0 - 0.50093) \approx -0.15$$

$$\Delta V_{CSB} = (0.3)(1.0)(0 - 0.\ 50093) \approx -0.15$$

$$\Delta V_{Context} = (0.3)(1.0)(0 - 0.\ 50093) \approx -0.15$$

Following eight such compound trials, the predictive value of each CS will have decreased from approximately 0.5 to approximately 0.3 (i.e., extinction learning is deepened; Figure 9).



Evidence *does* support this prediction of the model, described in detail in the Introduction section of Study 1 (Leung & Westbrook, 2008; Pineno et al, 2007; Rescorla, 2006). Thus, the aims of Study 1 were to investigate whether single-CS extinction trials followed by compound CS trials optimizes extinction learning in humans and, if so, the mechanism by which this enhanced learning occurs (enhanced associative change or enhanced responding).

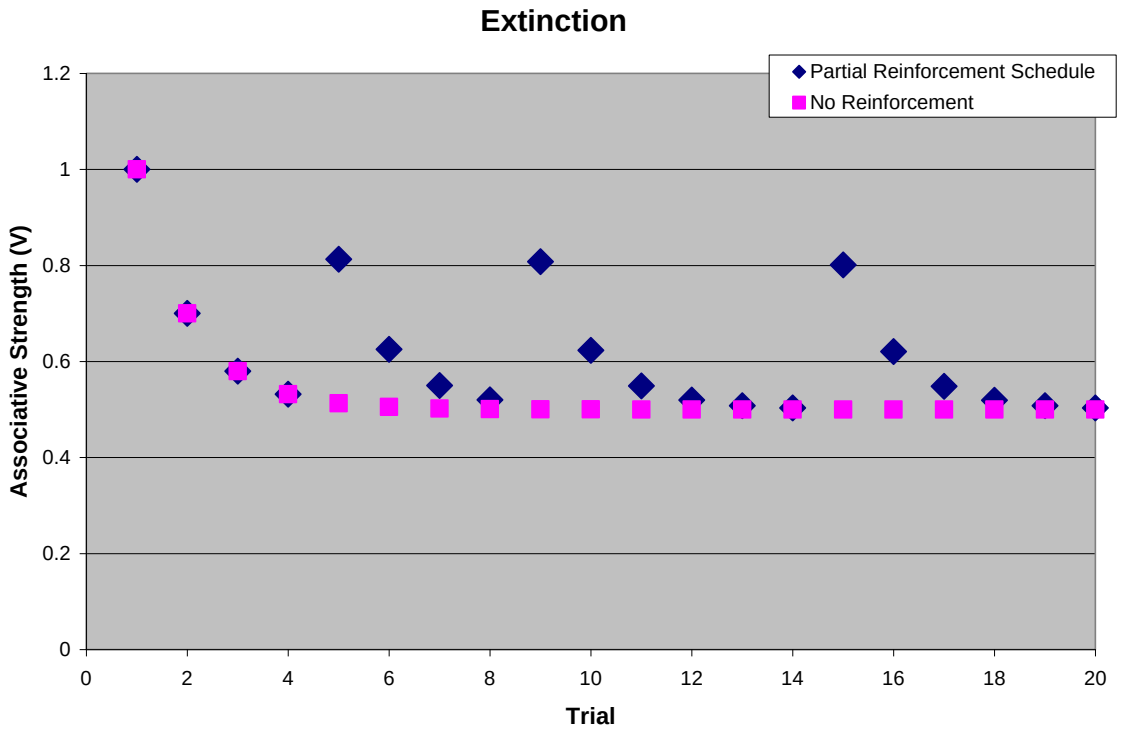
Study 2: Occasional Reinforced Trials during Extinction

A second way to enhance learning during exposure may be to present occasional reinforced trials (i.e., CS-US pairings) during extinction. This derives from a mechanism

called “trial signaling” (Ricker & Bouton, 1996). Trial signaling refers to the idea that, during acquisition training, the organism learns not only that the CS predicts the US but also that reinforced trials predict subsequent reinforced trials. Similarly, during extinction, the organism may learn not only that the CS no longer predicts the US but also that nonreinforced trials predict subsequent nonreinforced trials. In other words, during acquisition, the CS-US association is learned and a secondary association may be learned as well: [CS-US]-[CS-US]. During extinction, the CS-no US association is learned and a secondary association may be learned as well [CS-no US]-[CS-no US]. Thus, following extinction, if the CS and US occur together again, the animal or human may demonstrate rapid reacquisition of the original excitatory learning because of the prediction that CS-US pairings will follow one another. However, if some CS-US pairings are incorporated into the extinction context, there would be two secondary associations learned during extinction: [CS-no US]-[CS-no US] *and* [CS-US]-[CS-no US]. Now, when faced with a CS-US pairing following successful extinction training, the animal or human will not necessarily predict subsequent CS-US pairings. CS-US pairings have been paired with both subsequent CS-US pairings (during acquisition) and subsequent CS-no US pairings (during extinction). Such predictions are in line with Capaldi’s theory which emphasizes the *sequence* of events (Capaldi, 1967, 1994) and the idea that previous trials can control responding on subsequent trials. According to Capaldi, the memory trace of a trial (reinforced or nonreinforced) can become associated with what happens after that trial (e.g., a reinforced or nonreinforced trial). Thus, trial-signaling predicts that occasional reinforced trials during extinction may slow the rate of reacquisition following extinction training.

This prediction is somewhat at odds with what the Rescorla-Wagner model predicts will happen in the case of occasional reinforced trials during extinction. It seems that the Rescorla-Wagner model would predict enhanced extinction learning given occasional reinforced trials since expectation would continually fluctuate throughout extinction allowing the surprise factor to remain elevated instead of diminishing across the first few trials and remaining low. If surprise remains elevated, it would seem that learning would be enhanced. However, using the formal equation, both *lambda* (the maximum learning possible given a specific US) and *V* (the *expectancy* term) will fluctuate throughout extinction training given occasional reinforced trials. *Lambda* will either equal 0 (on a nonreinforced trial) or 1 (on a reinforced trial); *V* will increase following a reinforced trial and decrease following a nonreinforced trial. Although expectation will fluctuate throughout extinction which should facilitate inhibitory learning by maximizing discrepancy between expectation and reality, the predictive value of the CS also increases each time a reinforced trial occurs. Therefore, the model predicts that the overall effect of occasional reinforced trials will be to either lead to similar levels of decrement in the overall predictive value of the CS or actually lead to less of an overall decrement in the predictive value of the CS (depending on how many extinction trials are utilized and the percentage of those which are reinforced). For example, using the same parameter values for α and β as in the previous example, if twenty extinction trials are utilized and three random trials are reinforced the Rescorla-Wagner model predicts similar learning decrements with occasional reinforced trials as

with no reinforced trials:



Of course, although the Rescorla-Wagner model has been one of the most (if not *the* most) influential theory of associative learning and is incredibly successful in predicting many aspects of associative learning, it does have its limitations as well (see Miller, Barnet, & Grahame, 1995 for a review). One limitation which may be relevant to the paradigm of partial reinforcement during extinction is that the value of α for a given CS is not allowed to fluctuate. In other words, the model cannot account for changes in the attention paid to a CS. In the graph above, it seems conceivable that twenty nonreinforced presentations of the CS may lead to a decline in α. Why should the animal or human continue paying attention to the CS if it no longer predicts the US? Thus, the model may be overestimating the decrement in learning that would occur with no reinforced trials (i.e., because, if α diminished, learning at each trial would diminish).

Similarly, α may *increase* in the case of occasional reinforced trials. If CS presentations sometimes predict the US and sometimes do not, the animal or human may begin to pay more and more attention to the CS to try to distinguish a feature of the CS which predicts whether or not the US will occur. If α increases in the partial reinforcement group, rate of learning will increase and the partial reinforcement group may actually show more of a decrement than the control group.

A model of learning which does account for fluctuations in the salience of CS has been proposed by Pearce and Hall (1980). In this model, attention to a stimulus is necessary while subjects are learning about its significance; however, once learning has reached a stable asymptote no further attention to the stimulus is required. Obviously, the organisms needs to detect the stimulus in order to respond appropriately in its presence but Pearce and Hall (1980) regard this as a different form of attention than the one necessary for learning. They therefore proposed that attention to a CS, and hence its associability, will be governed by the following equation:

$$\alpha_{An} = (\lambda - V)_{n-1}$$

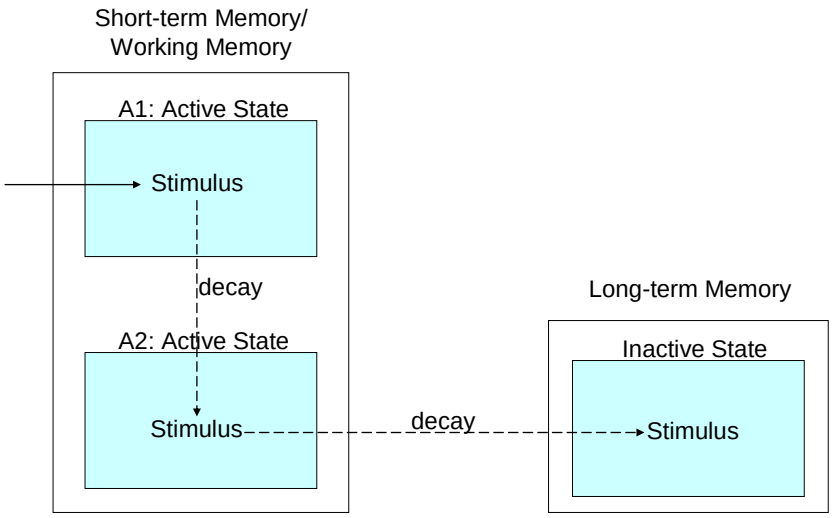
According to this equation, the associability of Stimulus A on trial n, is determined by the absolute value of the discrepancy $(\lambda - V_T)$ for the previous occasion on which A was presented (where V_T is determined in the same way as for the Rescorla-Wagner model). In other words, the associability of a stimulus will be high when followed by a US that is unexpected (when $\lambda - V_T$ is high), but its associability will be low when followed by a US that is expected (when $\lambda - V_T$ is low). In the case of occasional reinforced trials during extinction, the associability of the CS will decrease across multiple nonreinforced trials and then increase after a reinforced trial. Thus,

following a reinforced trial (during which the US is presented unexpectedly), the salience of the CS will increase thereby leading to greater inhibitory conditioning of the CS. Thus, the Pearce and Hall (1980) model provides a possible theoretical framework for the effectiveness of occasional reinforced trials enhancing learning during extinction.

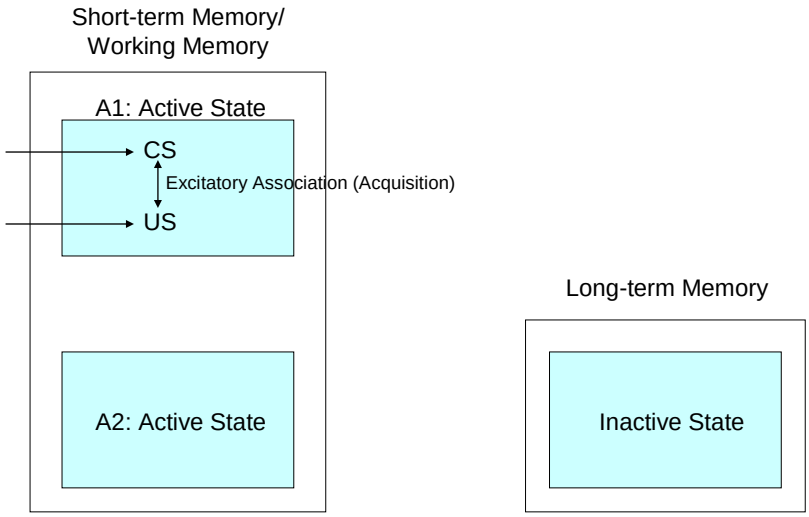
The idea of occasional reinforced trials during extinction also seems related to the concept of the CS-preexposure effect, a phenomenon that the Rescorla-Wagner model cannot explain. This occurs when the CS is presented several times without the US prior to acquisition training which can lead to retardation of acquisition learning when the CS is subsequently paired with the US (Lubow & Moore, 1959). Since no US is present during pretraining exposure to the CS, the Rescorla-Wagner model predicts that nothing should be learned about the CS at this time. Thus, a preexposed CS and a nonpreexposed CS should have the same associative status at the start of acquisition training and therefore demonstrate the same rate of training during acquisition. The most common explanation of the CS preexposure effect is a loss of attention to the CS as a result o the nonreinforced pretraining exposures to the CS (e.g., Lubow, Weiner, & Schnur, 1981). Although Rescorla and Wagner (1972) admitted that the CS-preexposure effect is best explained in terms of decreasing alpha, they chose not to incorporate changing alpha values in the model.

A model of learning theory which can explain the CS preexposure effect is Wagner's Standard Operating Procedures or Sometimes Opponent Process (SOP) model (Wagner, 1981). In SOP, memory is conceptualized as being comprised of representative nodes that are connected with directional links. These memory nodes are assumed to correspond with specific stimuli and each node consists of a large but finite set of

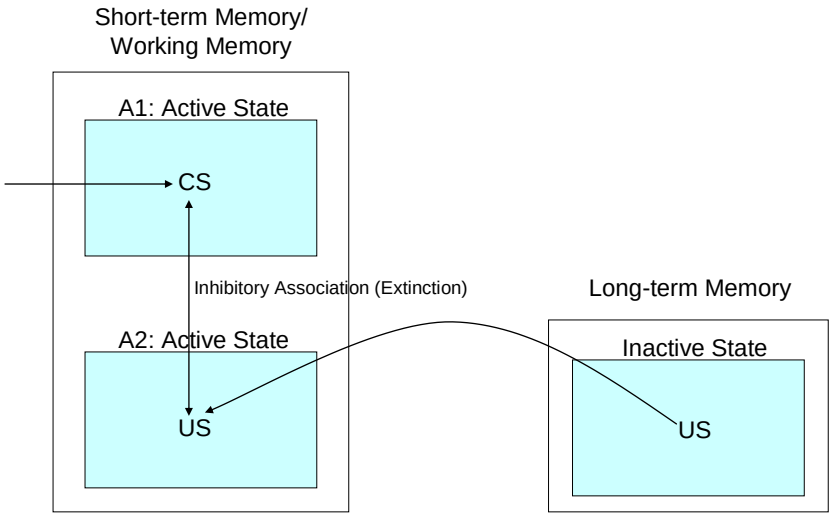
informational elements. The informational elements within a given node are thought to be in one of three states of activation: a primary "A1" state which represents the focal portion of working memory and is elicited when the corresponding stimulus is presented, a secondary "A2" state which represents the peripheral portion of working memory into which elements passively decay from A1, and an inactive "I" state which represents long-term memory and into which elements passively decay from A2.



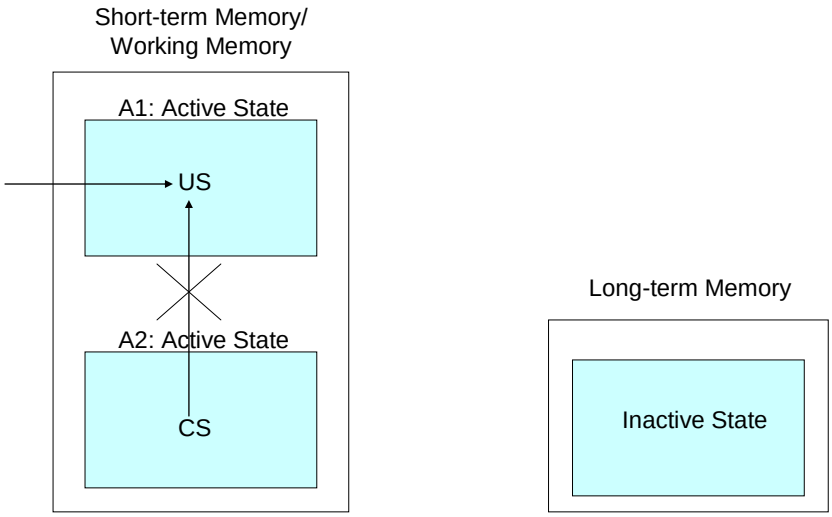
Wagner describes these states as being interconnected by specific directional associative links. For example, when an organism is presented with a CS-US pairing, both of these stimuli are activated into the A1 state simultaneously and this causes an excitatory association to form between the informational elements of the CS and the informational elements of the US.



During extinction procedures, presentation of the CS alone causes informational elements of the CS to be activated into A1. In addition, because the CS has been previously associated with the US, presentations of the CS cause informational elements of the US to be activated from the inactive state into the A2 state. When elements of the CS are activated in A1 and elements of the US are activated in A2, this allows an inhibitory connection to form between the two stimuli.



A crucial tenet of this model is that a node cannot be activated from the A2 state to the Al state. Thus, if a nonreinforced presentation of a CS occurs, the CS will be activated into A1 and then decay into A2. Subsequently, if a CS-US pairing occurs while elements of the CS are still in A2, the CS representation cannot be activated into A1 and therefore learning of the CS-US relationship will be attenuated.



Although the CS preexposure effect is usually applied to acquisition training, this can also be applied to extinction training in the case of a partial reinforcement schedule. If a few nonreinforced presentations of the CS are followed by a CS-US pairing during extinction, elements of the CS may still be activated into the A2 state. Therefore, during the CS-US pairing, the excitatory relationship between the CS and US is not enhanced. Even though excitatory associations between the CS and US may not be strengthened, cognitive processes may allow increased expectation of the US at the following trial. Thus, using Wagner's SOP model, it seems possible that occasional reinforced trials during extinction may lead to fluctuations in expectation of the US without actually strengthening the excitatory CS-US relationship. So, even though using the Rescorla-Wagner model equation does not necessarily predict enhanced learning with occasional reinforced trials during extinction, it is possible that the model is not accounting for two factors. One is that α may actually increase during such a procedure and thereby

maximize inhibitory learning. The second is that the CS-US pairings during extinction, since they occur on a partial reinforcement schedule, will not lead to as much excitatory learning as the Rescorla-Wagner equation predicts. Just as the Rescorla-Wagner model is not able to predict the CS preexposure effect, it may not be able to predict that excitatory CS-US learning may be attenuated during a partial reinforcement schedule. Given the predictions of Wagner's SOP model and the possibility that α values may fluctuate, it is possible that occasional reinforced trials during extinction may enhance learning.

Bouton and colleagues (2004) tested this procedure in rats and found that, using an appetitive paradigm, partial reinforcement during extinction slowed the rate of rapid reacquisition at subsequent testing (see Introduction section of Study 2 for details). Thus, the aims of the second study were to assess whether or not occasional reinforced trials during extinction lead to less recovery of fear as measured by tests of spontaneous recovery and rapid reacquisition.

Study 3: Sustained Arousal during Extinction

A third potential method for enhancing learning during extinction training may be to incorporate stimuli which elevate fear responding during CS presentations. There is some evidence that increased responding alone may optimize learning during extinction (Cain, Blouin, & Barad, 2004). In this study, when rats were administered yohimbine (which blocks autoinhibitory α_2 adrenergic receptors and is anxiogenic in humans) prior to extinction, learning was facilitated. The authors posited several possible mechanisms for these results including that increased adrenergic activity promotes consolidation of emotional memories (Cahill, Prins, Weber, & McGaugh, 1994; Cahill et al., 2000; Nielson & Jensen, 1994) so it is possible that administration of yohimbine facilitated

consolidation of extinction memories. Further evidence for the role of elevated fear responding during extinction comes from the finding that pharmacological procedures which inhibit arousal, if administered during extinction, can impede extinction learning in rats (Berman & Dudai, 2001). Thus, it is possible that elevated fear responding alone facilitates extinction learning and the mechanism by which this occurs is that increased adrenergic activity promotes consolidation of memories encoded during extinction (i.e., the CS-no US relationship).

Another possibility is that arousal mediates the rate of learning. Podlesnik and Sanabria (2011) have proposed a modified version of the Rescorla-Wagner equation which accounts for arousal elicited by US presentation. They did so by replacing the β term in the Rescorla-Wagner model with *A* (arousal) and allowed it to vary with US density. Thus, when the US is presented frequently (by itself or along with other stimuli), CSs are more rapidly learned, extinguished, and discriminated. As time since the last US elapses, learning progressively slows down. This arousal-mediated model of learning can account for phenomenon that the original Rescorla-Wagner model cannot; however, it has its own limitations (reviewed in Podlesnik & Sanabria, 2011). Nevertheless, it provides another possible account of how sustained fear responding alone may facilitate learning during extinction.

Rescorla (2000) demonstrated that extinction of a conditional stimulus (CS) in the presence of an additional excitatory stimulus was superior to extinction alone and also superior to extinction in the presence of an additional neutral stimulus. Although fear responding during extinction was greater when the CS underwent extinction training in the presence of an additional excitatory stimulus, these rats demonstrated significantly

less fear responding to that CS at follow-up testing. Just as with compound extinction procedures, two potential mechanisms may explain these findings: 1) enhanced associative change (i.e., US-expectation was elevated when the CS was presented with an additional excitatory stimulus thereby maximizing surprise and enhancing learning) or 2) enhanced responding. Given the findings of Cain and colleagues as well as the arousal-mediated model of learning proposed by Podlesnik and Sanabria, it seems possible that enhanced responding alone during extinction may enhance learning.

The aims of Study 3 were to test whether incorporating additional excitatory stimuli (which were predicted to elevate responding but not necessarily enhance US-expectation) during exposure exercises enhances learning and protects against spontaneous recovery effects in a sample of participants highly fearful of public speaking.

STUDY 1

COMPOUND STIMULUS PRESENTATIONS DURING EXTINCTION ATTENUATE FEAR RECOVERY AT FOLLOW-UP TESTING

Introduction

There is extensive evidence for the effectiveness of exposure therapy for phobias and anxiety disorders (e.g., Norton & Price, 2007). Exposure therapy involves systematic repeated exposure to anxiety-provoking stimuli. Through such repeated exposure, the individual learns to no longer fear the stimulus. One mechanism believed to account for the acquisition and maintenance of fears and phobias is Pavlovian conditioning and extinction is believed to be at least one mechanism responsible for the effects of exposure therapy (Eelen & Vervliet, 2006). However, following successful exposure therapy, the fear response can return. In fact, numerous animal and human studies have shown instances where presentation of a fear-provoking stimulus (the conditional stimulus or CS) following extinction re-elicits the fear response (the CR): if tested in a different context than the extinction context (renewal; Bouton 1993), if tested after time has passed since extinction (spontaneous recovery; Baum, 1988), upon re-exposure to the US after extinction (reinstatement; Rescorla & Heth, 1975), or if the CS and US occur together again (rapid reacquisition; Kehoe & Macrae, 1997).

This suggests that extinction is *not* unlearning of the previously learned CS-US association. Instead of erasing the CS-US memory, extinction is hypothesized to involve new inhibitory learning ("CS-no US") that then competes with the CS-US memory (e.g., Bouton, 1993). Retrievability of the CS-US memory may explain return of fear

following exposure therapy. Clearly, this subsequent return of fear (e.g., Rachman, 1989) presents a challenge for both researchers and clinicians. One way to reduce the return of fear is to enhance learning during extinction (i.e., facilitate learning of the CS-no US association) and the Rescorla-Wagner Model (Rescorla & Wagner, 1972) makes specific predictions for ways in which this can be accomplished. A basic tenet of the Rescorla-Wagner model is that learning (during acquisition and extinction) is determined by the amount by which the organism is surprised. In fact, according to the model, the amount of learning on a given trial is directly proportional to the element of surprise. Surprise is defined by the discrepancy between what is expected to occur and what actually occurs and is therefore maximized during the first few trials of acquisition or extinction learning. For example, the US is especially surprising at the first trial of acquisition when the CS is neutral (i.e., expectation of an aversive event is 0). Following the first few trials, learning greatly diminishes because expectation becomes closer and closer to reality thereby minimizing surprise.

This phenomenon may partially explain the return of fear sometimes observed following successful extinction training. Perhaps learning is not maximized during extinction training because expectation of the US decreases rapidly during the first few trials of extinction, such that no new learning regarding the CS takes place during subsequent extinction trials. According to the Rescorla-Wagner model, one way to maximize learning during extinction learning is to repeatedly elevate expectancy of the US to provide more opportunities for discrepancy between what is expected and what actually occurs.

Presenting two fear-provoking CSs simultaneously (i.e., compound extinction trials) is one potential method for elevating expectation of the US. However, data have not supported the value of compound extinction in either animals (Pineno, Zilski, & Schachtman, 2007) or humans (Vervliet, Vansteenwegen, Hermans, & Eelen, 2007). Pineno and colleagues utilized a taste aversion paradigm to investigate compound extinction in rats. During acquisition training, rats received a pairing of a vinegar-CS with the US (an intraperitoneal injection of LiCl) on one day and, following a day of recovery, received a pairing of a sucrose-CS with the US. Rats were subsequently randomly assigned to one of four groups for extinction training: 1) presentations of a sucrose + vinegar solution, 2) presentations of sucrose-alone solution, 3) presentations of vinegar-alone solution, and 4) no presentations of any solutions (these rats served as a no-extinction control group). Results indicated that, across extinction training, rats in the group presented with sucrose-alone and the group presented with vinegar-alone increased their consumption of solution while rats who received the compound sucrose + vinegar solution did not. During the test phase, rats who had received the compound solution during extinction performed similarly to the rats who received no extinction training at all; there were no significant differences between these two groups in consumption of vinegar or sucrose. Both of these groups consumed significantly less vinegar-only solution and sucrose-only solution during the test phase than rats in groups who had received either sucrose-only or vinegar-only solutions during extinction. Thus, presentation of the compound sucrose + vinegar solution during extinction appeared to *prevent* extinction learning regarding each individual CS (the sucrose solution and the vinegar solution).

In humans, Vervliet and colleagues (2007) utilized a fear conditioning paradigm to investigate whether compound CS trials during extinction enhanced learning. During acquisition, two geometric figures (CS+'s) were paired with electric shock four times while two other geometric figures (CS-'s) were presented without shock. During extinction, the two CS+ shapes and two CS- shapes were presented in shape equations forming one compound CS+ and one compound CS- shape. At test, one CS+ and one CS- were each presented three times. Results indicated *impaired* extinction to the individual CS+ presented at test, as measured in subjective rated shock-expectancy and skin conductance responses.

The results obtained by Pineno and colleagues (2007) as well as Vervliet and colleagues (2007) are difficult to explain with elemental models of associative learning (e.g., the Rescorla-Wagner model) but it may be possible to explain such results with configural models of associative learning (e.g., Pearce, 1987). Elemental models of learning assume that, when presented with a collection of stimuli, an individual will summate the predictive values of each individual stimulus. That is, elemental theories (such as the Rescorla-Wagner model) propose that the collection of stimuli will have no separate predictive value apart from the summation of its component parts. However, configural models of learning (e.g., Pearce, 1987) assume that, when presented with a collection of stimuli, an individual will process these stimuli in a consolidated manner. Thus, an individual's responding will be determined by information about the whole collection.

In the case of a compound presentation of two CSs, elemental theories make markedly different predictions than configural theories. Elemental theories posit that

each CS will be processed separately and that prediction of the US will be determined by summating the predictive value of each individual CS. Configural theories, on the other hand, posit that the compound CS presentation will be perceived as one consolidated stimulus. Thus, any new learning that occurs during extinction will be in regards to this compound stimulus rather than to each of the individual stimuli which comprise the compound. Therefore, to maximize extinction learning regarding each individual stimulus, it is necessary to facilitate elemental processing of the stimuli comprising the compound (i.e., the two CSs). One method for accomplishing this may be to present each CS on its own several times during extinction training prior to pairing two CSs together.

There is evidence to support the value of this method for enhancing elemental processing. For example, Leung and Westbrook (2008) paired a tone-CS and light-CS to a shock-US during acquisition training in rats. Each CS then underwent separate extinction training. Following this, the two CSs were presented in compound and rats demonstrated recovery of fear to this compound stimulus (i.e., as predicted by the model, expectation of the US increased when two extinguished CSs were presented together). This compound stimulus then underwent extinction training. Because the aims of these investigators were not to directly test compound extinction, there was no control group that only received single extinction trials; thus, it cannot be ascertained from this experiment whether compound extinction enhanced extinction learning compared with single-CS trials during extinction. However, rats did demonstrate evidence of extinction learning at test; therefore, the disruption of extinction learning reported by Pineno and colleagues (2007) as well as Vervliet and colleagues (2007) did not occur in this case.

Some evidence for enhanced extinction learning using such a paradigm is found in the results of a second experiment by Pineno and colleagues (2007). As in the first experiment discussed previously, conditioning consisted of training taste aversions to two flavors. During extinction, one group received compound solutions while another group received some trials of one flavor followed by compound solutions; rats in the latter group demonstrated enhanced extinction learning at test.

Lastly, Rescorla (2006) performed a set of studies aimed at investigating the effectiveness of compound extinction. In pigeons and rats, using both an appetitive paradigm (CS paired with food pellet US) and an aversive paradigm (CS paired with foot shock US), extinction learning was optimized when each CS underwent separate extinction and then the two CSs were presented together. Compared with animals that only received single extinction trials, animals that received compound extinction trials demonstrated decreased spontaneous recovery and weaker reinstatement. Thus, this set of studies provides evidence for enhanced extinction learning when each CS is presented separately at first and then the two CSs are presented in compound. Furthermore, Rescorla investigated whether this enhanced learning during extinction can be attributed to the enhanced associative change predicted by the Rescorla-Wagner model. He posited that there was another possible explanation for enhanced extinction learning during compound CS presentations: increased responding. Because two CSs are presented simultaneously, the organism is predicted to perform the conditional (fear) response at an enhanced level and there is evidence that this enhanced responding alone can facilitate extinction learning (e.g., Cain, Blouin, & Barad, 2004).

The present study aimed to investigate whether single CS extinction trials followed by compound CS trials optimizes extinction learning in humans and, if so, which mechanism (enhanced associative change or enhanced responding) accounts for this increased learning. Following fear conditioning procedures, half of the participants were randomly assigned to receive compound extinction trials following some single extinction trials while the other half were presented with single extinction trials only. It was hypothesized that participants who received presentations of compound extinction trials would demonstrate significantly more fear responding and elevated US-expectancy ratings throughout extinction but reduced fear recovery at the spontaneous recovery and reinstatement tests one week following extinction training (compared with participants who only received single extinction trials). In addition, within each of these extinction groups, half of the participants were randomly assigned to ingest caffeine prior to extinction training. This was done to test the mechanism by which compound extinction trials may facilitate extinction learning. Ingestion of caffeine was expected to elevate fear responding without increasing US-expectancy during extinction training. Based on the findings of Rescorla (2006) and the predictions of the Rescorla-Wagner model, it was hypothesized that increased responding alone would not reduce fear recovery at the spontaneous recovery and reinstatement tests because the mechanism by which compound extinction was hypothesized to enhance learning is enhanced associative change, not enhanced responding. Thus, it was predicted that, regardless of whether they ingested caffeine or not prior to extinction, participants presented with compound extinction trials would demonstrate significant less fear responding at the spontaneous

recovery and reinstatement tests than participants presented with only single extinction trials.

Method

Design

Participants were randomly assigned to one of four experimental groups according to a 2 (Extinction Group: single [S] or compound [C] stimulus trials) x 2 (Drug Group: caffeine ingestion [C] or placebo ingestion [P]) design. The distribution of participants across the four groups was as follows: SP = 18, CP = 18, SC = 16, CC = 18.

Participants

Seventy participants (49 females, 21 males), with a mean age of 19.2 (range 18 to 23), were recruited through mass testing sessions from several Introduction to Psychology classes. Participants were from a variety of ethnic backgrounds: 32.9% Asian, 32.9% Caucasian, 18.6% Latino, 8.5% Biracial, 4.3% Middle Eastern, 1.4% Arab, 1.4% Indian. Participants were recruited if they: 1) scored in the top quartile of scores on the Behavioral Inhibition Scale; 2) on average, ingested between 100mg of caffeine per week and 500mg of caffeine per day and 3) were in the average range (18.5-24.9) for Body Mass Index. Exclusion criteria for study participation included 1) any heart, respiratory, or neurological problems, 2) current or a history of seizures, 3) pregnancy, and 4) current ingestion of drugs or medications which can interact with caffeine including Baycip, Ciloxan, Ciflox, Cipro, Ciproxin, Proquin, Xanthine, Zanaflex, Sirdalud, Ephedra, cough or cold medications containing ephedrine or pseudoephedrine, or guarana.

Measures

Self Report Questionnaires

Two self report measures were completed at Baseline to assess group differences: the Behavioral Inhibition Scale (BIS; Carver & White, 1994) and the Beck Depression Inventory (BDI; Beck, Steer, & Brown 1996). The BIS, a 7-item scale, was developed to measure individual differences in sensitivity to one of two general motivational systems which are thought to underlie behavior: a behavioral approach system (BAS) is believed to regulate appetitive motives in which the goal is to move toward something desired while a behavioral avoidance or inhibition system (BIS) is believed to regulate aversive motives in which the goal is to move away from something unpleasant (Carver & White, 1994). The BIS has demonstrated good internal consistency, $\alpha = 0.74$. In a sample of 732 undergraduate students, the mean BIS score was 19.99 with a standard deviation of 3.79.

The Beck Depression Inventory is a widely used screening instrument for depression. It is a 21-item measure where each item is rated on a 4-point Likert-type scale ranging from 0 to 3 with higher scores indicating higher levels of depression. Scores range from 0 to 63 and, according to the BDI manual, scores of 0 to 13 denote minimal depression, scores of 14 to 19 denote mild depression, scores of 20 to 28 denote moderate depression, and scores of 29 to 63 denote severe depression. In a sample of 229 young adults recruited from undergraduate courses, the mean score on the BDI was 9.12 with a standard deviation of 8.32 (Segal, Coolidge, Cahill, & O'Riley, 2008). In this sample, the BDI also demonstrated good internal reliability ($\alpha = 0.92$).

Subjective Measures

Across all phases of the experiment (acquisition, extinction, and test), participants were asked to rate their expectancy of the US during CS presentations. They were instructed to immediately record US-expectancy at the onset of each CS presentation. This expectancy was rated on a scale between -6 = "certain no noise" to +6 = "certain noise" with a midpoint of 0 = uncertain.

At four time points, participants reported valence ratings for each CS. They were asked to rate each CS on a scale of -50 = "very unpleasant" to +50 = "very pleasant" with a midpoint of 0 = "neutral." These time points were: immediately following acquisition, immediately following extinction, immediately upon arriving for the one-week follow-up test on Day 2, and immediately following the test phase on Day 2.

Physiological Measures

Heart rate (HR) and skin conductance responses (SCRs) were measured using a Biopac MP150 unit running Acqknowledge 4.0 software (Biopac Systems, Inc., Goleta, CA) with one GSR 100C amplifier and one electrocardiogram (ECG) 100C amplifier attached. The GSR amplifier was set to direct current and had a sensitivity of 5 μohm/V, with a 1.0-Hz low-pass filter. Skin conductance responses (SCRs) were measured at the onset of each CS. The ECG amplifier gain was set at 1,000, the R-wave detector was switched on, and the filter was switched off. Data were acquired at 200 samples per second, providing a temporal resolution of 5 ms.

To measure SCRs, two disposable 1 cm diameter AG-AgCl electrodes were placed on the distal phalanx of the index and middle fingers of the non-dominant hand. The magnitude of SCRs was calculated as the difference between the maximum skin conductance level (measured in microsiemens) within 1–6 seconds following CS onset

and the mean skin conductance level within the 2-second period prior to CS onset. Amplitudes were range corrected using the largest response elicited by the US for each individual participant. To do this, for each participant, SCRs to each US presentation were calculated as the difference between the maximum skin conductance level within 1-6 seconds following US-onset and the mean skin conductance level within the 2-second period prior to CS-onset (the CS which occurred immediately prior to that particular US presentation). For each participant, all SCRs to CSs were divided by that person's maximum SCR to the US. These range-corrected responses were then subjected to a square root transformation in order to normalize the distribution prior to statistical analysis. SCRs were rejected for a given CS presentation if behavioral observations indicated movement or behavior such as coughing and sneezing. 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.

To monitor HR, two disposable 1 cm Ag-AgCl ECG electrodes were placed crossing the chest – one 1 inch below the collar bone and one 1 inch above the last rib bone. The former was placed on the same side of the chest as the participant's non-dominant hand (i.e., the hand on which the SCR electrodes were placed) while the latter was placed on the side of the chest of the participant's dominant hand. These two electrodes served as the positive and negative electrodes while one of the SCR electrodes served as the ground to triangulate the ECG signal.

Apparatus and Stimuli

Three geometrical shapes (a blue circle, red triangle, and green trapezoid) were used as the conditional stimuli (CSs). CSA always appeared on the left side of the screen

and was paired with the US during acquisition training. CSB always appeared on the right side of the screen and was also paired with the US during acquisition training. CS- always appeared in the middle of the screen and was never paired with the US. Which of the three geometrical shapes served as CSA, CSB, or CS- was counterbalanced across participants. The unconditional stimulus (US) was a 1-second scream presented binaurally through headphones at 82 decibels. Such auditory stimuli have successfully served as USs in previous studies (e.g., Lau et al., 2008). In fact, auditory USs have been found to demonstrate equivalent or superior conditioning effects as shock USs (as measured by US-expectancy ratings and skin conductance responses) without the risk of causing pain or excessive anxiety (Neumann & Waters, 2006). Across different auditory US stimuli, a scream-US has been found to produce more robust conditioning effects than a white noise-US (Joos, Vansteenwegen, & Hermans, 2012); thus a scream-US was utilized in the current study. Stimulus delivery was controlled by one computer which presented participants with the CSs and US through E-prime software (Psychology Software Tools, Inc., Pittsburgh, PA, USA). Physiological data acquisition was controlled by a second computer using Acqknowledge software (Biopac Systems, Inc., Goleta, CA, USA).

Procedure

The experiment consisted of several phases that were completed over the course of two days. All participants were asked to abstain from eating or drinking anything (except water) for 8 hours prior to the first session and were scheduled to come in between 8am and 12pm. 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 the BIS and BDI.

Four electrodes were placed on participants for recording HR and SCR and leads were connected from these electrodes to the Biopac system. Next, psychophysiological measures was recorded for a five-minute resting period during which participants were instructed to "please sit quietly and remain still" and left alone in the room (the researcher monitored physiological data acquisition in a room adjacent to the experimental room; the two rooms were interconnected by a door that remained partially open to allow for communication between the researcher and the participant if necessary). Following this baseline period, participants were seated 3 feet in front of a 21" computer monitor placed at eye level which was used to display the CSs. They were asked to put on headphones and told that they may sometimes hear a loud noise through the headphones. Next the researcher trained participants on the US-expectancy scale and asked them to rate their expectancy of hearing the sound as soon as soon as each geometric shape appeared on the screen (participants placed their dominant hand on the desk on top of an expectancy ratings sheet and held a pen so they could record their ratings with minimal movement). Participants were instructed to return their attention immediately to the computer screen following each US-expectancy rating. The experimenter then left the room (throughout all experimental phases of the study, the researcher monitored physiological data acquisition from the interconnected room).

On the first day, participants first underwent a *habituation* phase in which they were presented with four 8-second presentations of each of the three CSs; the inter-trial interval (ITI) varied across 20, 25, and 30 seconds (mean = 25 seconds). The CSs were

presented in random order with the caveat that no more than two trials of each CS were presented sequentially. Immediately following habituation, participants underwent an *acquisition* phase in which they received eight 8-second presentations of each CS using the same randomization procedure and mean ITI. During the last second of each CSA and CSB presentation, the scream-US was presented through the headphones; the CS- was never paired with the US. The acquisition phase was designed to teach subjects to associate both CSA and CSB with the aversive scream so that both stimuli would elicit a conditional response of fear when presented alone.

Following acquisition, participants were instructed to remove the headphones. They then provided subjective valence ratings for each CS. The experimenter disconnected the leads between the Biopac and the electrodes and asked the participant to sit in a recliner chair on the other side of the room from the computer monitor. Participants were given 4 oz of grapefruit juice which they knew may or may not contain 600 mg of crushed caffeine pills. The experimenter was blind to the participant's drug group assignment. Following ingestion of the drink, participants were asked to sit for 45 minutes (they were provided with magazines) to allow for optimal absorption of caffeine. After 45 minutes had elapsed, the experimenter asked the participant to again sit at the desk 3 feet from the computer monitor and reattached the leads from the Biopac to the electrodes. Another 5-minute baseline was recorded prior to commencement of extinction training. During the *first phase of extinction*, all participants received eight 8-second presentations of CSA, CSB, and CS- (with ITIs ranging across 25, 30, and 35 seconds) without any presentations of the US. Following this, during the *second phase of extinction*, half of the participants within each drug group received eight 8-second trials

of CSA while the other half received eight 8-second compound trials during which CSA and CSB appeared simultaneously next to each other on the screen. All participants also received eight 8-second trials of the CS-. Across both extinction phases, CSs were presented in random order with the caveat that no more than two trials of each CS were presented sequentially. Following extinction training, participants again provided subjective valence ratings for each CS.

Group	Habituation	Conditioning	Drug Ingestion	Extinction Phase 1	Extinction Phase 2
Single Placebo	CSA (4) CSB (4) CS- (4)	CSA + US (8) CSB + US (8) CS- (8)	Placebo	CSA (8) CSB (8) CS- (8)	CSA (8) CS- (8)
Compound Placebo	CSA (4) CSB (4) CS- (4)	CSA + US (8) CSB + US (8) CS- (8)	Placebo	CSA (8) CSB (8) CS- (8)	CSAB (8) CS- (8)
Single Caffeine	CSA (4) CSB (4) CS- (4)	CSA + US (8) CSB + US (8) CS- (8)	Caffeine	CSA (8) CSB (8) CS- (8)	CSA (8) CS- (8)
Compound Caffeine	CSA (4) CSB (4) CS- (4)	CSA + US (8) CSB + US (8) CS- (8)	Caffeine	CSA (8) CSB (8) CS- (8)	CSAB (8) CS- (8)

On Day 2, all participants underwent testing to determine the effectiveness of extinction learning and to investigate if a US-only presentation would reinstate conditional responding to CSA. Upon arriving in the laboratory, participants provided subjective valence ratings for each CS. The electrodes were placed just as during Day 1 for psychophysiological recording and they sat in front of the same monitor wearing the same headphones. They were re-familiarized with the expectancy scale and asked to again record their expectancy of hearing the noise at the onset of each CS presentation. The *test* phase consisted of eight 8-second presentations of CSA and CS-, presented in random order with the caveat that no more than two trials of each CS were presented sequentially and with ITIs ranging across 20, 25, and 30 seconds. Halfway through this

test phase (i.e., following four CSA and four CS- presentations), all participants received a presentation of the scream-US during an intertrial interval. Upon completion of this phase, participants again provided subjective valence ratings for each of the three CSs.

Group	Spontaneous Recovery Test	Reinstatement	Reinstatement Test
Single Placebo	CSA (4) CS- (4)	US during intertrial interval (1)	CSA (4) CS- (4)
Compound Placebo	CSA (4) CS- (4)	US during intertrial interval (1)	CSA (4) CS- (4)
Single Caffeine	CSA (4) CS- (4)	US during intertrial interval (1)	CSA (4) CS- (4)
Compound Caffeine	CSA (4) CS- (4)	US during intertrial interval (1)	CSA (4) CS- (4)

Results

For results of Acquisition, Extinction, and Test phases, only significant findings are reported. Statistical values (i.e., F-values, t-ratios, and p-values) for all nonsignificant findings are reported in Appendix A.

Baseline

The mean BIS score in this sample was 22.61 and the mean BDI score was 7.57. One-way ANOVAs revealed no significant differences among the four groups on the BIS ($F(3,69) = 0.25$, $p = 0.84$) or the BDI ($F(3,69) = 0.63$, $p = 0.60$). There were also no significant differences among the groups on age ($F(3,69) = 1.61$, $p = 0.20$), gender ($\chi^2(3, N = 70) = 5.22$ ($p = 0.16$), or ethnicity ($\chi^2(21, N = 70) = 23.23$ ($p = 0.33$).

Acquisition

Skin Conductance Response (SCR)

HLM analyses were conducted for SCRs to each conditional stimulus across the eight acquisition trials. For CSA, the intercept (i.e., SCR at the first acquisition trial) was

significantly different from zero (b = 0.28, t(61) = 4.60, p < 0.001) and the slope of SCRs to CSA across acquisition was significantly different from zero (b = 0.03, t(439) = 3.08, p < 0.005), indicating that all participants demonstrated a significant increase in SCR to CSA across acquisition (Figure 1). Similarly, for CSB, the intercept (i.e., SCR at the first acquisition trial) was significantly different from zero (b = 0.24, t(61) = 4.10, p < 0.001) and the slope of SCRs to CSB across acquisition was significantly different from zero (b = 0.03, t(439) = 2.72, p < 0.01), indicating that all participants demonstrated a significant increase in SCR to CSB across acquisition (Figure 2). For CS-, the intercept (i.e., SCR at the first acquisition trial) was significantly different from zero (b = 0.36, t(61) = 4.89, p < 0.001) and the slope of SCRs to CS- across acquisition was significantly different from zero (b = -0.05, t(439) = -4.26, p < 0.001) indicating that all participants demonstrated a significant decrease in SCR to CS- across acquisition (Figure 3).

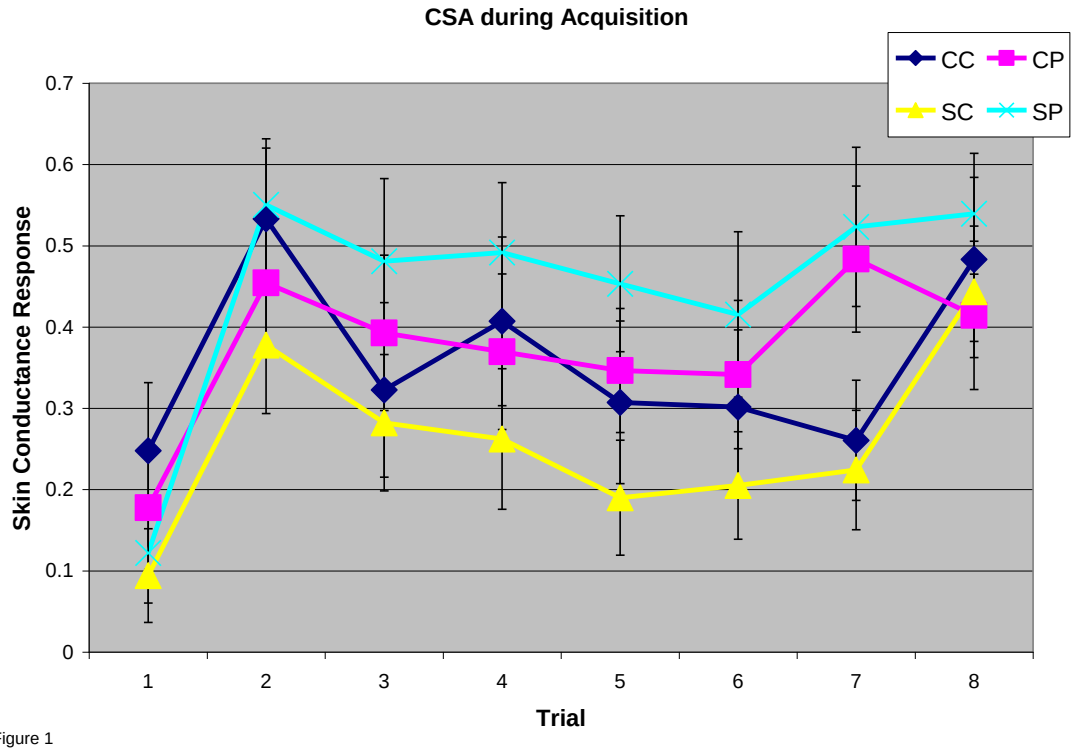


Figure 1

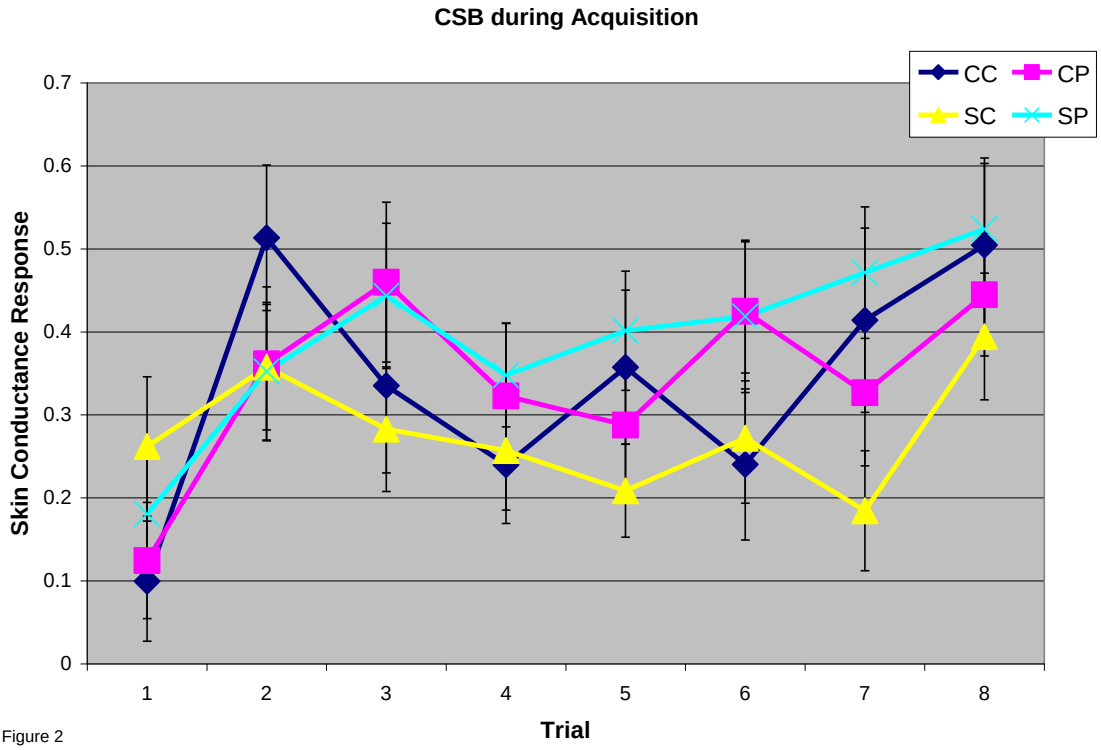


Figure 2

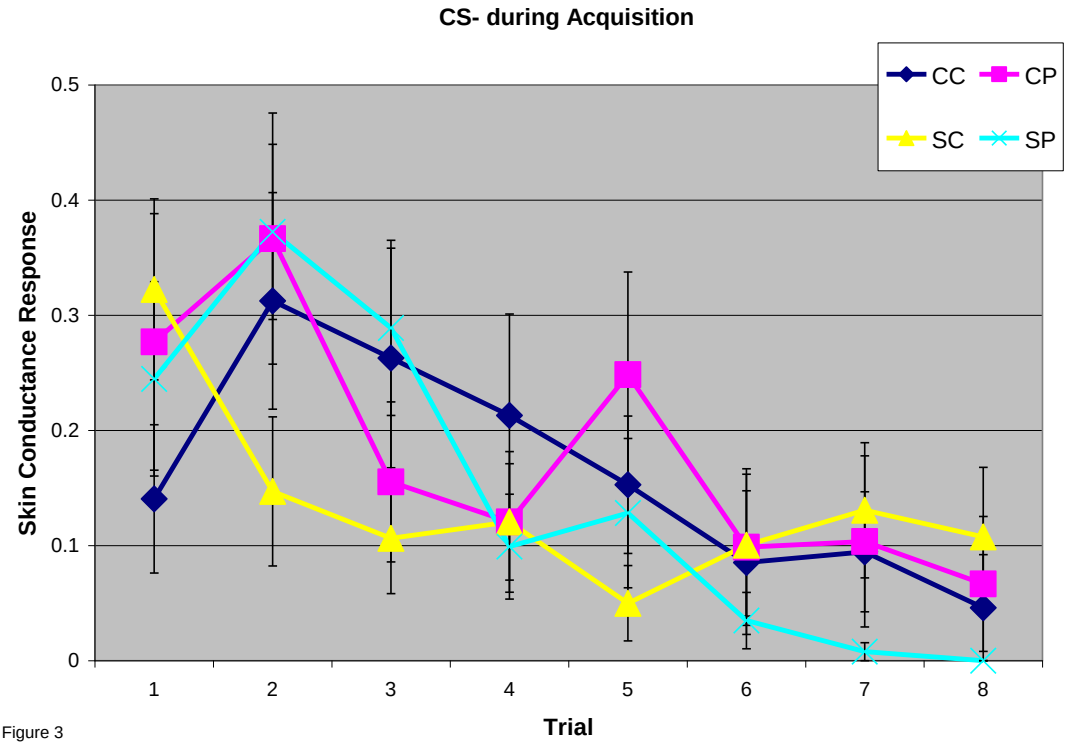


Figure 3

Expectancy Ratings

HLM analyses were conducted for subjective US-expectancy ratings to each conditional stimulus across the eight acquisition trials. For CSA, the slope of expectancy ratings to CSA across acquisition was significantly different from zero ($b = 0.49$, $t(355) = 3.03$, $p < 0.005$) indicating that all participants demonstrated a significant increase in US-expectancy ratings to CSA across acquisition (Figure 4). Similarly, for CSB, the slope of expectancy ratings across acquisition was significantly different from zero ($b = 0.42$, $t(355) = 2.34$, $p < 0.05$) indicating that all participants demonstrated a significant increase in US-expectancy ratings to CSB across acquisition (Figure 5). For CS-, participants demonstrated no changes in US-expectancy ratings across acquisition (Figure 6).

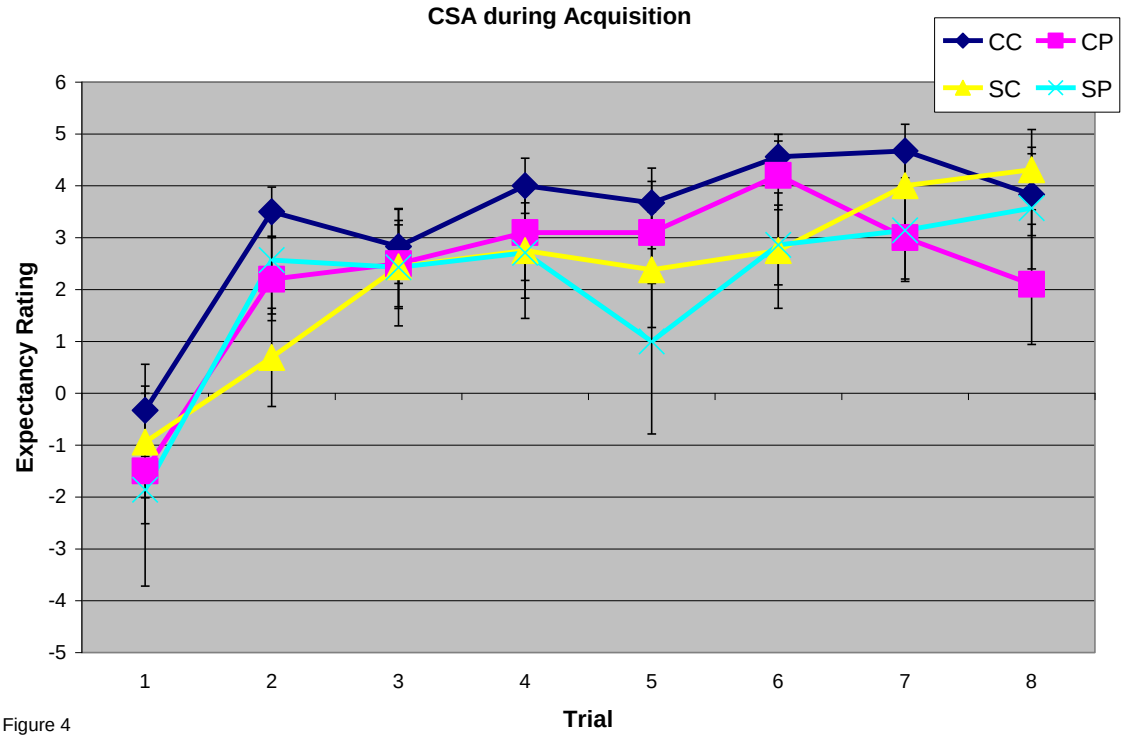


Figure 4

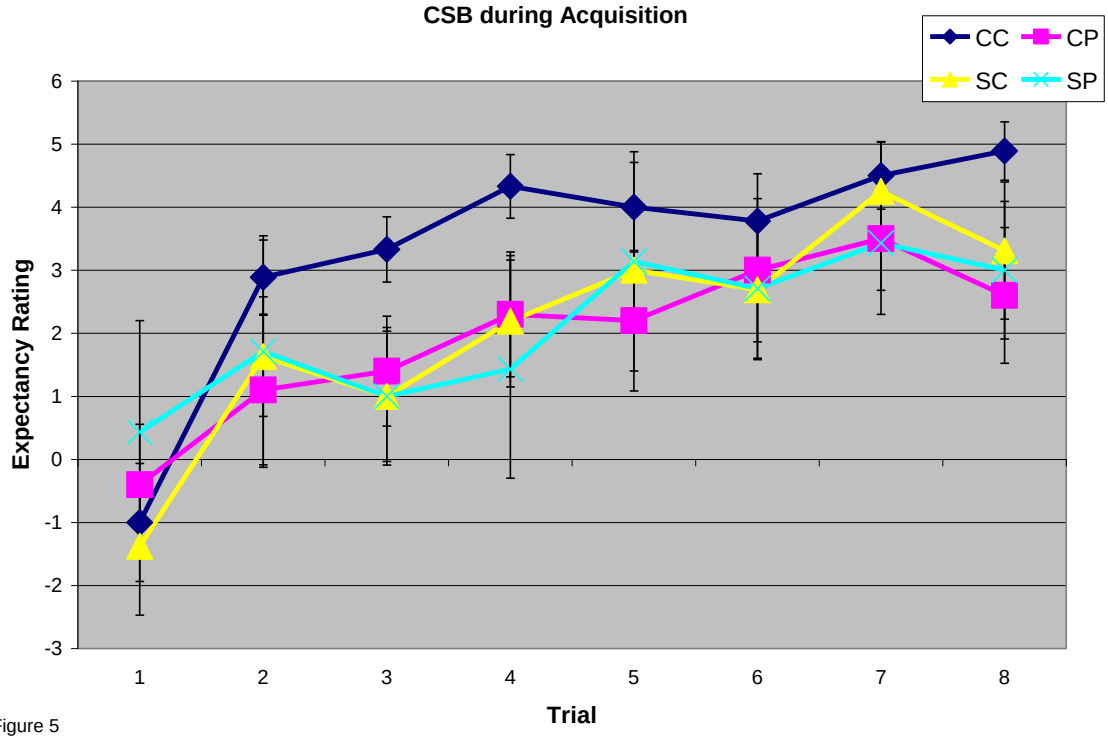


Figure 5

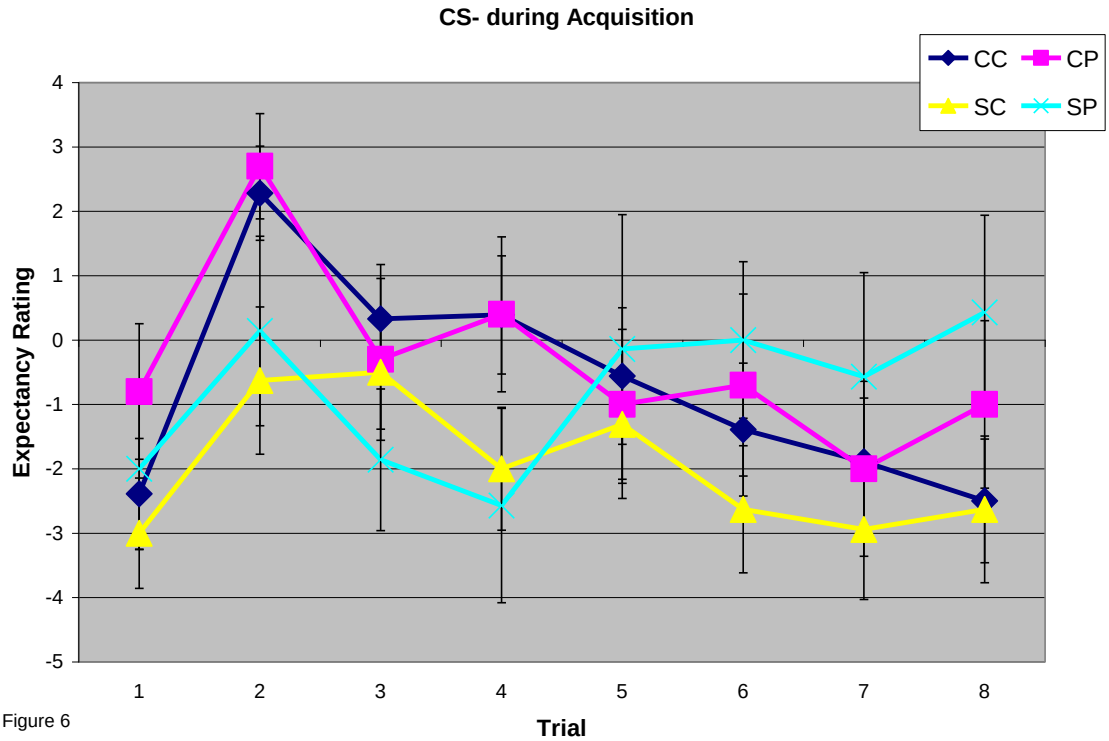


Figure 6

Heart Rate

HLM analyses were conducted for mean heart rate during each conditional stimulus across the eight acquisition trials. For each of the three conditional stimuli, the intercept (i.e., mean heart rate during the first trial of acquisition) was significantly different from zero (CSA: $b = 72.10$, $t(61) = 35.712$, $p < 0.001$; CSB: $b = 71.52$, $t(61) = 33.23$, $p < 0.001$; CS-: $b = 73.25$, $t(61) = 38.66$, $p < 0.001$).

Acquisition to Extinction

A 2 (Drug Group: Placebo, Caffeine) x 2 (Time: Baseline prior to Habituation and Acquisition, Baseline prior to Extinction) repeated measures ANOVA examined whether ingestion of caffeine impacted mean skin conductance levels during the baseline period. Results indicated a significant Time x Group interaction effect ($F(1,62) = 5.64$, $p < 0.05$, $\eta^2 = 0.08$). Tests of simple effects examined the interaction and revealed that participants who ingested caffeine demonstrated a significant increase in mean skin conductance levels during baseline from pre-acquisition to pre-extinction ($F(1,62) = 9.01$, $p < 0.005$, $\eta^2 = 0.13$) whereas participants who did not ingest caffeine demonstrated no change from the pre-acquisition baseline period to the pre-extinction baseline period ($F(1,62) = 0.13$, $p = 0.72$, $\eta^2 = 0.00$; Figure 7). A 2 (Drug Group: Placebo, Caffeine) x 2 (Time: Baseline prior to Habituation and Acquisition, Baseline prior to Extinction) repeated measures ANOVA on heart rate mean scores revealed no significant effects of time, group, or their interaction.

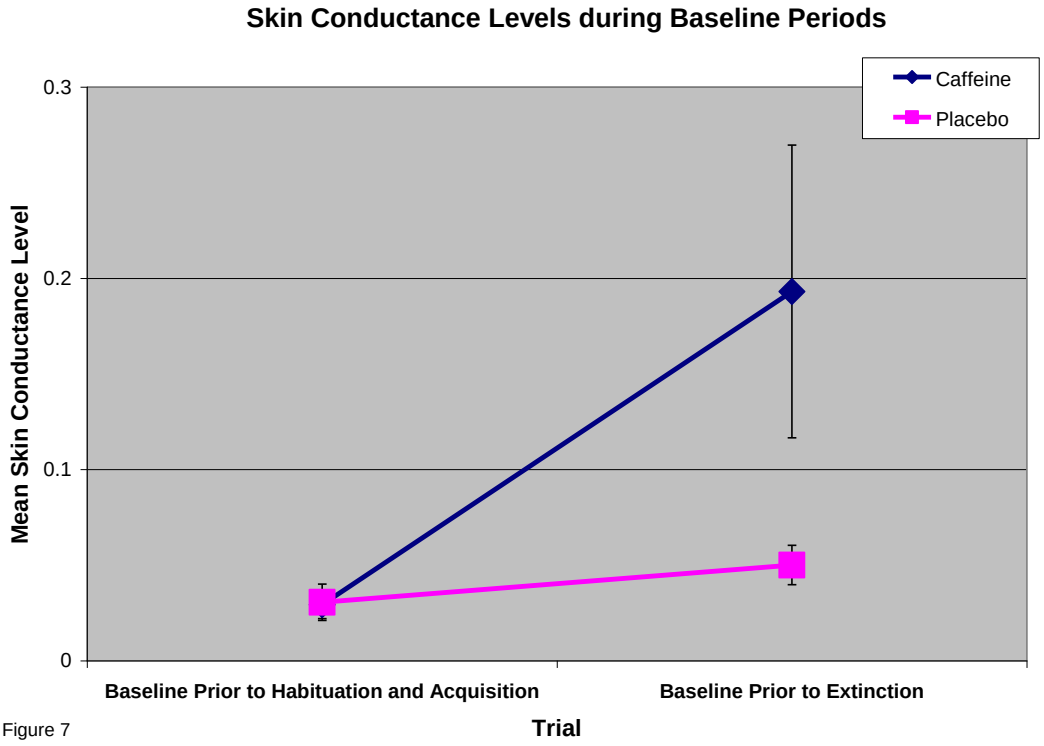


Figure 7

Extinction Phase 1

Skin Conductance Response (SCR)

HLM analyses were conducted for SCRs to each conditional stimulus across the eight trials of the first phase of extinction. For CSA, the intercept (i.e., SCR at the first trial of extinction) was significantly different from zero ($b = 0.42$, $t(61) = 7.34$, $p < 0.001$). The slope was also significantly different from zero ($b = -0.05$, $t(439) = -5.41$, $p < 0.001$) and the group slopes were significantly different from each other ($b = 0.01$, $t(439) = 2.65$, $p < 0.01$; Figure 8).

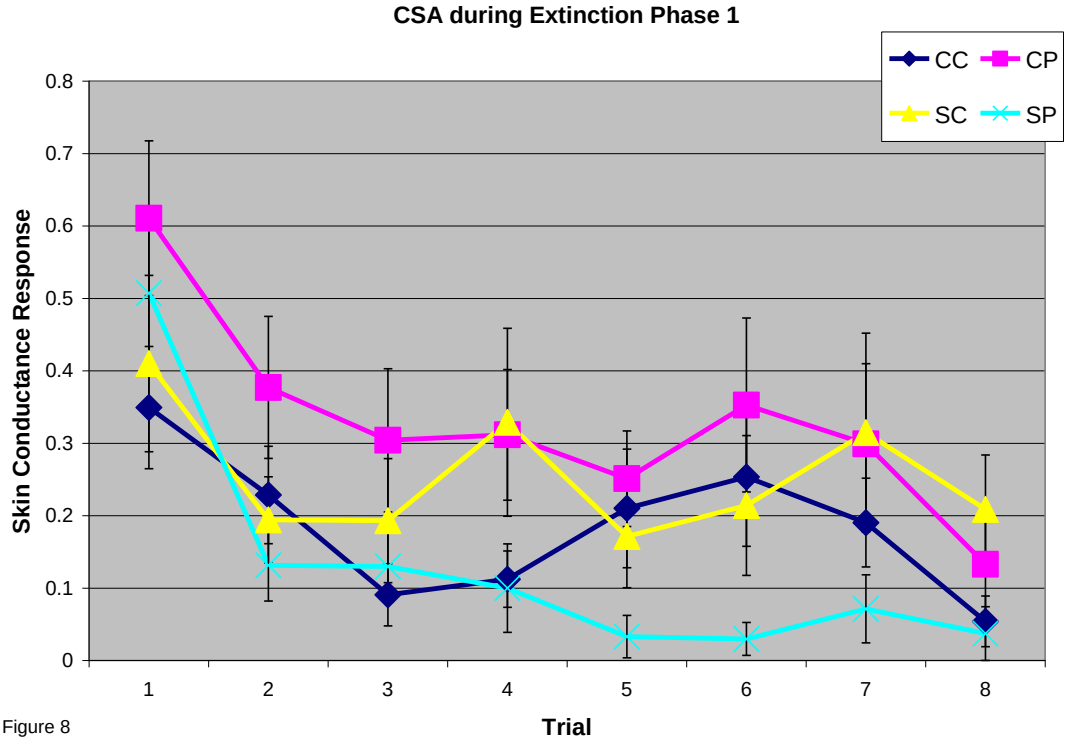


Figure 8

To more closely examine these group differences, participants were divided into caffeine versus placebo groups. The analyses revealed that the intercept was significantly different from zero ($b = 0.43$, $t(61) = 7.84$, $p < 0.001$) and there was a significant difference between the caffeine group and the placebo group ($b = -0.14$, $t(61) = -2.02$, $p < 0.05$) such that participants who ingested caffeine demonstrated significantly lower SCRs to CSA at the first trial of extinction compared with participants who ingested placebo. The slope of SCRs to CSA was significantly different from zero ($b = -0.05$, $t(439) = -6.30$, $p < 0.001$) and the group slopes were significantly different from each other ($b = 0.03$, $t(439) = 3.19$, $p < 0.005$) such that participants who ingested caffeine had a significantly flatter slope than those who ingested placebo (Figure 9).

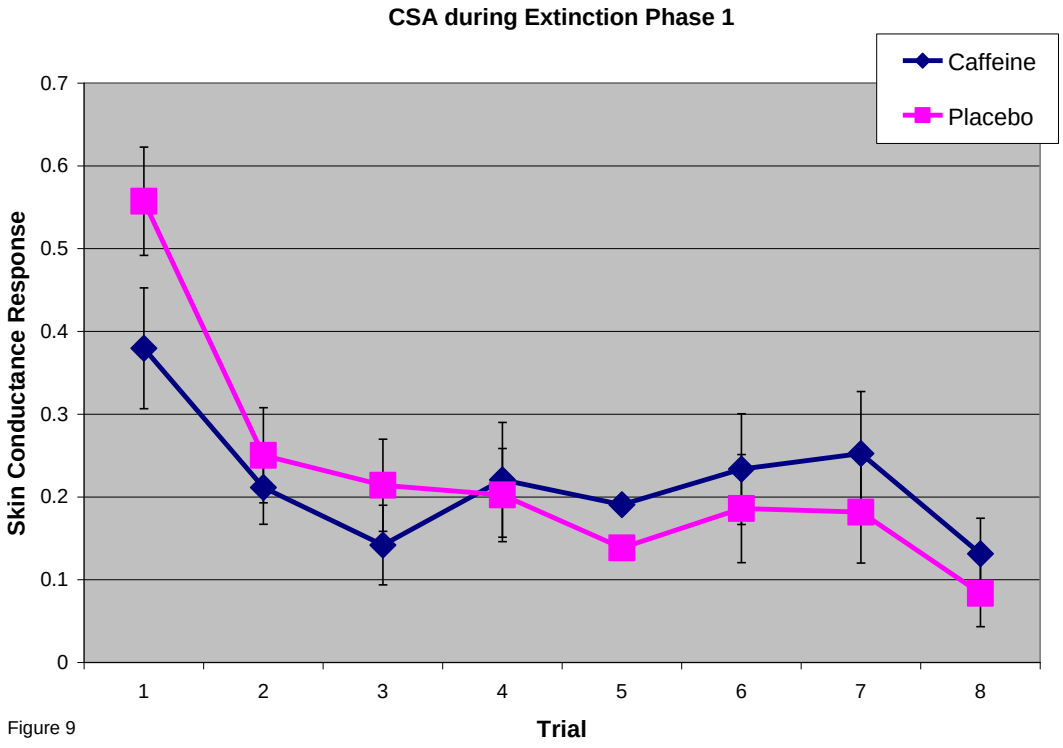


Figure 9

For CSB, the intercept (i.e., SCR at the first extinction trial) was significantly different from zero ($b = 0.36$, $t(61) = 5.69$, $p < 0.001$) and the slope of SCRs to CSB was significantly different from zero ($b = -0.04$, $t(439) = -4.16$, $p < 0.001$; Figure 10). Although there were no significant group differences, given the a priori hypothesis that ingestion of caffeine versus placebo would have a significant effect on extinction, participants were divided into caffeine versus placebo groups. The analyses revealed that the intercept was significantly different from zero ($b = 0.40$, $t(61) = 6.09$, $p < 0.001$). The slope of SCR to CSB was also significantly different from zero ($b = -0.04$, $t(439) = -4.52$, $p < 0.001$) and the group slopes were significantly different from each other ($b = 0.03$, $t(439) = 1.92$, $p = 0.05$) such that those participants who ingested caffeine had a significantly flatter slope than those who ingested placebo (Figure 11).

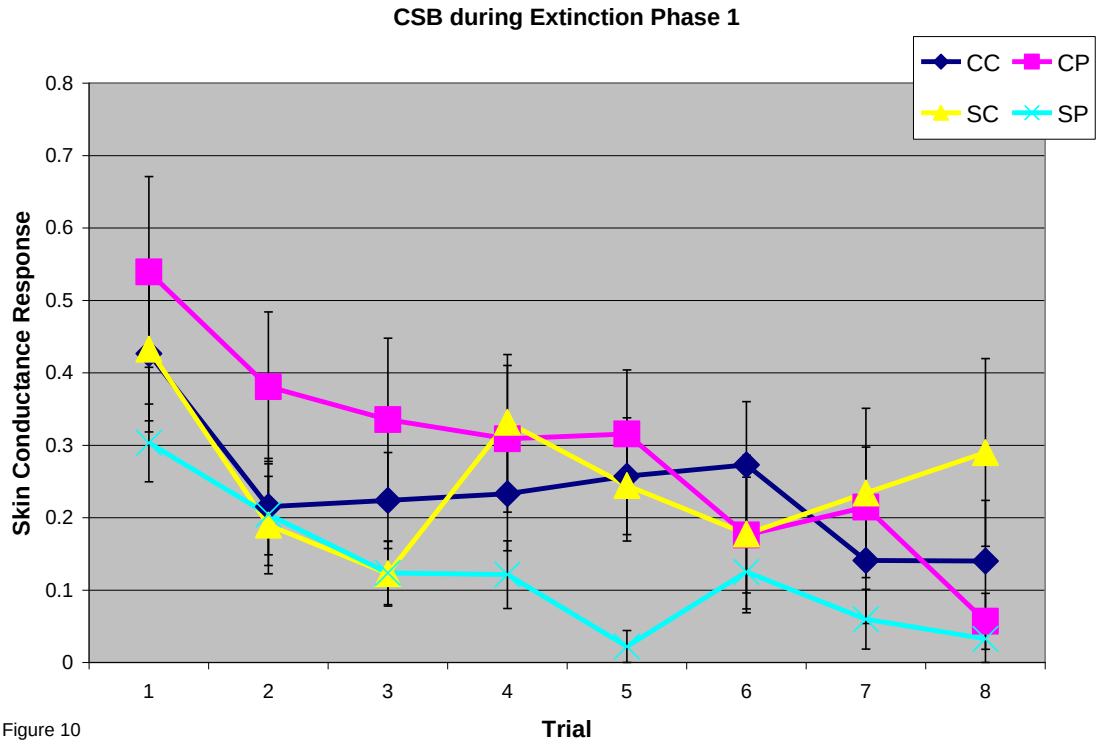


Figure 10

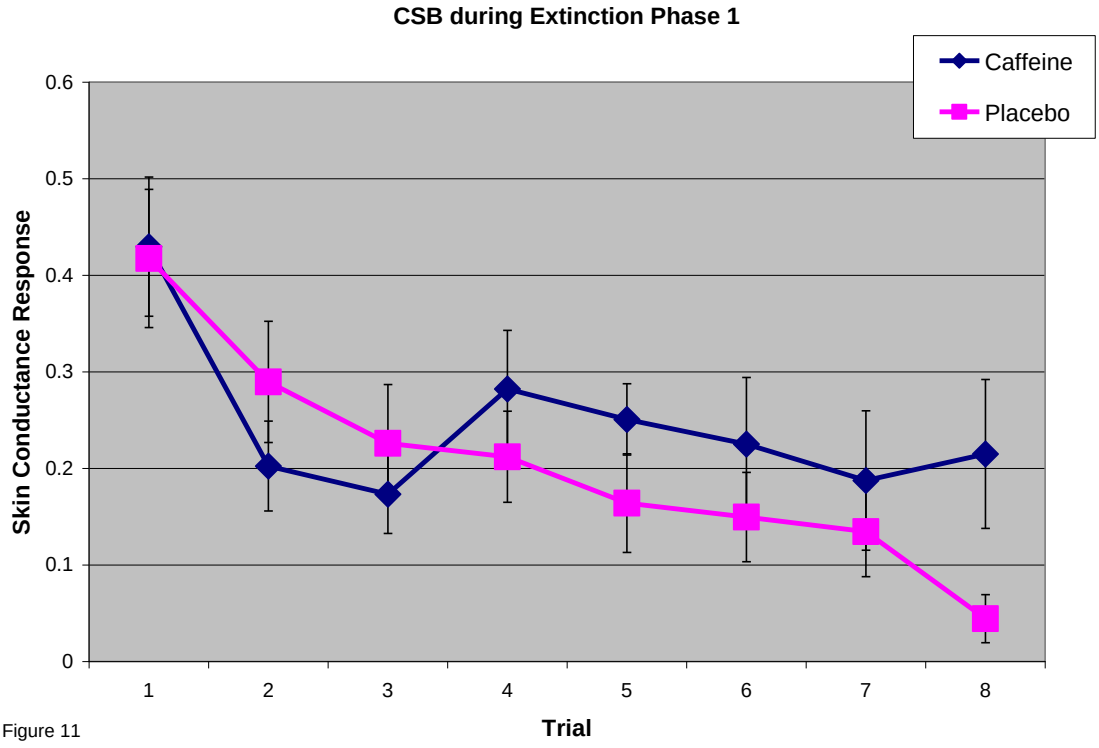


Figure 11

For CS-, the intercept (i.e., SCR at the first extinction trial) was significantly different from zero ($b = 0.36$, $t(61) = 4.89$, $p < 0.001$) and the slope of SCR to CS- was significantly different from zero ($b = -0.05$, $t(439) = -4.26$, $p < 0.001$; Figure 12). Dividing participants into caffeine versus placebo groups yielded very similar results: the intercept was significantly different from zero ($b = 0.24$, $t(61) = 6.13$, $p < 0.001$) and the slope was significantly different from zero ($b = -0.02$, $t(439) = -4.83$, $p < 0.001$; Figure 13).

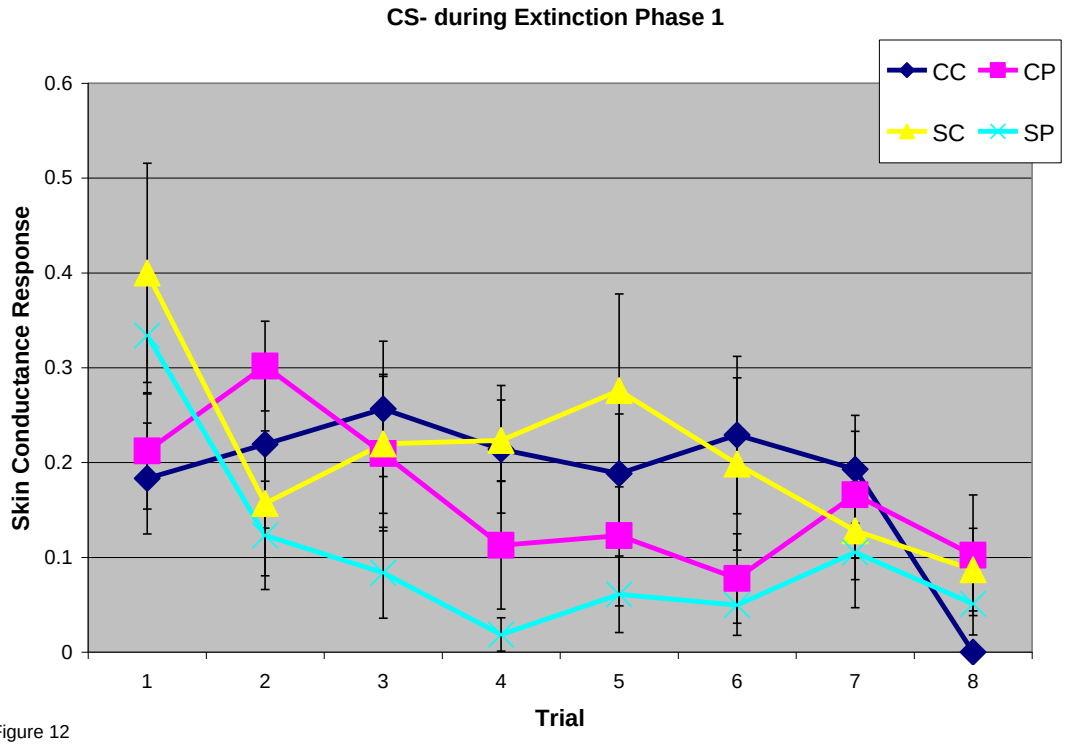


Figure 12

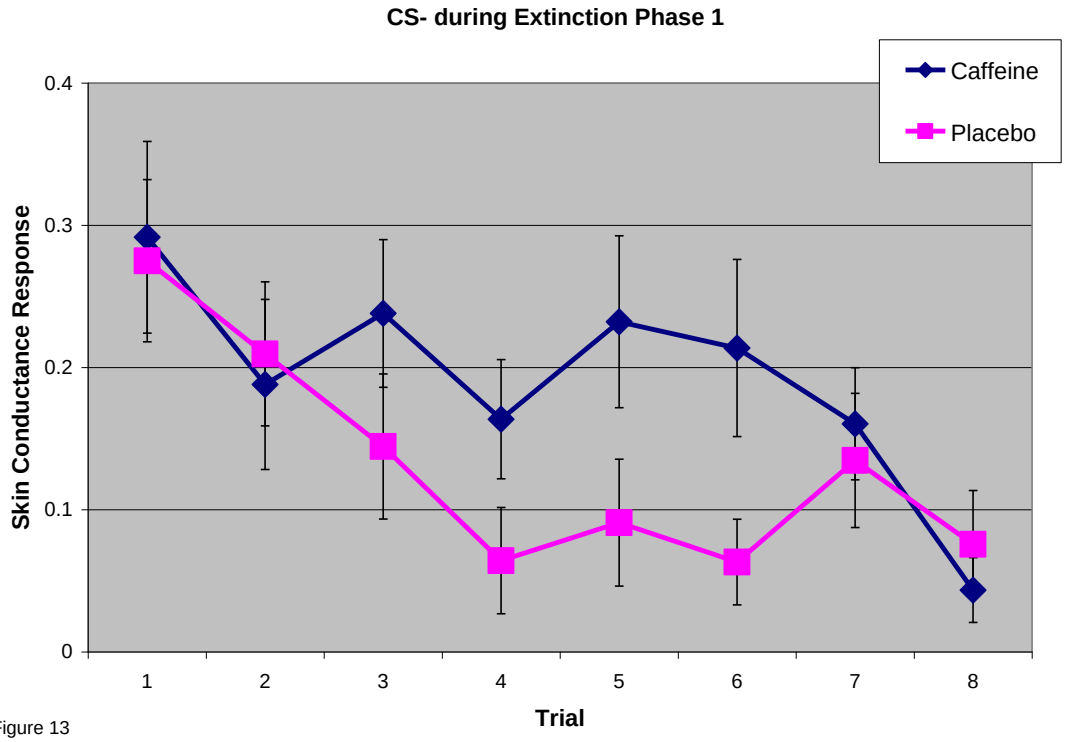


Figure 13

Expectancy Ratings

For CSA, the slope of expectancy ratings to CSA was significantly different from zero ($b = -0.23$, $t(488) = -1.95$, $p = 0.05$; Figure 14). Dividing participants into caffeine versus placebo groups yielded the same results: the slope was significantly different from zero ($b = -0.23$, $t(488) = -2.25$, $p < 0.05$; Figure 15). For CSB, there were no significant findings.

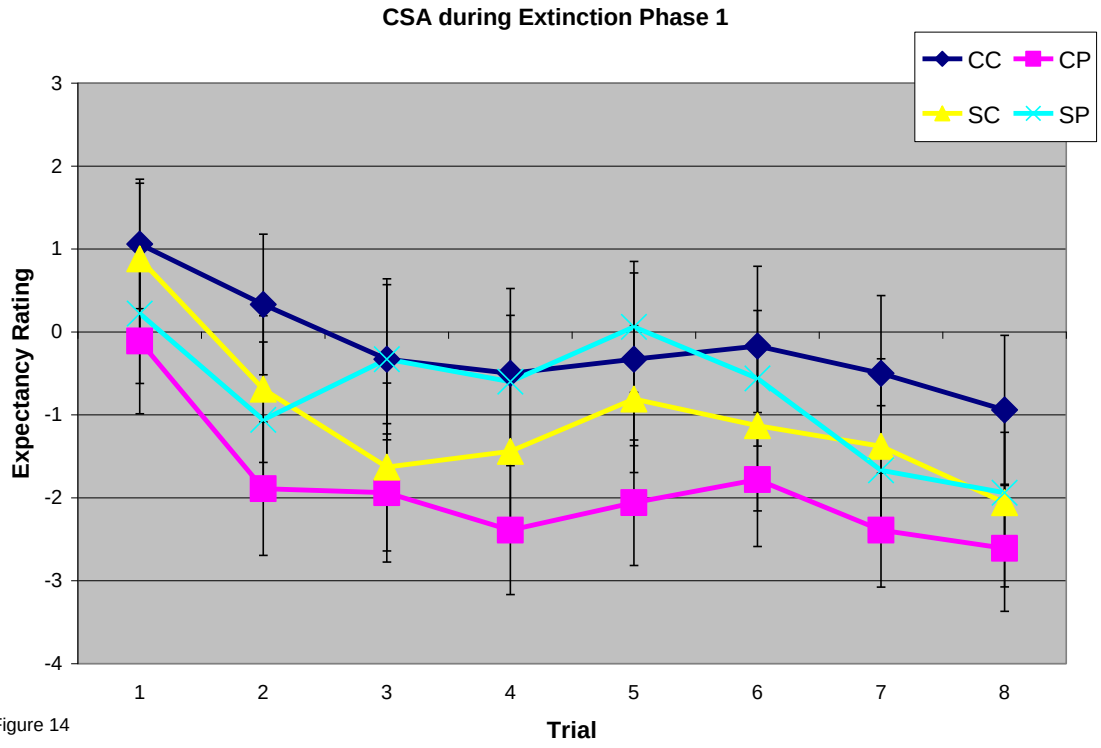


Figure 14

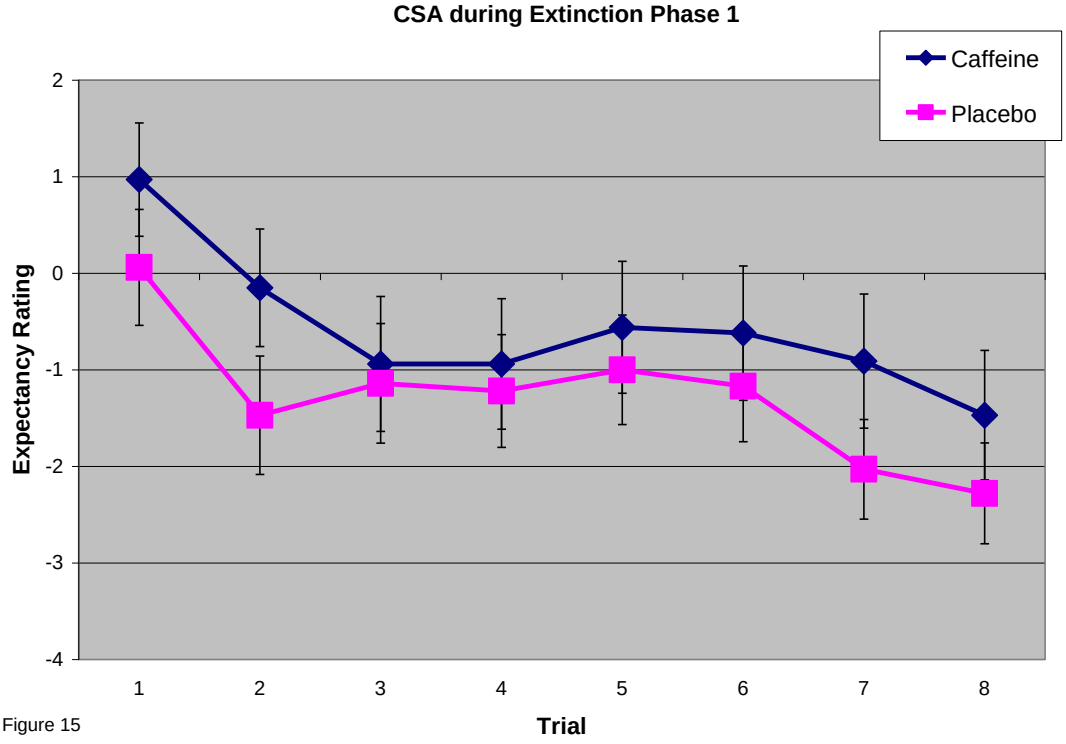


Figure 15

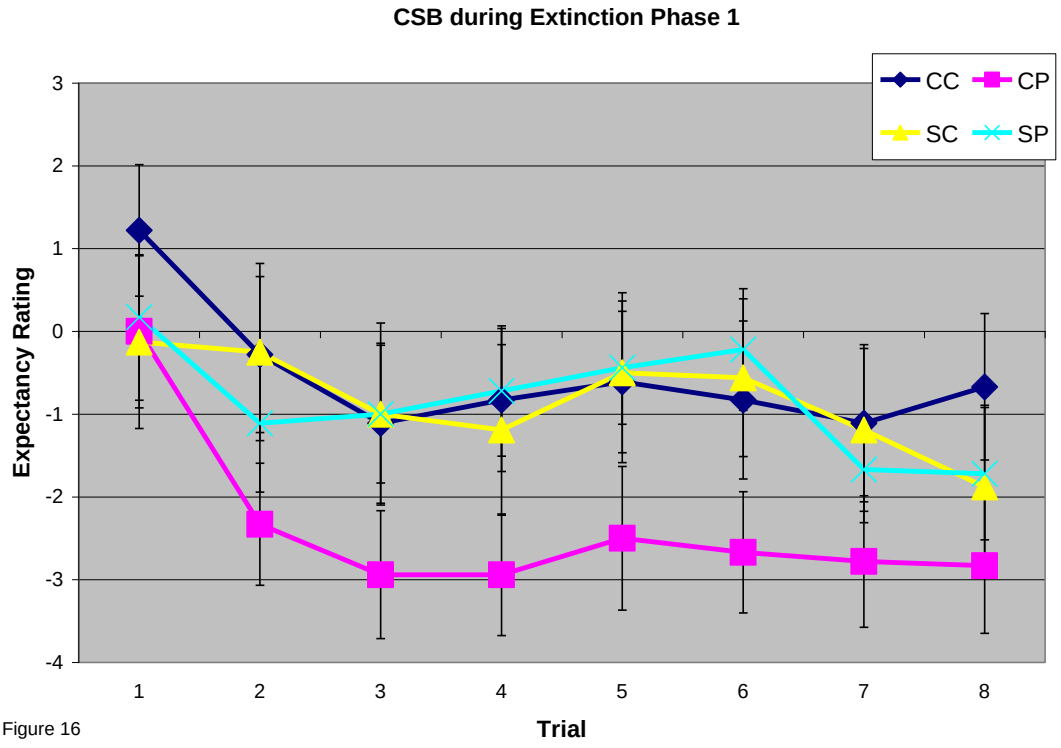


Figure 16

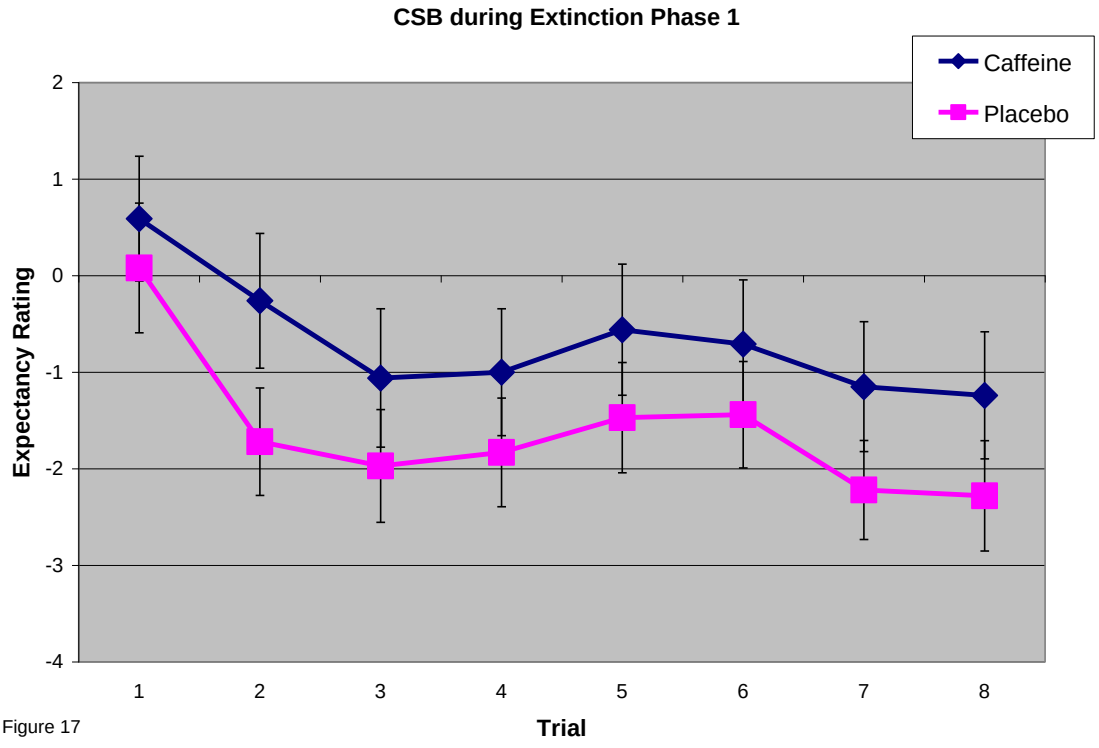


Figure 17

For CS-, the intercept (i.e., the US-expectancy rating at the first extinction trial) was significantly different from zero ($b = -2.27$, $t(68) = -3.28$, $p < 0.005$; Figure 18). Dividing participants into caffeine versus placebo groups yielded slightly different results: the intercept was still significantly different from zero ($b = -2.27$, $t(68) = -4.16$, $p < 0.001$) and the slope was also significantly different from zero ($b = -0.20$, $t(488) = -2.56$, $p < 0.05$; Figure 19).

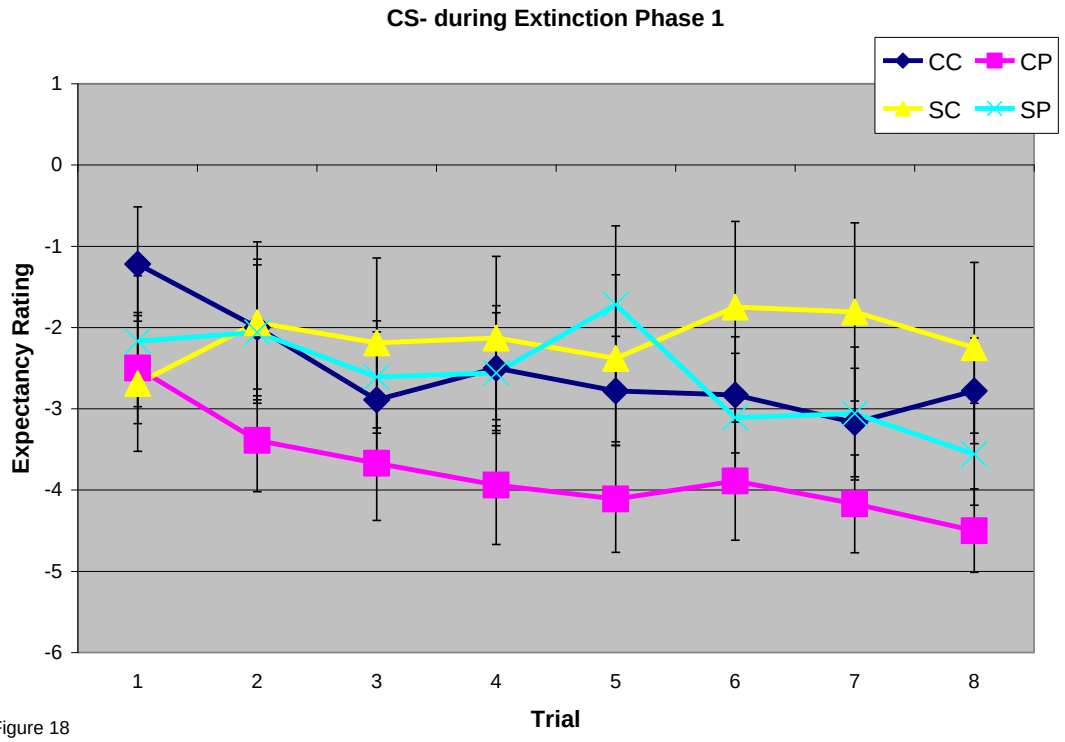


Figure 18

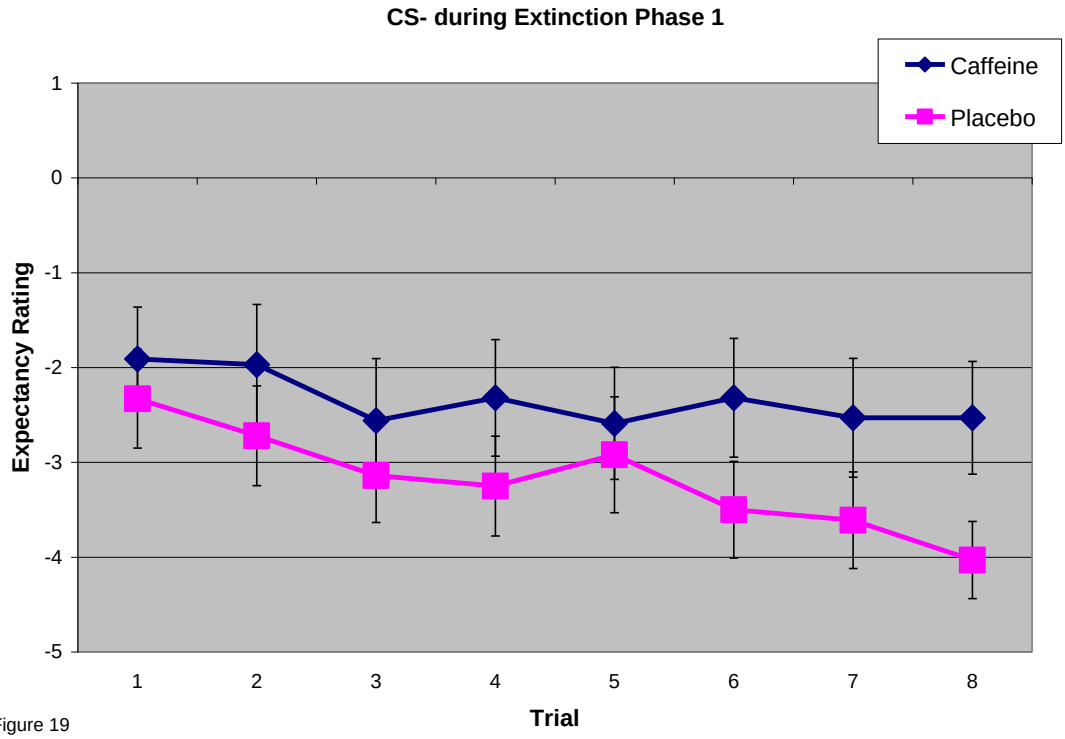


Figure 19

Heart Rate

For CSA, the intercept (i.e., mean heart rate during the first trial of extinction) was significantly different from zero ($b = 78.07$, $t(63) = 32.87$, $p < 0.001$) and there were significant group differences at this intercept ($b = -3.84$, $t(63) = -3.084$, $p < 0.005$). Dividing participants into caffeine versus placebo groups yielded slightly different results. The intercept was still significantly different from zero ($b = 76.80$, $t(63) = 40.07$, $p < 0.001$) with significant group differences at this intercept ($b = -8.89$, $t(63) = -3.60$, $p < 0.001$). In addition, there were significant group slope differences ($b = 0.37$, $t(453) = 1.92$, $p = 0.05$) such that participants who ingested caffeine had a significantly steeper and positive slope than those who ingested placebo who had a flatter slope (Figure 20).

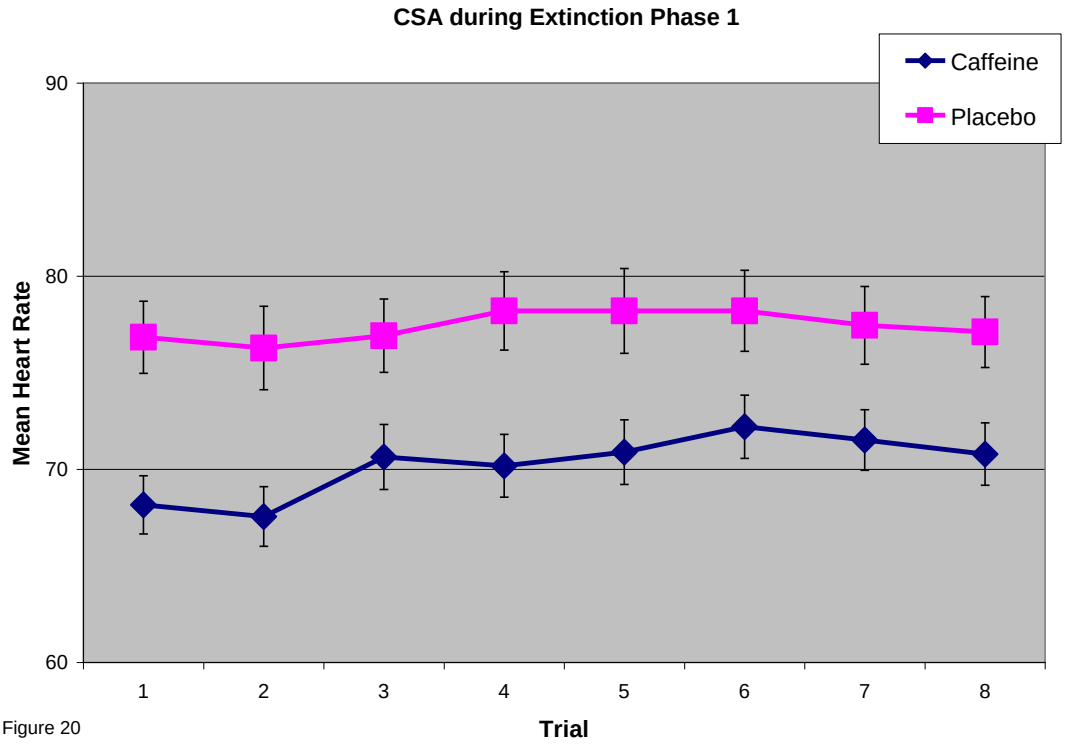


Figure 20

For CSB, the intercept (i.e., mean heart rate during the first trial of extinction) was significantly different from zero ($b = 78.52$, $t(63) = 32.56$, $p < 0.001$) and there were significant group differences at this intercept ($b = -4.09$, $t(63) = -3.40$, $p = 0.001$). In addition, there were significant group slope differences ($b = 0.16$, $t(451) = 1.97$, $p = 0.05$). Dividing participants into caffeine versus placebo groups yielded very similar results. The intercept was significantly different from zero ($b = 77.15$, $t(63) = 39.70$, $p < 0.001$) with significant group differences at this intercept ($b = -9.47$, $t(63) = -4.01$, $p < 0.001$). In addition, there were significant group slope differences ($b = 0.38$, $t(453) = 2.03$, $p < 0.05$) such that participants who ingested caffeine had a significantly steeper and positive slope than those who ingested placebo who had a flatter slope (Figure 21).

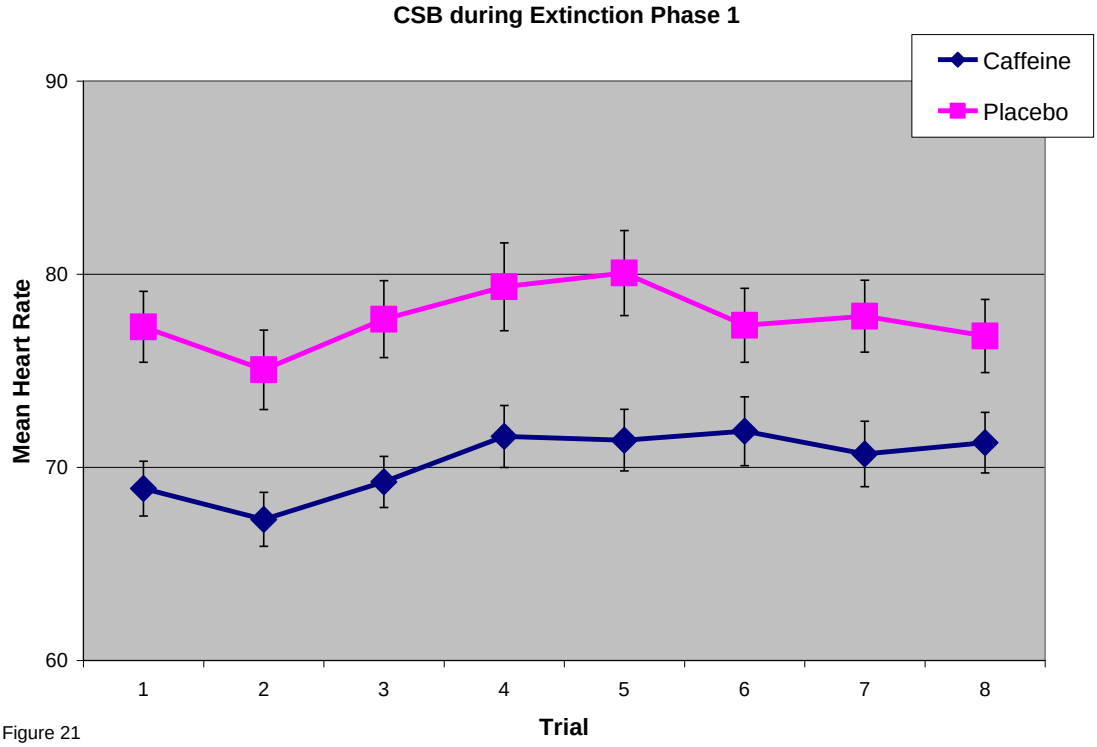


Figure 21

For CS-, the intercept (i.e., mean heart rate during the first trial of extinction) was significantly different from zero ($b = 79.03$, $t(63) = 35.43$, $p < 0.001$) and there were significant group differences at this intercept ($b = -4.34$, $t(63) = -3.58$, $p < 0.001$). In addition, there were significant group slope differences ($b = 0.25$, $t(453) = 2.75$, $p < 0.01$). Dividing participants into caffeine versus placebo groups yielded similar results. The intercept was significantly different from zero ($b = 77.59$, $t(63) = 42.21$, $p < 0.001$) and there were significant group differences at this intercept ($b = -10.01$, $t(63) = -4.17$, $p < 0.001$). In addition, there were significant group slope differences ($b = 0.65$, $t(453) = 3.14$, $p < 0.005$) such that participants who ingested caffeine had a significantly steeper and positive slope than those who ingested placebo who had a flatter slope.

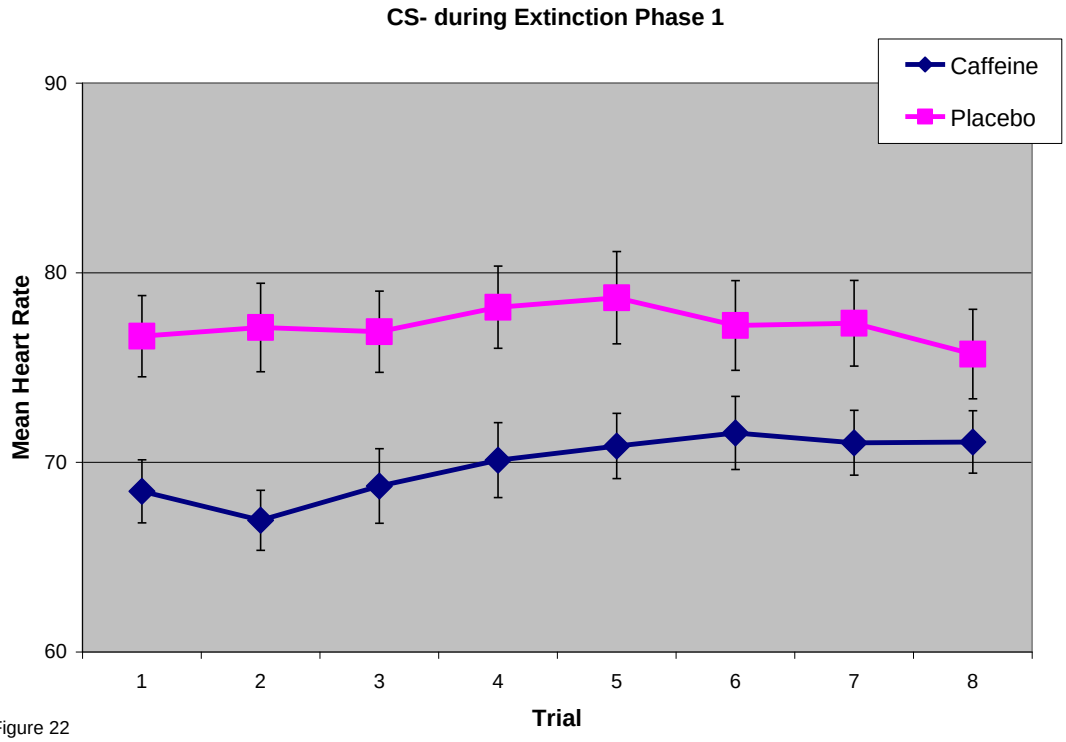


Figure 22

Extinction Phase 2

Skin Conductance Response (SCR): CSA

For CSA, the intercept (i.e., the SCR at the first trial of the second phase of extinction training) was significantly different from zero ($b = 0.18$, $t(61) = 2.54$, $p = 0.01$) and there were significant group differences at this intercept ($b = 0.14$, $t(61) = 3.27$, $p < 0.005$). In addition, there were significant group slope differences ($b = -0.01$, $t(439) = -2.60$, $p = 0.01$; Figure 23). To more closely examine these group differences, pairwise HLM analyses were conducted.

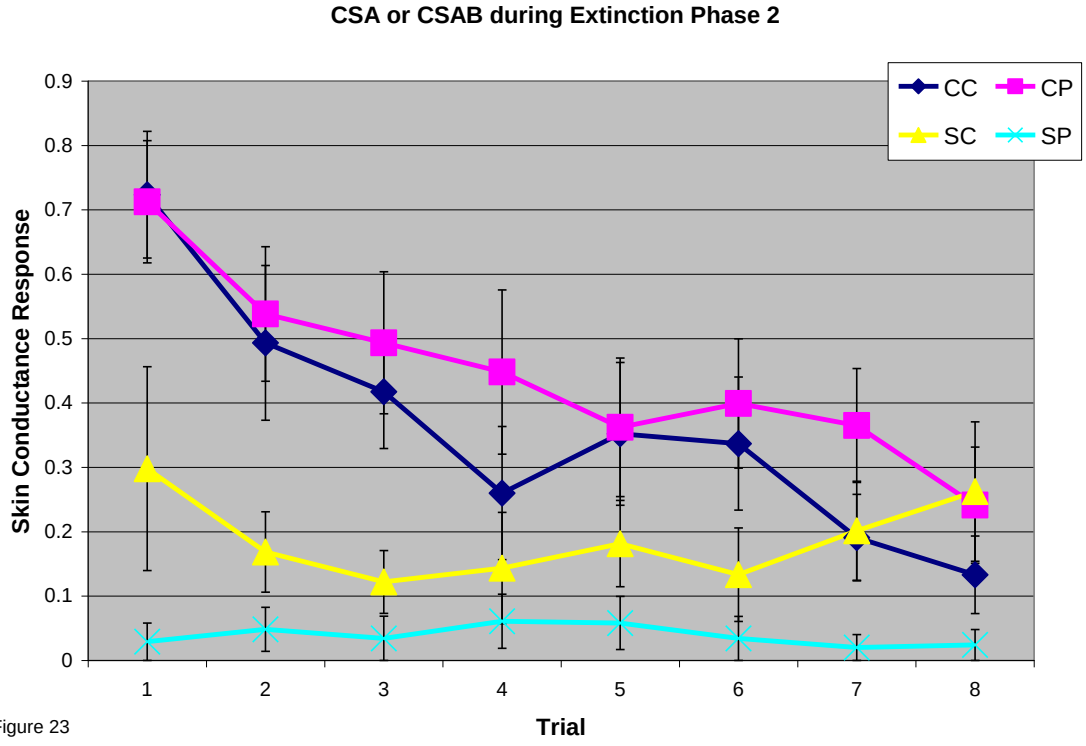


Figure 23

SP versus CP: What is the impact of compound trials on extinction?

There was a significant group difference at the intercept (b = 0.64, t(29) = 5.14, p < 0.001) indicating that CP demonstrated a significantly higher SCR at the first trial of the second phase of extinction than SP. There was also a significant group slope difference (b = -0.05, t(215) = -2.69, p< 0.01) indicating CP had a significantly steeper slope than SP.

SP versus SC: What is the impact of caffeine on extinction?

There was a significant group difference at the intercept (b = 0.07, t(30) = 2.01, p = 0.05) indicating that SC demonstrated a significantly higher SCR at the first trial of the second phase of extinction than SP.

SC versus CC: What is the impact of caffeine on the difference between single and compound trials during extinction?

The intercept was significantly different from zero ($b = -0.78$, $t(30) = -2.57$, $p < 0.05$) and there was a significant difference between the groups ($b = 0.48$, $t(30) = 3.58$, $p = 0.001$) indicating that CC demonstrated a significantly higher SCR at the first trial of the second phase of extinction than SC. Similarly, the slope was significantly different from zero ($b = 0.14$, $t(222) = 3.43$, $p < 0.001$) and there was a significant difference between the groups ($b = -0.07$, $t(222) = -3.75$, $p< 0.001$) indicating that CC had a significantly steeper slope than SC.

Skin Conductance Response (SCR): CS-

For CS-, the intercept (i.e., the SCR at the first trial of the second phase of extinction training) was significantly different from zero ($b = 0.12$, $t(61) = 2.66$, $p = 0.01$) and there were significant group differences at this intercept ($b = 0.06$, $t(61) = 2.52$, $p < 0.05$; Figure 24). To more closely examine the group differences at the intercept, pairwise HLM analyses were conducted.

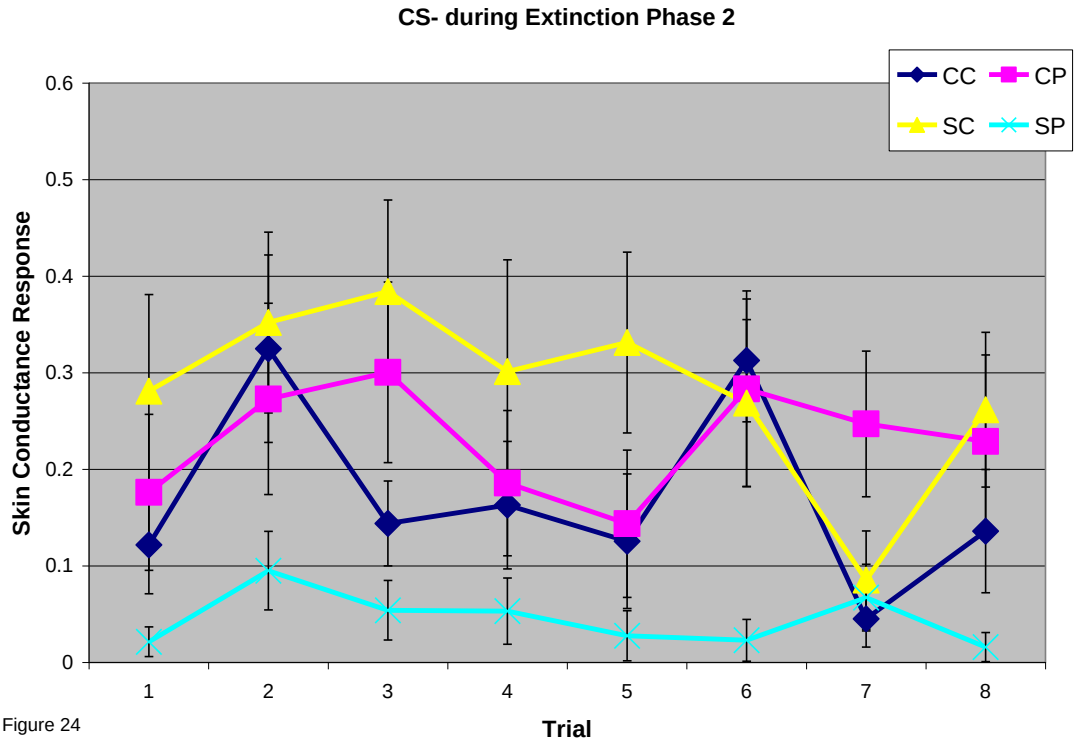


Figure 24

SP versus CP: What is the impact of compound trials?

There were no significant findings.

SP versus SC: What is the impact of caffeine?

There was a significant group difference at the intercept ($b = 0.16$, $t(30) = 3.27$, $p < 0.005$) indicating that SC demonstrated a significantly higher SCR to CS- at the first trial of the second phase of extinction than SP.

SC versus CC: What is the impact of caffeine on the difference between single and compound trials?

The intercept was significantly different from zero ($b = 0.70$, $t(30) = 2.37$, $p < 0.05$).

Expectancy Ratings: CSA

For CSA, the intercept (i.e., the US-expectancy rating at the first trial of the second phase of extinction training) was significantly different from zero ($b = 1.41$, $t(68) = -2.18$, $p < 0.05$) and the slope was significantly different from zero ($b = -0.27$, $t(488) = -2.84$, $p = 0.005$; Figure 25). Although this analysis did not reveal a significant group difference at the intercept, given the a priori hypothesis that compound versus single trials as well as caffeine ingestion versus placebo ingestion would have an impact on expectancy ratings, pairwise HLM analyses were conducted.

SP versus CP: What is the impact of compound trials on extinction?

Analyses revealed that there was a significant group slope difference ($b = -1.42$, $t(250) = -3.59$, $p < 0.001$) indicating that CP had a significantly steeper slope than SP.

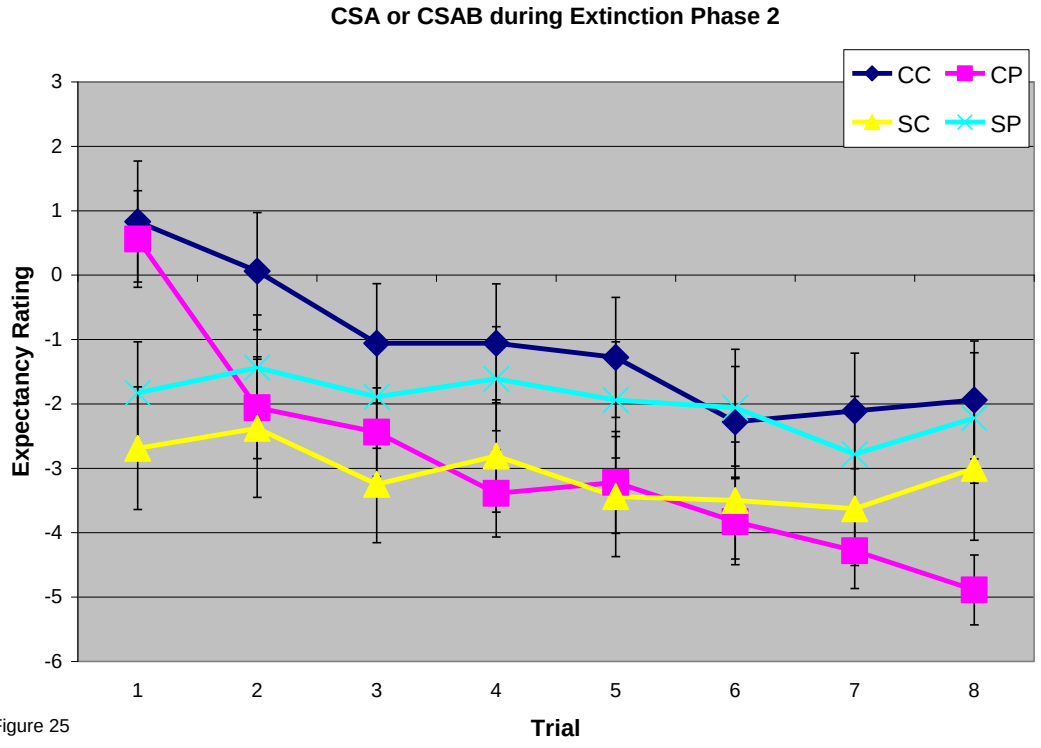


Figure 25

SP versus SC: What is the impact of caffeine?

Analyses revealed no significant findings.

SC versus CC: What is the impact of caffeine on the difference between single and compound trials?

Analyses revealed that the intercept was significantly different from zero (b = -9.13, t(32) = -2.54, $p < 0.05$) and there was a significant difference between the groups b = 3.28, (t(32) = 2.35, $p < 0.05$), indicating that the mean expectancy rating in CC was significantly higher than in SC.

Expectancy Ratings: CS-

For CS-, the intercept was significantly different from zero (b = -3.56, t(68) = -5.63, $p < 0.001$). Pairwise HLM analyses also indicated that the intercept was significantly different from zero (SP versus CP: b = -3.30, t(34) = -4.26, $p < 0.001$; SP versus SC: b = -3.30, t(32) = -4.26, $p < 0.001$; SC versus CC: b = 0.30, t(30) = 2.37, $p < 0.05$).

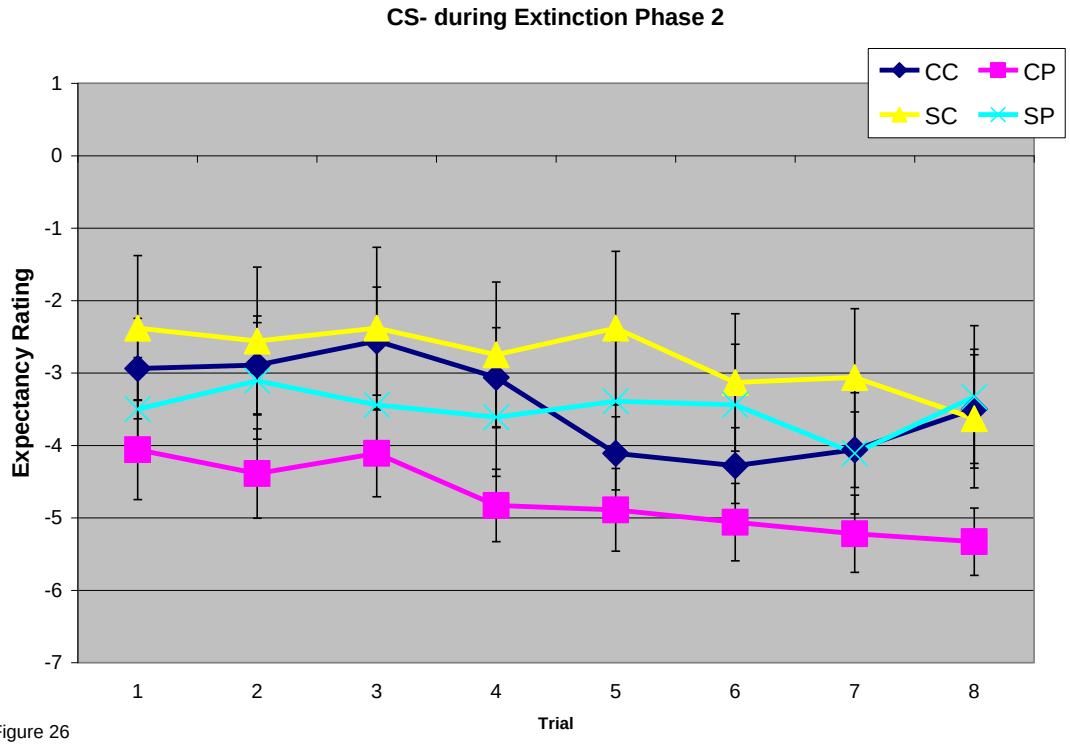


Figure 26

Heart Rate

For CSA, the intercept (i.e., mean heart rate during the first trial of the second phase of extinction training) was significantly different from zero ($b = 79.04$, $t(63) = 36.28$, $p < 0.001$) and there were significant group differences at this intercept ($b = -2.76$, $t(63) = -2.35$, $p < 0.05$; Figure 27). Pairwise HLM analyses were conducted to more closely examine group differences. Across these analyses, the only significant finding was that the intercept was different from zero (SP versus CP: $b = 80.97$, $t(30) = 29.99$, $p < 0.001$; SP versus SC: $b = 80.98$, $t(31) = 29.99$, $p < 0.001$; SC versus CC: $b = 66.86$, $t(31) = 9.49$, $p < 0.001$). Only one pairwise analysis revealed a significant group difference at this intercept, SP versus SC: $b = -5.03$, $t(31) = -3.21$, $p < 0.005$, indicating that SC demonstrated significantly lower mean heart rate during the first trial of the second phase of extinction than SP.

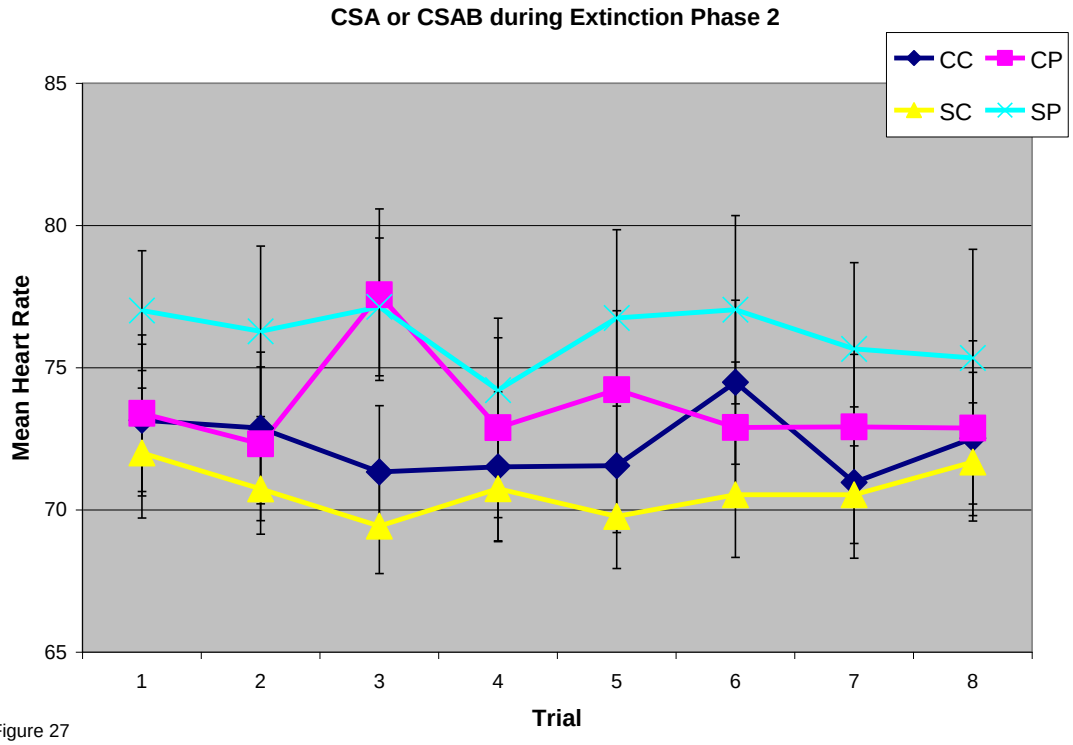


Figure 27

For CS-, the intercept (i.e., mean heart rate during the first trial of the second phase of extinction training) was significantly different from zero ($b = 79.71$, $t(63) = 34.49$, $p < 0.001$) and there were significant group differences at this intercept ($b = -2.42$, $t(63) = -1.99$, $p = 0.05$; Figure 28). Pairwise HLM analyses were conducted to more closely examine group differences. Across these analyses, the only significant finding was that the intercept was different from zero (SP versus CP: $b = 80.79$, $t(30) = 28.32$, $p < 0.001$; SP versus SC: $b = 80.79$, $t(31) = 28.32$, $p < 0.001$; SC versus CC: $b = 72.81$, $t(31) = 9.36$, $p < 0.001$). Only one pairwise analysis revealed a significant group difference at this intercept, SP versus SC: $b = -3.70$, $t(31) = -2.16$, $p < 0.05$, indicating that SC demonstrated significantly lower mean heart rate during the first trial of the second phase of extinction than SP.

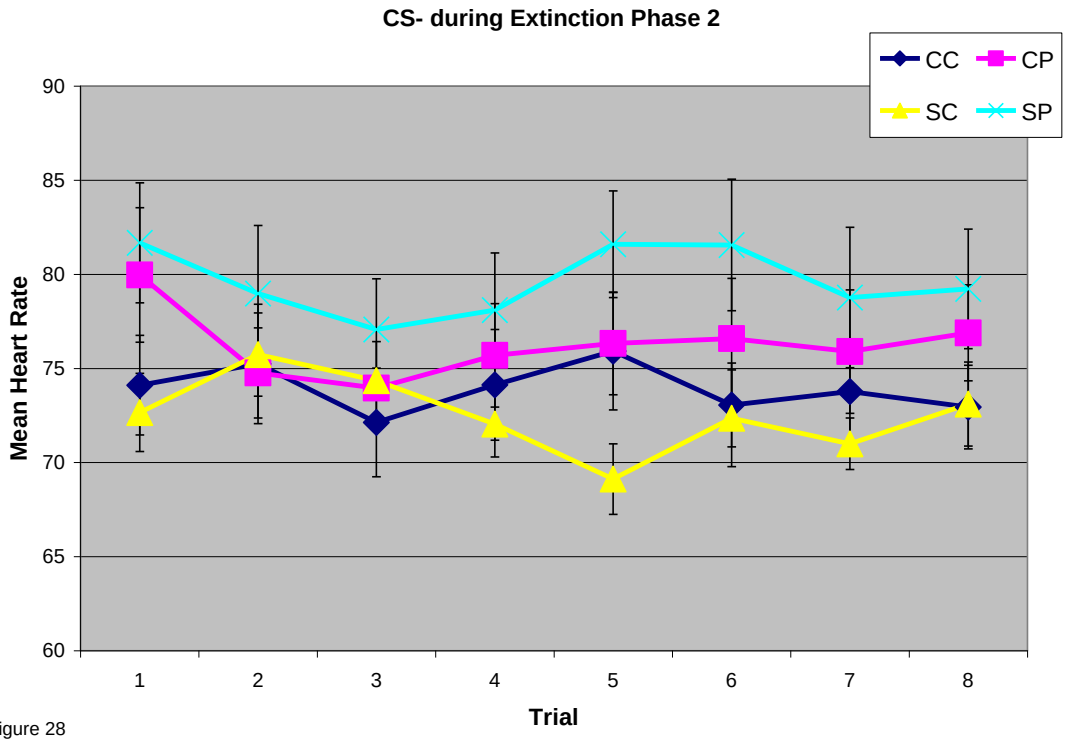


Figure 28

Change from Extinction Phase 1 to Extinction Phase 2

Skin Conductance Response (SCR)

To examine the impact of compound trials, a 4 (Group: SP, CP, SC, CC) x 2 (Time: Extinction Phase 1 Trial 8, Extinction Phase 2 Trial 1) repeated measures ANOVA to CSA revealed a significant effect of Time ($F(1,59) = 33.95$, $p < 0.001$, $\eta^2 = 0.37$), Group ($F(3,59) = 7.78$, $p < 0.001$, $\eta^2 = 0.28$), and the Time x Group interaction ($F(3,59) = 9.00$, $p < 0.001$, $\eta^2 = 0.31$; Figure 29). Tests of simple effects examined the interaction and revealed that participants in the CP and CC groups demonstrated a significant increase in skin conductance responses from the last trial of Extinction Phase 1 to the first trial of Extinction Phase 2 (CP: $F(1,59) = 24.58$, $p < 0.001$, $\eta^2 = 0.29$; CC: $F(1,59) = 34.96$, $p < 0.001$, $\eta^2 = 0.37$) whereas participants in the SP and SC groups demonstrated no change in skin conductance responses. Thus, there was a significant increase in SCRs only for those participants who were presented with the novel compound stimulus but no change for participants who continued to be presented with CSA on its own.

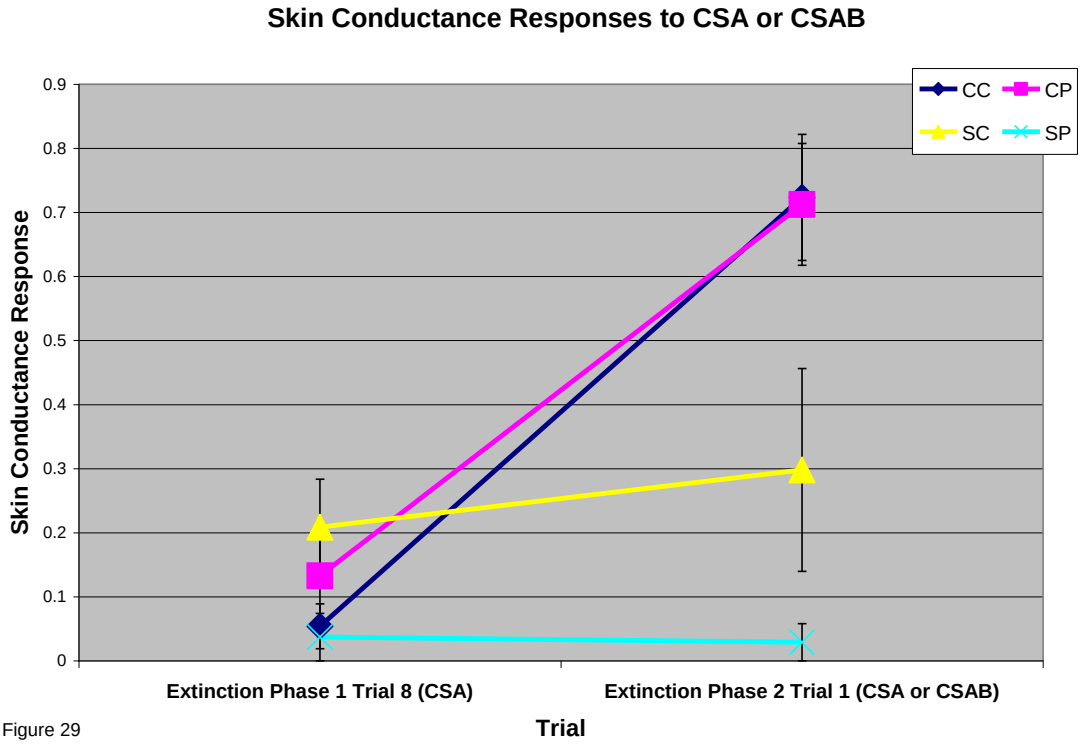


Figure 29

For CS-, a 4 (Group: SP, CP, SC, CC) x 2 (Time: Extinction Phase 1 Trial 8, Extinction Phase 2 Trial 1) repeated measures ANOVA revealed a significant effect of Time ($F(1,59) = 7.23$, $p < 0.01$, $\eta^2 = 0.11$).

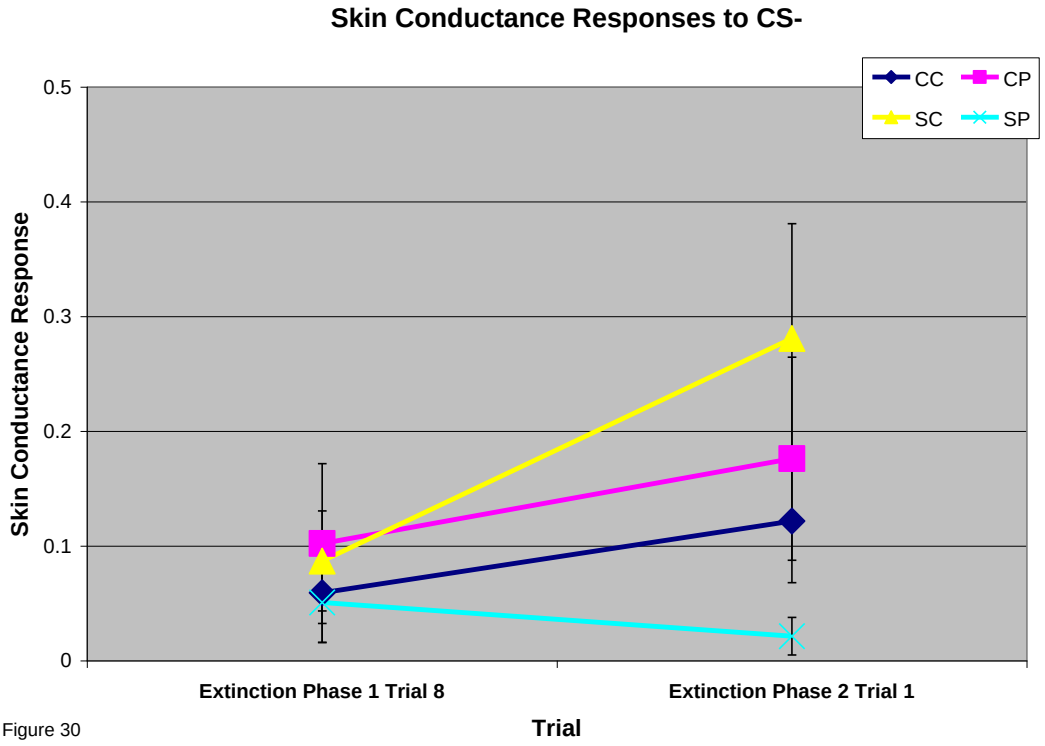


Figure 30

Expectancy Ratings

For CSA, a 4 (Group: SP, CP, SC, CC) x 2 (Time: Extinction Phase 1 Trial 8, Extinction Phase 2 Trial 1) repeated measures ANOVA revealed a significant effect of Time ($F(1,66) = 9.73$, $p < 0.005$, $\eta^2 = 0.13$) and the Time x Group interaction ($F(3,66) = 5.68$, $p < 0.005$, $\eta^2 = 0.21$; Figure 31). Tests of simple effects examined the interaction and revealed that participants in the CP and CC groups demonstrated a significant increase in US-expectancy ratings from the last trial of Extinction Phase 1 to the first trial of Extinction Phase 2 (CP: $F(1,66) = 20.50$, $p < 0.001$, $\eta^2 = 0.24$; CC: $F(1,66) = 6.46$, $p < 0.05$, $\eta^2 = 0.10$) whereas participants in the SP and SC groups demonstrated no change in skin conductance responses. Thus, there was a significant increase in US-expectancy ratings only for those participants who were presented with the novel compound stimulus

but no change for participants who continued to be presented with CSA on its own. For CS-, there were no significant effects of time, group, or their interaction.

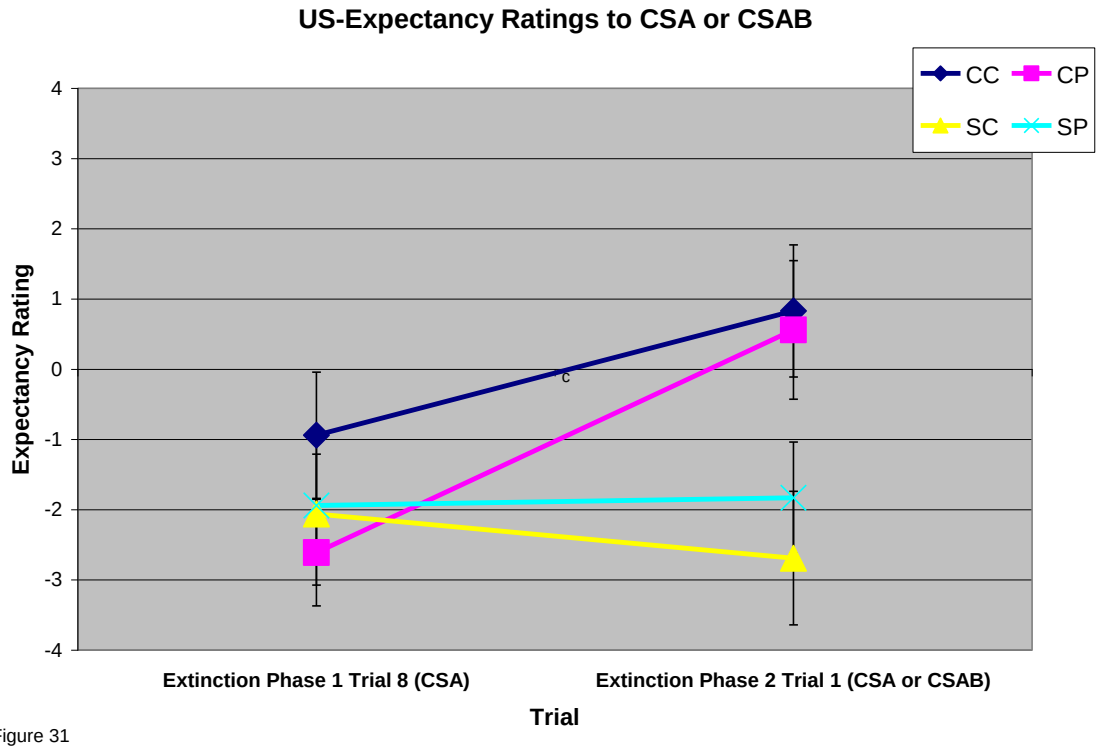


Figure 31

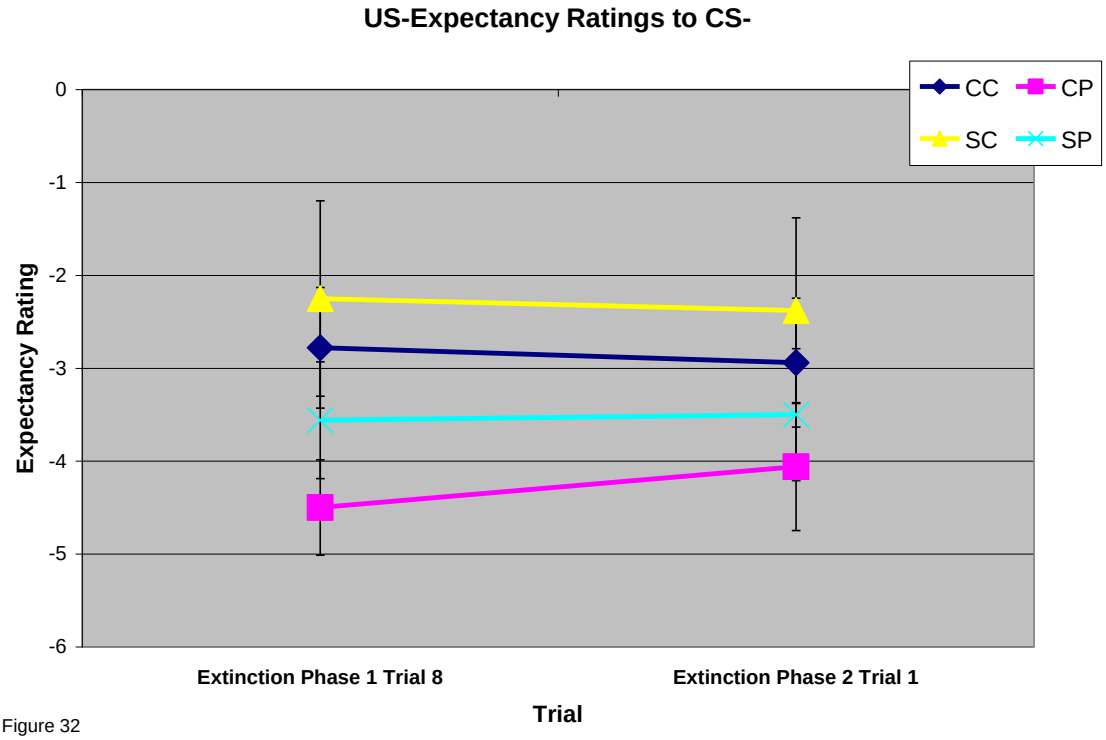


Figure 32

Spontaneous Recovery Test

Skin Conductance Response (SCR)

A one-way ANOVA revealed a significant effect of group at the first trial of CSA at one-week follow-up ($F(3,61) = 6.21$, $p = 0.001$; Figure 33)). A series of independent-samples t-tests were conducted to more closely examine these group differences. To examine the impact of compound versus single extinction trials, SP and CP were compared ($t(30) = 4.67$, $p < 0.001$) and revealed that SCR in SP was significantly higher than in CP. There were no significant group differences between SP and SC or between SC and CC. Interestingly, SP versus CC revealed a significant group difference ($t(30) = 2.78$, $p < 0.01$) indicating that SCR in SP was significantly higher than in CC.

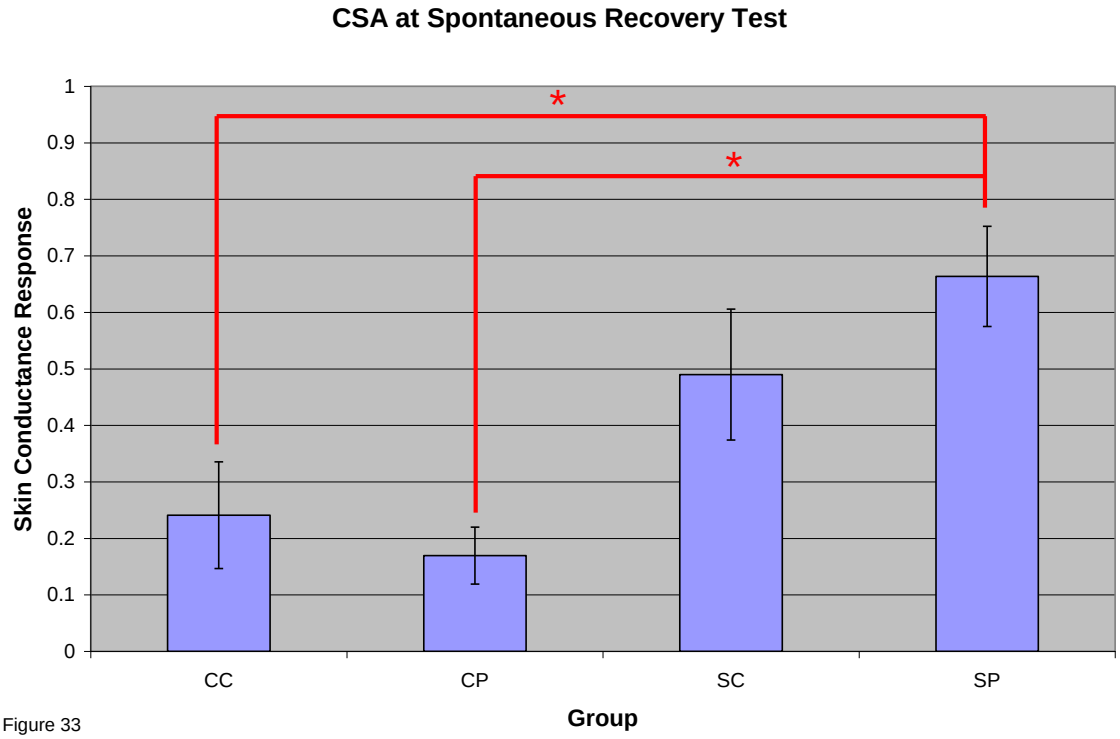


Figure 33

A one-way ANOVA revealed a significant effect of group at the first trial of CS- at one-week follow-up ($F(3,63) = 2.68$, $p = 0.05$; Figure 34). SP versus CP revealed a significant group difference ($t(29) = 2.80$, $p < 0.01$) such that the mean SCR in SP was significantly higher than in CP. There were no significant group differences between SP and SC or between SC and CC. Interestingly, SP versus CC revealed a significant group difference ($t(30) = 2.09$, $p < 0.05$) indicating that SCR in SP was significantly higher than in CC.

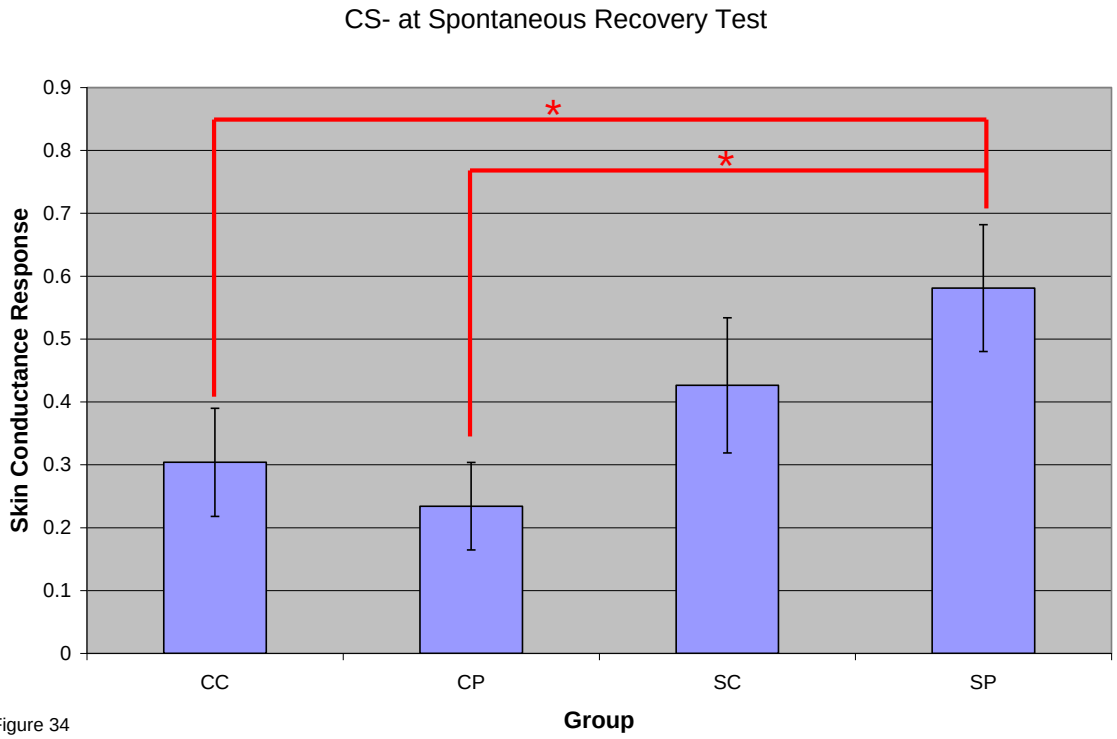


Figure 34

Expectancy Ratings

One-way ANOVAs revealed no significant group effect at the first trial of CSA or at the first trial of CS- at one-week follow-up (Figures 35-36).

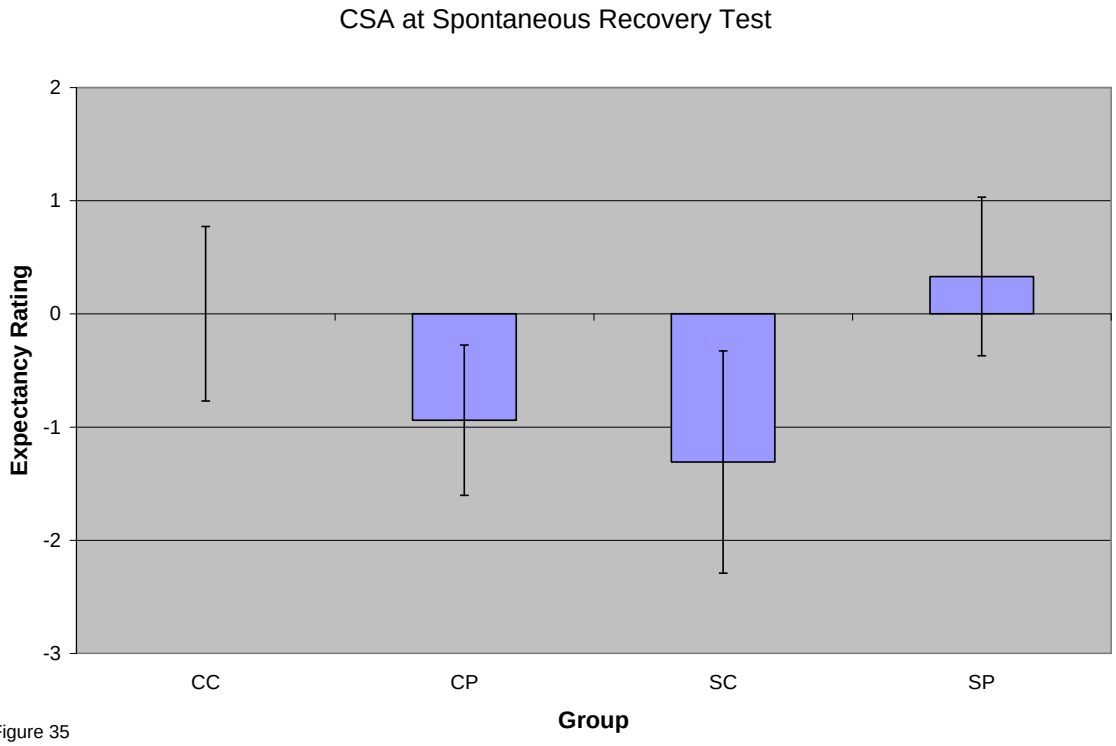


Figure 35

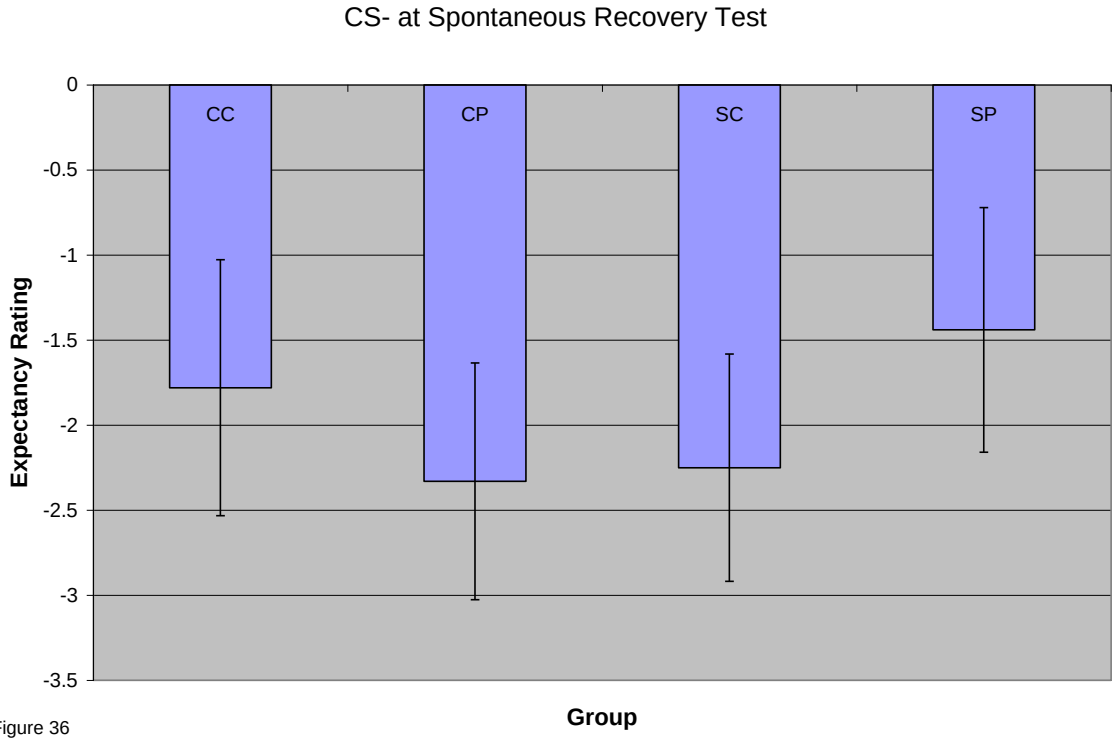


Figure 36

Heart Rate

A one-way ANOVA revealed a significant effect of group at the first trial of CSA at one-week follow-up ($F(3,63) = 2.83$, $p < 0.05$; Figure 37). There were no significant group differences between SP and CP or between SC and CC. SP versus SC was significant ($t(31) = 2.40$, $p < 0.05$) such that mean heart rate in SP was significantly higher than in SC. Interestingly, SP versus CC revealed a significant group difference ($t(32) = 2.50$, $p < 0.05$) indicating that mean heart rate in SP was significantly higher than in CC. For CS-, there were no significant findings.

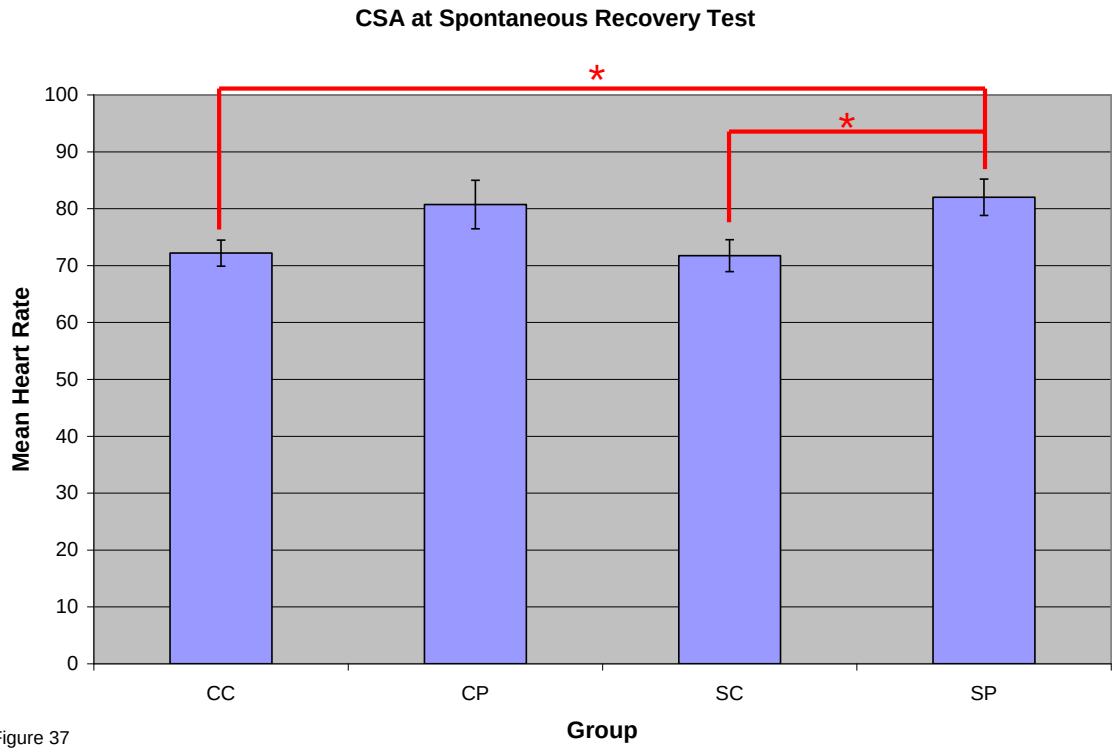


Figure 37

Valence Ratings

A one-way ANOVA revealed a significant effect of group at one-week follow-up ($F(3,65) = 3.32$, $p < 0.05$; Figure 38). SP versus CP was significant ($t(34) = -2.69$, $p = 0.01$) such that CSA was rated significantly more unpleasant in SP than in CP. SP versus

SC was significant (t(31) = -2.79, p < 0.01) such that CSA was rated significantly more unpleasant in SP than in SC. SC versus CC revealed no significant group differences and SP versus CC revealed a trend towards significance (t(34) = -1.93, p = 0.06) such that CSA tended to be rated more unpleasant in SP than in CC. For CSB and CS-, there were no significant findings.

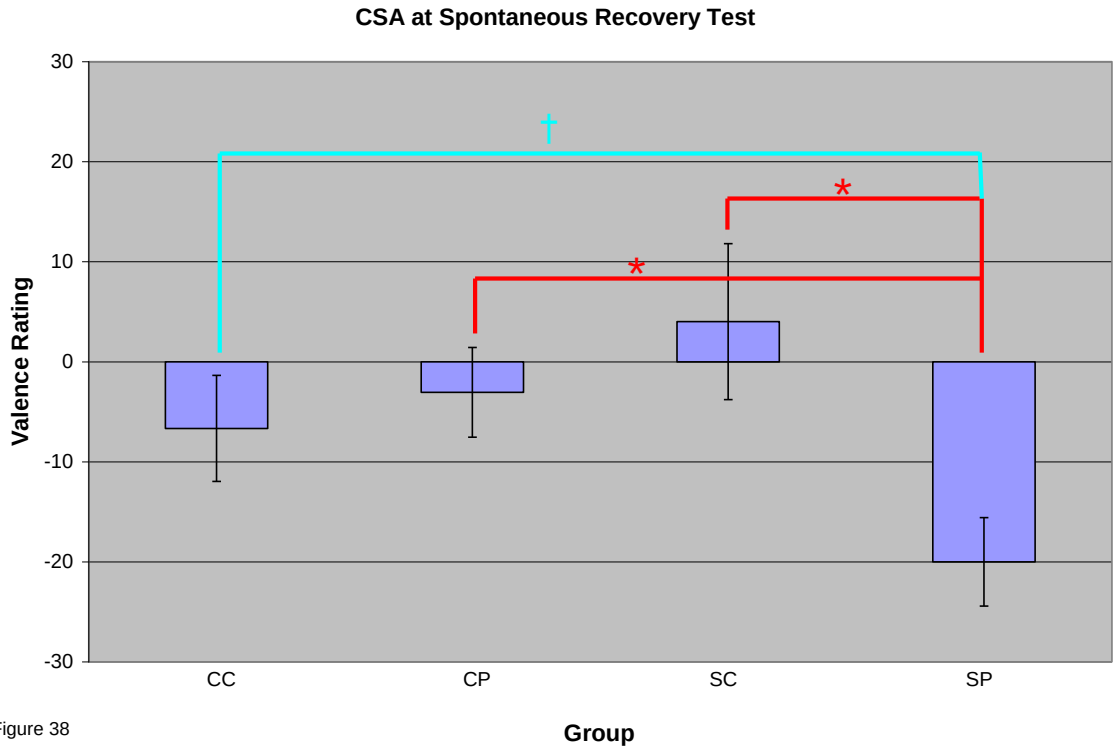


Figure 38

Change from Endpoint of Extinction to Spontaneous Recovery Test

Skin Conductance Response (SCR)

A 4 (Group: SP, CP, SC, CC) x 2 (Time: End of Extinction, Spontaneous Recovery Test) repeated measures ANOVA revealed a significant effect of Time ($F(1,59) = 13.64$, $p < 0.001$, $\eta^2 = 0.19$), Group ($F(3,59) = 3.31$, $p < 0.05$, $\eta^2 = 0.14$), and their interaction ($F(1,59) = 6.01$, $p = 0.001$, $\eta^2 = 0.23$; Figure 39). Tests of simple effects examined this interaction and revealed that participants in the SP group demonstrated a

significant increase in skin conductance responses to CSA from the End of Extinction to the Spontaneous Recovery ($F(1,59) = 27.94$, $p < 0.001$, $\eta^2 = 0.32$) whereas participants in the other three groups demonstrated no significant change in skin conductance responses to CSA from the End of Extinction to the Spontaneous Recovery Test.

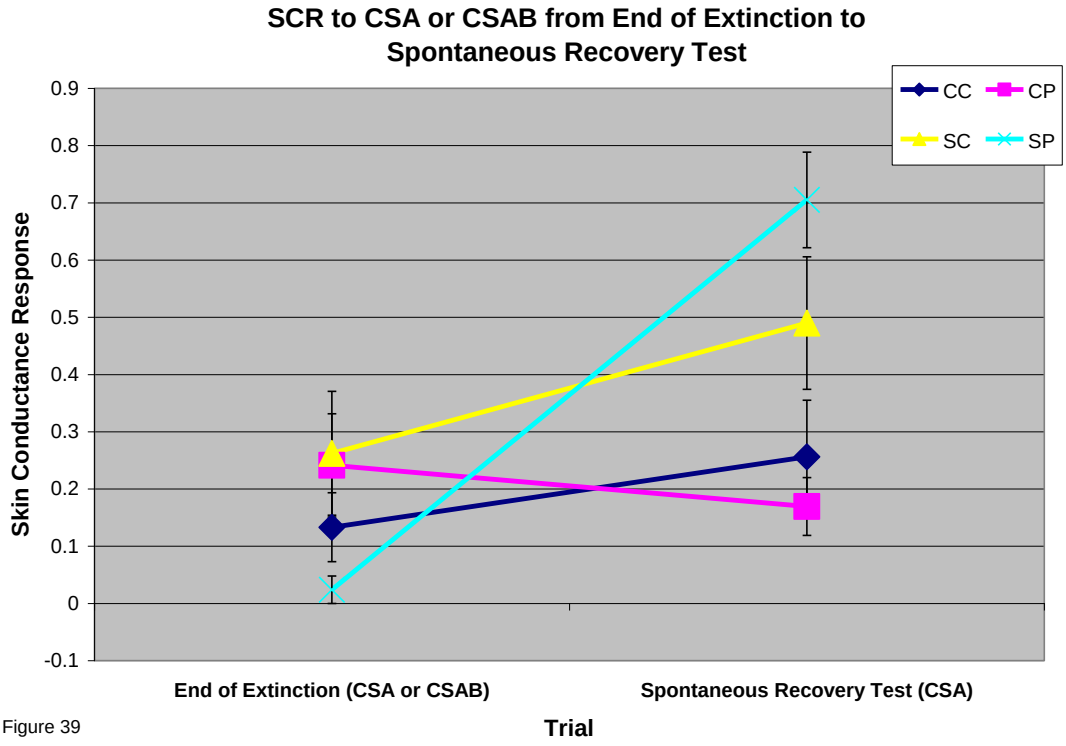


Figure 39

For CS-, A 4 (Group: SP, CP, SC, CC) x 2 (Time: End of Extinction, Spontaneous Recovery Test) repeated measures ANOVA revealed a significant effect of Time ($F(1,59) = 19.69$, $p < 0.001$, $\eta^2 = 0.25$) and the Time x Group interaction ($F(1,59) = 5.49$, $p < 0.005$, $\eta^2 = 0.22$; Figure 40). Tests of simple effects examined this interaction and revealed that participants in the SP group demonstrated a significant increase in skin conductance responses to CS- from the End of Extinction to the Spontaneous Recovery ($F(1,59) = 31.37$, $p < 0.001$, $\eta^2 = 0.35$) whereas participants in the other three groups

demonstrated no significant change in skin conductance responses to CS- from the End of Extinction to the Spontaneous Recovery Test.

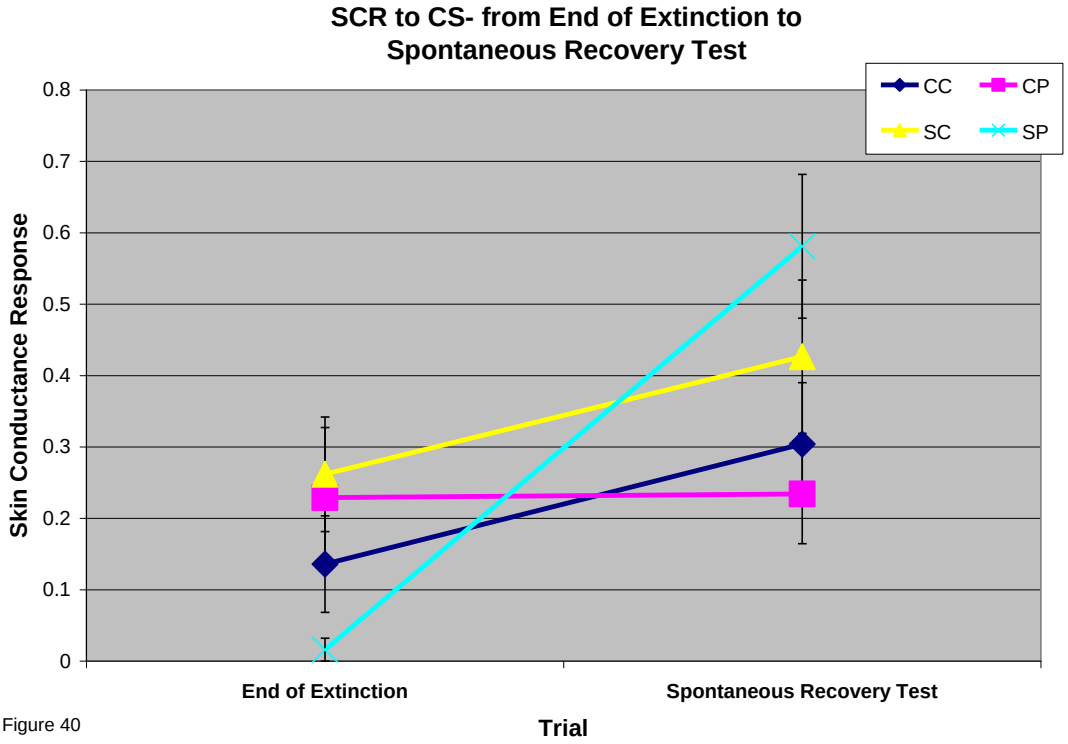


Figure 40

Expectancy Ratings

For CSA, a 4 (Group: SP, CP, SC, CC) x 2 (Time: End of Extinction, Spontaneous Recovery Test) repeated measures ANOVA revealed a significant effect of Time ($F(1,66) = 24.68$, $p < 0.001$, $\eta^2 = 0.27$) indicating that all participants demonstrated a significant increase in US-expectancy ratings to CSA from the end of extinction to follow-up testing one week later (Figure 41).

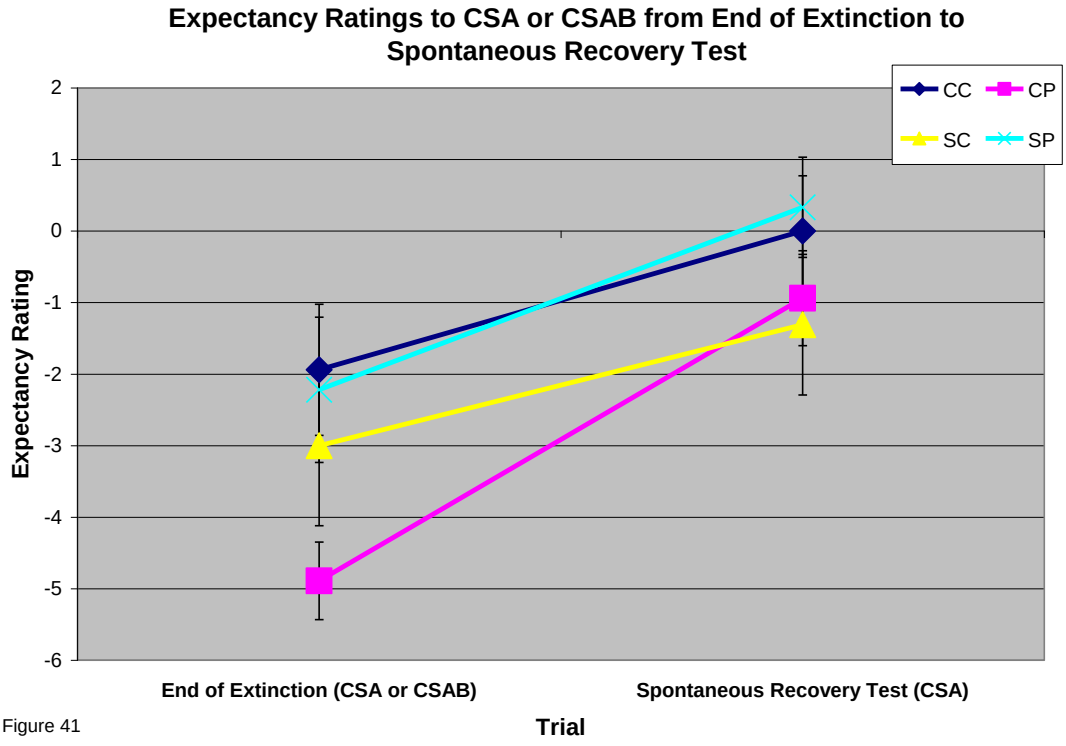


Figure 41

For CS-, a 4 (Group: SP, CP, SC, CC) x 2 (Time: End of Extinction, Spontaneous Recovery Test) repeated measures ANOVA revealed a significant effect of Time ($F(1,66) = 22.65$, $p < 0.001$, $\eta^2 = 0.26$) indicating that all participants demonstrated a significant increase in US-expectancy ratings to CS- from the end of extinction to follow-up testing one week later (Figure 42).

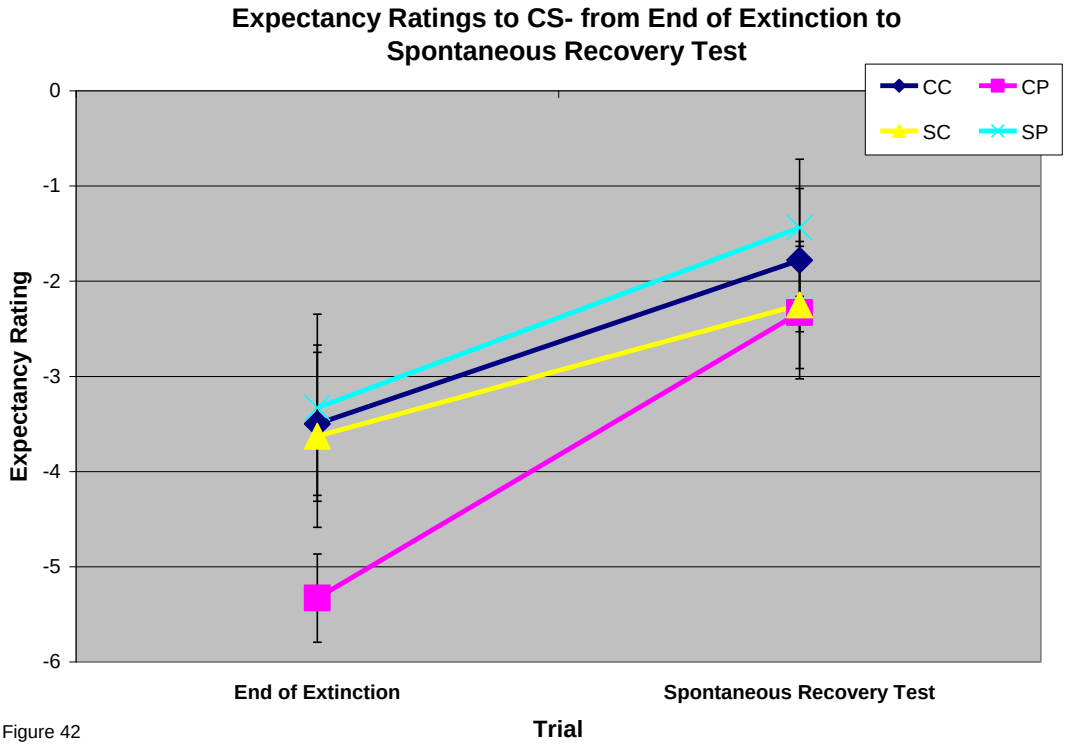


Figure 42

Reinstatement Test

Skin Conductance Response (SCR)

A one-way ANOVA revealed a significant effect of group at the first trial of CSA following reinstatement ($F(3,65) = 3.42$, $p < 0.05$; Figure 43). A series of independent-samples t-tests were conducted to more closely examine these group differences and the only significant difference was between SP and CP ($t(30) = 3.13$, $p < 0.005$) indicating that SCR in SP was higher than in CP.

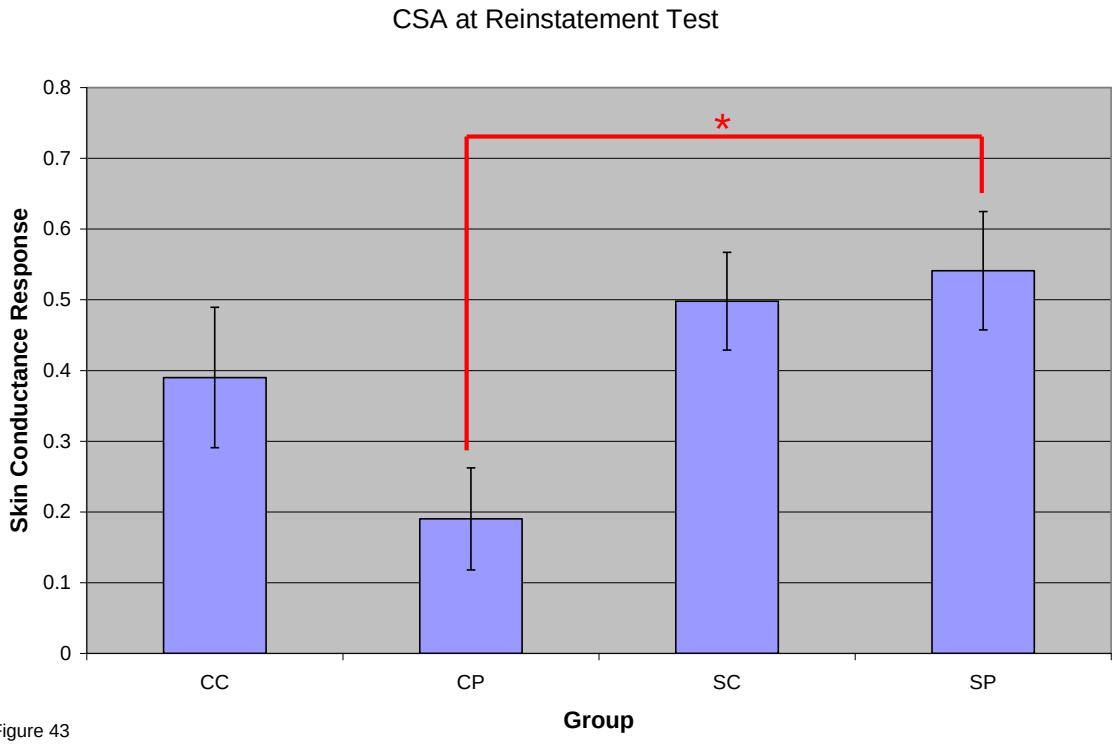


Figure 43

A one-way ANOVA revealed a nonsignificant trend towards a group effect at the first trial of CS- following reinstatement ($F(3,63) = 2.40$, $p = 0.08$; Figure 44). A series of independent-samples t-tests revealed a significant difference between CP and SC ($t(29) = -2.22$, $p < 0.05$) as well as between SP and CP ($t(29) = 2.42$, $p < 0.05$).

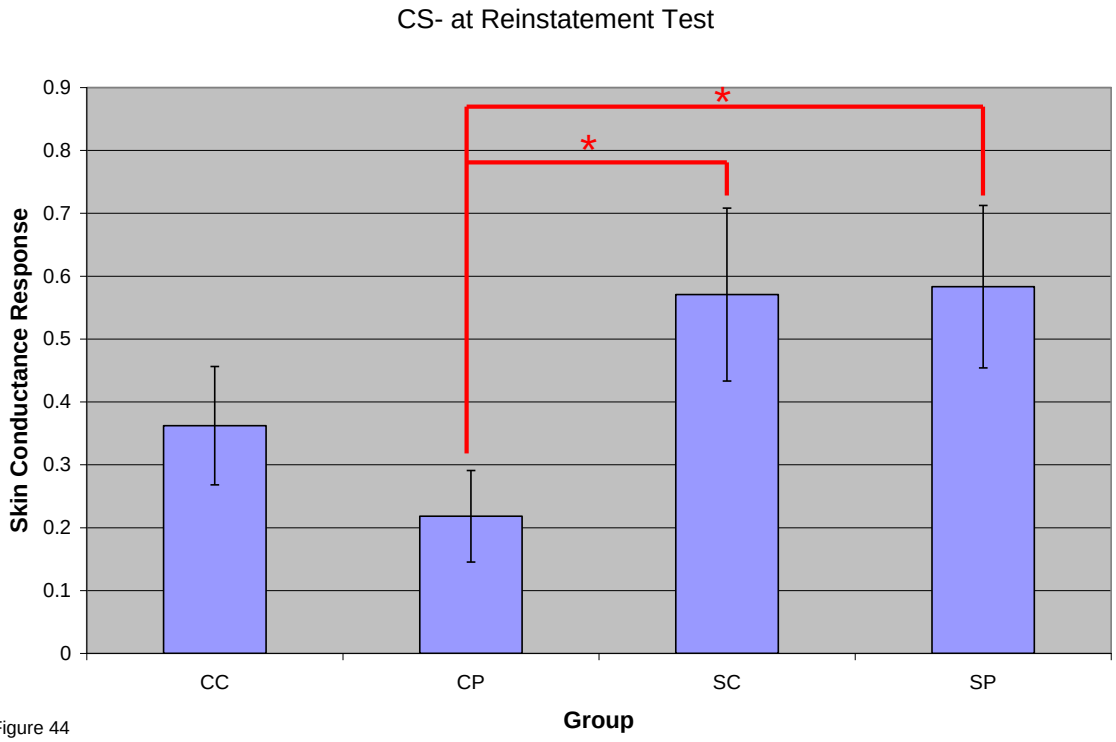


Figure 44

Expectancy Ratings

A one-way ANOVA revealed a significant effect of group at the first trial of CSA following reinstatement ($F(3,69) = 3.51$, $p < 0.05$; Figure 45). SP versus CP revealed a significant group difference ($t(34) = 2.87$, $p < 0.01$) such that the mean expectancy rating in SP was significantly higher than in CP. There were no significant group differences between SP and SC or between SC and CC. Interestingly, CP versus CC revealed a significant effect ($t(34) = -2.89$, $p < 0.01$) indicating that the mean expectancy rating in CC was significantly higher than in CP.

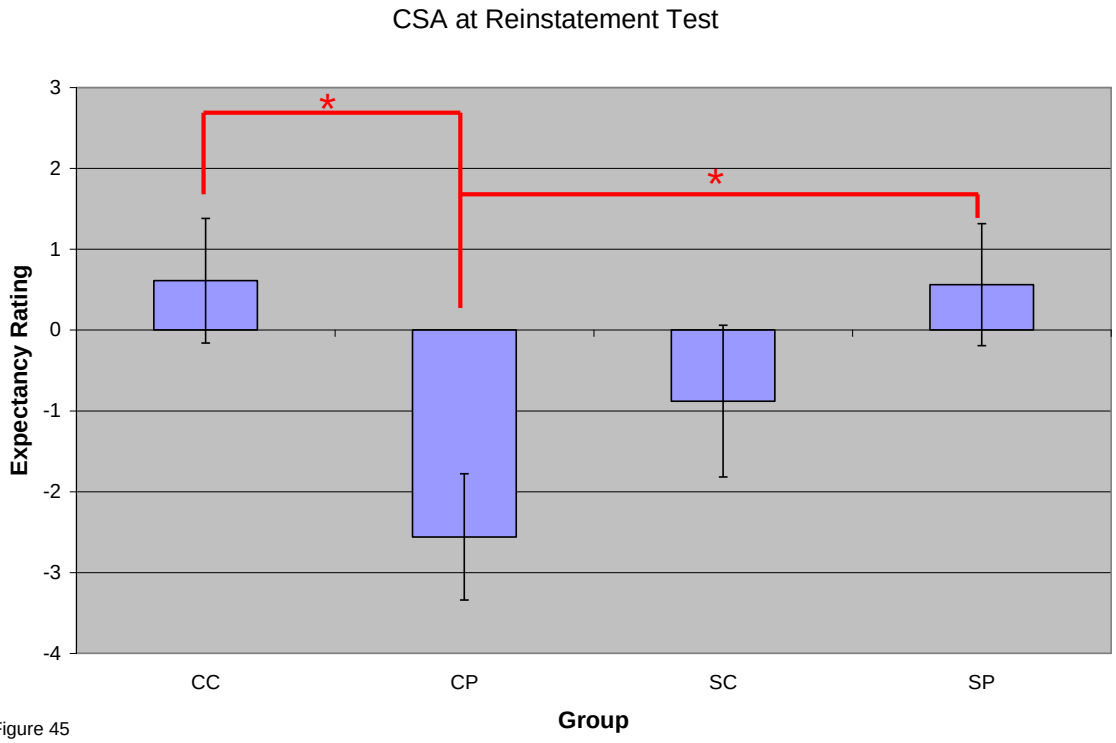


Figure 45

A one-way ANOVA revealed no significant group effect at the first trial of CS-following reinstatement ($F(3,69) = 1.70$, $p = 0.18$; Figure 46). A series of independent sample t-tests revealed a significant group difference between SP and CP ($t(34) = 2.20$, $p < 0.05$) indicating higher expectancy ratings in SP than in CP.

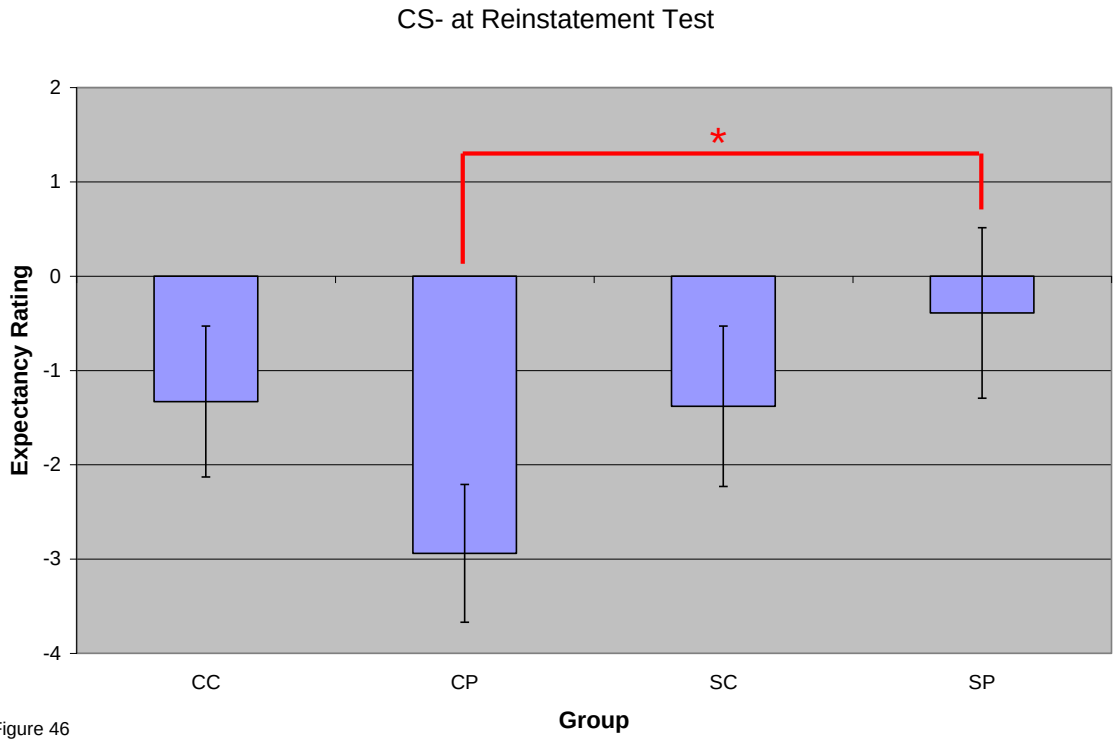


Figure 46

Heart Rate

There were no significant findings for CSA or CS-.

Discussion

Two hypotheses were tested in this study based on the findings of Rescorla (2006): first, it was hypothesized that compound stimulus presentations during extinction would enhance extinction learning and lead to less recovery of conditional fear responding at the spontaneous recovery and reinstatement tests and, second, the mechanism by which compound stimulus trials enhance extinction learning was hypothesized to be enhanced discrepancy between expectation and reality (i.e., enhanced associative change) rather than enhanced conditional responding during extinction. Therefore, ingestion of caffeine was hypothesized to have no impact on extinction learning or recovery of fear. These results largely support the study hypotheses.

Spontaneous Recovery

The impact of single versus compound extinction trials

At the spontaneous recovery test, among those who did not ingest caffeine, participants presented with compound extinction trials (Compound Placebo group) demonstrated significantly lower SCRs to CSA than participants presented with single extinction trials only (Single Placebo group). This is an especially noteworthy finding given that participants in the Compound Placebo group demonstrated significantly higher SCRs to CSA than those in the Single Placebo group throughout the second phase of extinction and at the endpoint of extinction. Thus, even though fear responding remained elevated in the Compound Placebo group during extinction, these participants demonstrated significantly less physiological fear responding at spontaneous recovery than participants in the Single Placebo group. This is also demonstrated in the difference from the end of extinction to the spontaneous recovery test – Single Placebo showed a significant increase whereas Compound Placebo showed no change, indicating that participants in the Single Placebo group demonstrated significant recovery of fear whereas participants in the Compound Placebo group did not. Furthermore, the Compound Caffeine group demonstrated significantly lower SCRs to CSA at spontaneous recovery than the Single Placebo group. Thus, regardless of drug group assignment, participants presented with compound stimulus trials during extinction demonstrated significantly lower skin conductance responses to CSA than the Single Placebo group at the test of spontaneous recovery one week later.

Subjective valence ratings provided further evidence for the effectiveness of compound extinction trials in attenuating fear recovery. Participants in the Compound

Placebo group rated CSA as significantly less aversive at the spontaneous recovery test than did participants in the Single Placebo group. In addition, participants in the Compound Caffeine group also rated CSA as less aversive than did participants in Single Placebo although this trend did not quite reach significance. Thus, both skin conductance responses and subjective valence ratings indicate that compound extinction trials provided protection from spontaneous recovery effects one week following extinction training. This is consistent with the findings reported by Rescorla (2006) and extends those findings to humans.

It is unclear why US-expectancy ratings did not converge with the skin conductance and valence rating results. There is evidence that subjective US-expectancy ratings sometimes diverge from SCRs (McAndrew, Jones, McLaren, & McLaren, 2012). This comes from a body of literature investigating whether Pavlovian conditioning in humans is a unitary system in which conscious awareness of contingencies is necessary to produce a physiological conditional response (e.g., Hinchy, Lovibond, & Ter-Horst, 1995) or whether it is driven by dual systems, one under conscious control and the other utilizing automatic associative processes (McLaren, Green, & Mackintosh, 1994). The former theory attributes a physiological CR to conscious expectancy of the US; however, the latter theory posits that there may sometimes be a dissociation between conscious awareness of contingencies (i.e., US-expectancy) and a physiological CR. There are data demonstrating such dissociations (e.g., McAndrew et al., 2012) supporting the idea that Pavlovian conditioning in humans is a dual-process system with conscious processes governing US-expectancy and automatic associative processes determining at least some of the strength of physiological CRs to CSs. The conscious processes determining US-

expectancy ratings are susceptible to a myriad of factors including cognitive processes which drive the gambler's fallacy, a well-document phenomenon in which people believe that a run of similar events will be broken (Tune, 1964). In the current study, participants may have based US-expectancy ratings at spontaneous recovery on a false belief such as "I know this shape was paired with the sound last time; so, this time, maybe the other shape will be paired with the sound." Thus, additional cognitive processes may have influenced US-expectancy ratings at the spontaneous recovery test.

The impact of caffeine

Interestingly, valence ratings and mean heart rate scores indicate that there may be some benefit to ingesting caffeine prior to extinction training. Valence ratings indicate that participants in the Single Caffeine group rated CSA as significantly less aversive than did participants in the Single Placebo group at spontaneous recovery. Mean heart rate data also supports this finding: participants in the Compound Caffeine and Single Caffeine groups demonstrated significantly lower mean heart rate scores at spontaneous recovery than participants in the Single Placebo group.

These data are somewhat difficult to interpret. Based on previous literature (Cain, Blouin, & Barad, 2004), it was hypothesized in this study that, if caffeine ingestion enhanced extinction learning, the mechanism would be enhanced conditional fear responding during extinction. However, mean heart rate scores during extinction were significantly *lower* in the caffeine groups across extinction than in the placebo groups. These data are actually consistent with several studies which have reported that ingestion of caffeine has no impact on heart rate (e.g., Smith, Brice, Nash, Rich, & Nutt, 2003) or a nonsignificant trend towards lowering heart rate (Flaten, Aasli, & Blumenthal, 2003).

Among individuals who regularly consume moderate amounts of caffeine (as is true of the current sample), ingestion of caffeine has been found to significantly decrease resting heart rate and does not predict elevated heart rate in response to stress (Lane & Williams, 1987). Thus, heart rate may not be a good measure of arousal in the context of caffeine ingestion. In contrast, skin conductance responses do provide support that caffeine elevated arousal and conditional fear responding throughout extinction training, which is consistent with previous studies demonstrating caffeine increases skin conductance levels (Barry et al., 2005). If skin conductance data is a better measure of arousal in the context of caffeine ingestion than heart rate data, these results indicate that enhanced responding alone during extinction training may provide some protection against spontaneous recovery effects.

Summary of Spontaneous Recovery Results

Results of the skin conductance responses and valence ratings indicate that compound extinction trials predict significantly lower fear responding at the spontaneous recovery test (in both cases, the Compound Caffeine and Compound Placebo groups demonstrated lower fear responding than the Single Placebo group). Mean heart rate scores, on the other hand, indicate that caffeine ingestion predicts significantly lower fear responding at spontaneous recovery (Compound Caffeine and Single Caffeine demonstrated lower fear responding than Single Placebo) and the valence ratings partially support this (Single Caffeine demonstrated lower fear responding than Single Placebo). Thus, presentation of compound trials during extinction seems to protect from spontaneous recovery effects and ingestion of caffeine prior to extinction training may also do so.

As discussed by Rescorla (2006), compound extinction trials may enhance extinction learning by two possible mechanisms – enhanced associative change or enhanced responding. Enhanced associative change refers to the idea that, when two CSs are presented simultaneously, expectancy of a negative outcome (and therefore conditional fear responding) will increase. In fact, this has been shown to occur even when each CS has undergone some extinction training individually prior to being paired together (Reberg, 1972) As predicted by the Rescorla-Wagner model (Rescorla & Wagner, 1972), increasing US-expectancy during extinction training and then violating that expectation will enhance extinction learning. The current results provide support for this theory. As measured by skin conductance responses and expectancy ratings, when two previously extinguished stimuli were presented simultaneously for the first time during the second phase of extinction training, participants demonstrated a significant increase in fear responding (i.e., the Reberg Effect). Thus, it seems likely that the Compound Placebo and Compound Caffeine groups demonstrated significantly less fear responding at spontaneous recovery as a result of enhanced associative change during extinction training (i.e., learning of the CS-no US relationship was optimized in these groups). However, the Single Caffeine group also showed some protection from spontaneous recovery effects. This would indicate that increased responding alone also enhanced extinction learning. Therefore, the results of the spontaneous recovery test provide evidence for the effectiveness of compound trials but the mechanism remains unclear. If enhanced associative change is the mechanism by which compound extinction trials enhance learning, the finding that the Single Caffeine group demonstrated less fear responding cannot be explained. If enhanced responding alone is the mechanism, the

finding that the Compound Placebo group demonstrated less fear responding cannot be explained. Thus, results of the spontaneous recovery test implicate both mechanisms.

Reinstatement

At the reinstatement test, among those who did not ingest caffeine, participants presented with compound extinction trials demonstrated significantly less fear responding to CSA (as measured by skin conductance responses and US-expectancy ratings) than participants who experienced single extinction trials only. Among those who ingested caffeine, compound versus single extinction trials did not predict significant differences at the reinstatement test. Interestingly, participants in the Compound Placebo group demonstrated significantly less fear at reinstatement than participants in the Compound Caffeine group, as measured by US-expectancy ratings. Taken together, these findings indicate that compound trials during extinction protect against reinstatement effects. Ingestion of caffeine provided no benefit at reinstatement. Perhaps this is due to the prediction that compound trials lead to greater inhibitory learning during extinction (as posited by the Rescorla-Wagner model and supported by the current findings).

One possible mechanism underlying reinstatement is that presentation of the US leads to an excitatory relationship between the US and the context (Fanselow, 1980; Vansteenwegen et al., 2008). Thus, on the next CS trial, prediction of the US (and level of conditional fear responding) will be determined by the predictive value of the CS plus the predictive value of the context. The context now has excitatory predictive value as a result of the Context-US presentation. If compound extinction trials enhanced inhibitory CS-US learning during extinction, the sum predictive value will be lower in the compound groups than in the single extinction groups.

Fear Responding to CS-

Group differences in fear responding to CS- at spontaneous recovery and reinstatement were an unexpected and interesting finding in this study. At spontaneous recovery, SCRs to CS- were significantly lower in both compound extinction groups (Compound Caffeine and Compound Placebo) than in group Single Placebo. At reinstatement, SCRs to CS- were significantly lower in group Compound Placebo than in both of the single extinction groups (Single Caffeine and Single Placebo); furthermore, group Compound Placebo demonstrated significantly lower US-expectancy to CS- than group Single Placebo. Taken together, these findings seem to indicate that compound extinction trials predict lower fear responding to safety signals at one-week follow-up testing. One possible explanation for these findings involves predictions of the Rescorla-Wagner model that each CS is comprised of several elements and there can be shared common elements between the stimuli. Thus, when two of the shape-CSs were paired with the US, common elements shared by those two shapes with the third CS also became associated with the US eliciting some fear responding to the CS-. The current results do indicate that participants demonstrated some fear responding to the CS- (although much greater responding to the CS+ shapes). During extinction, participants in the single extinction groups did not receive the same number of CSB trials as participants in the compound extinction groups. This was due to logistical constraints since it was not possible to equate number of extinction trials, spacing of extinction trials, and stimuli presented between the compound and single groups. Thus, participants in the compound extinction groups received twice as many CSB presentations; therefore, any common shared elements between CSB and CS- underwent twice as many extinction trials in the

compound groups than in the single extinction groups which may explain the differences in fear responding to CS- at test.

Conclusion

Overall, these findings support the prediction that compound stimulus presentations during extinction enhance learning during extinction and provide protection from spontaneous recovery and reinstatement effects. Based on these results, the mechanism by which compound trials enhance extinction learning seems to be enhanced associative change rather than enhanced responding alone. Although caffeine ingestion prior to extinction training did provide some protection against spontaneous recovery effects, this was limited to mean heart rate scores and subjective valence ratings. In addition, caffeine ingestion provided no attenuation of reinstatement effects (whereas compound extinction trials did). Thus, it appears that compound extinction trials enhance extinction learning by increasing expectancy of an aversive outcome which maximizes the discrepancy between expectation and reality driving learning as predicted by the Rescorla-Wagner model.

It is interesting that the current study yielded different results from a previous study of compound extinction in humans in which extinction learning was completely blocked when participants were presented with compound extinction trials (Vervliet, Vansteenwegen, Hermans, & Eelen, 2007). The key difference between these two studies is that, in the current study, participants were first presented with some single extinction trials of each of the two CS+ shapes before the two shapes were paired together for compound trials. It seems likely that participants in the study by Vervliet and colleagues processed the compound trials during extinction as a new stimulus as

predicted by configural models of learning (Pearce, 1987). Rather than responding to the compound trials as a collection of two parts, they perceived it as one new stimulus. This would explain the blockage of extinction effects reported. However, in the current study, presentation of some single extinction trials prior to compound trials allowed participants to process the CSs elementally. Thus, when the two shapes were paired together, participants perceived the compound trials as a collection of two CSs rather than as a new stimulus. In fact, this is consistent with what participants reported; at the conclusion of the study, participants in the two compound extinction groups were asked about their experience of the compound trials and all of them stated that they experienced the trials as two of the old shapes appearing together (i.e., that they processed the compound trials elementally). Therefore, presenting fearful stimuli individually first prior to pairing them together seems imperative to activating elemental processing of the fearful stimuli thereby increasing expectancy of an aversive outcome; violation of this increased expectancy seems to be the mechanism by which compound trials can enhance extinction learning and provide protection against recovery of the fear response.

Limitations

This study had several limitations including that BIS scores were not as elevated as is likely in the population of individuals with anxiety disorders. Thus, the results may not be as generalizable to those with a vulnerability to develop anxiety disorders. However, BIS scores were still slightly elevated above the mean reported in nonanxious samples and the participants in this sample demonstrated significant fear conditioning indicating that the results of extinction training should be relevant in enhancing the effectiveness of exposure therapy. Also, the US used in the study may not be as aversive

and therefore not as generalizable to the unconditional stimuli in the context of anxiety disorders. However, participants demonstrated significant physiological fear responding to the US indicating that it was sufficiently aversive. Lastly, it could be argued that caffeine may not have been sufficiently arousing since it increased skin conductance but not heart rate means. Based on previous literature, the impact of caffeine on heart rate is mixed and there is evidence that caffeine ingestion sometimes decreases heart rate; however, it is true that the effects of caffeine on physiological arousal in this study were not universal since we did not find effects on both skin conductance and heart rate.

Clinical Implications

The results of the present study indicate that the effects of exposure treatments for anxiety disorders may be enhanced if individuals are first exposed to one fear-provoking stimulus at a time and then exposed to two fear-provoking stimuli in compound. For example, in the treatment of panic disorder, individuals are often taken through exercises which involve exposure to internal cues of panic attacks, then exercises which involve exposure to external cues of panic attacks, and then both simultaneously. The current investigation provides a rationale for the effectiveness of such a paradigm. This can be done in the treatment of other anxiety disorders as well. In exposure treatment for post-traumatic stress disorder, for instance, individuals complete exercises which involve exposure to the trauma memory. Often this involves writing a detailed account of the trauma. In addition to the trauma memory, fear-provoking stimuli for individuals with PTSD may include loud noises (e.g., the sound of fireworks for a combat veteran returning from active duty). Exposure therapy in this case could involve writing exposures to the trauma, exposure to the sound of fireworks, and then compound

exposure could entail writing about the trauma while simultaneously listening to the sound of fireworks. Thus, the results of the current study have significant clinical implications. It will be important for future studies to replicate the findings reported here and to also investigate whether compound stimulus presentations during exposure enhances treatment effectiveness in clinical samples.

STUDY 2

OCCASIONAL REINFORCED TRIALS DURING EXTINCTION ATTENUATES SPONTANEOUS RECOVERY AND RAPID REACQUISITION

Introduction

The behavioral treatment most commonly used in the treatment of specific fears and phobias involves systematic exposure to fear-provoking stimuli. Typically, individuals are encouraged to gradually expose themselves for prolonged periods to the feared stimulus so that fear may extinguish over time. Despite being an effective treatment (e.g., Norton & Price, 2007), there are circumstances which re-elicit fear following treatment. One circumstance especially problematic for successful therapy is rapid reacquisition (Kehoe & Macrae, 1997; Napier, Macrae, & Kehoe, 1992; Ricker & Bouton, 1996). In other words, sometimes fear responding to the conditional stimulus (CS) can be extinguished during exposure only to return rapidly if the CS and US occur together again. For example, an individual with social anxiety disorder may successfully extinguish fear responding (the conditional response or CR) in social situations (CS) only to have that fear response return quickly after just one subsequent pairing of a social situation with a negative outcome (e.g., rejection or negative evaluation, the unconditional stimulus or US). Rapid reacquisition is particularly likely in social anxiety given the relatively common occurrence of negative social outcomes.

The rapid reacquisition phenomenon provides further evidence that extinction is not unlearning of the previously learned CS-US association. Instead of erasing the CS-

US memory, extinction is hypothesized to involve new inhibitory learning ("CS-no US") that then competes with the CS-US memory (e.g., Bouton, 1993). Retrievability of the CS-US memory may explain return of fear following exposure therapy. In the case of rapid reacquisition, the first CS-US pairing following extinction may trigger retrieval of the CS-US memory and interfere with retrieval of the CS-no US memory, thereby facilitating rapid reacquisition. Clearly, this presents a problem for the successful treatment of phobias and other anxiety disorders. Thus, investigations aimed at finding methods for attenuating rapid reacquisition are warranted.

Although counterintuitive, a potential method for attenuating rapid reacquisition may be to present occasional CS-US pairings during extinction training. This has been effective in animal studies using Pavlovian conditioning (Bouton, Woods, & Pineno, 2004) and operant conditioning (Woods & Bouton, 2007) paradigms. Using an appetitive conditioning procedure, Bouton and colleagues (2004) examined the rate of reacquisition after typical extinction procedures (multiple nonreinforced presentations of the CS) or partial reinforcement procedures during extinction (in which some CS presentations were paired with the US). Occasional reinforced trials during extinction slowed the rate of reacquisition, even though this partial reinforcement procedure also slowed the loss of conditional responding during extinction training. Similarly, Woods and Bouton (2007) examined reacquisition of an operant conditioning response after typical extinction procedures (nonreinforced CS presentations) versus occasional reinforced responses during extinction. Across three experiments, reacquisition was significantly slower after extinction that included occasional reinforced trials.

One potential mechanism by which occasional reinforced trials during extinction can slow the rate of reacquisition is by connecting CS-US pairings with the extinction context rather than solely with the acquisition context (e.g., Bouton et al., 2004). Specifically, Bouton and colleagues (2004) suggest that, during acquisition, reinforced trials become associated with other reinforced trials. During extinction training, nonreinforced trials become associated with other nonreinforced trials. Thus, following extinction training, if the animal or human is subsequently faced with a reinforced trial (i.e., the CS and US occur together again), this will trigger memories of acquisition training and lead to the prediction that the next CS presentation will also be reinforced. In contrast, if some CS-US pairings occur during extinction, this allows for reinforced trials to be associated with *non*reinforced trials (during extinction) and reinforced trials (during acquisition). Thus, when faced with a CS-US pairing following extinction training, the organism will be less likely to expect the next CS presentation to predict the US because CS-US pairings have been associated with both further CS-US pairings (in acquisition) *and* CS-no US pairings (during extinction). This ambiguity will slow the rate of reacquisition. Another mechanism for the effectiveness of occasional reinforced trials during extinction derives from the Rescorla-Wagner model of learning (Rescorla & Wagner, 1972). According to this model, learning at each trial is directly proportional to the degree to which the organism is surprised. Surprise is operationalized as the discrepancy between expectation and reality. Thus, according to the Rescorla-Wagner model, learning is optimal during the first few trials of extinction since the animal or human expects an aversive outcome (i.e., the US) when presented with the CS but the US does not occur, thereby maximizing the surprise factor. However, after several trials of

nonreinforced CS presentations, the animal or human is no longer surprised by the outcome – expectation of an aversive outcome has greatly diminished, thereby diminishing the discrepancy between expectation and reality. According to the Rescorla-Wagner model, one way to maximize learning during extinction, and thereby attenuate effects such as rapid reacquisition, is to enhance the surprise factor. A potential method for doing so is to present some CS-US pairings during extinction training. Following a few extinction trials, if the CS is again paired with the US, expectation of the US should be elevated at the subsequent CS trial, thereby enhancing surprise when the US does not occur. Thus, according to the Rescorla-Wagner model, occasional reinforced trials during extinction may enhance learning and provide protection from future fear recovery effects.

The effectiveness of occasional reinforced trials in enhancing extinction learning has never been tested in humans; thus, the current investigation aimed to do so. Following fear conditioning procedures, participants were randomly assigned to undergo typical extinction procedures during which all CS presentations were nonreinforced (i.e., never paired with the US) versus extinction procedures utilizing a partial reinforcement schedule during which some CS presentations were paired with the US. The schedule of partial reinforcement in the current study was determined by the findings of Bouton and colleagues (2004). Two partial reinforcement schedules were tested in that experiment, a 1:8 schedule in which one out of every eight trials were reinforced during extinction and a 2:8 schedule in which two out of every eight trials were reinforced during extinction. Results indicated that the 2:8 schedule led to greater reduction in the rate of reacquisition. Thus, a 2:8 schedule was chosen in the current design. It was hypothesized that

participants in the partial reinforcement group would demonstrate higher levels of fear responding (as measured by skin conductance responses and subjective US-expectancy ratings) throughout extinction than the control group but significantly less fear responding at the spontaneous recovery test and significantly slower rate of reacquisition.

Method

Design

Participants were randomly assigned to one of two experimental groups: Partial Reinforcement [R; n = 11] or Control [C; n = 12] indicating whether they received some or no reinforced trials during extinction.

Participants

Twenty-three participants (18 female, 5 male), with a mean age of 19.2 (range 18 to 22), were recruited through mass testing sessions from several Introduction to Psychology classes. Participants were from a variety of ethnic backgrounds: 35.3% Asian, 35.3% Caucasian, 17.6% Latino, 5.9% Biracial, 5.9% Indian. Participants were recruited if they scored in the top quartile of scores on the Behavioral Inhibition Scale (BIS; Carver & White, 1994). Exclusion criteria for study participation included 1) any heart, respiratory, or neurological problems, 2) current or a history of seizures, and 3) pregnancy.

Measures

Self Report Questionnaires

Two self report measures were completed at Baseline to assess group differences: the Behavioral Inhibition Scale (BIS; Carver & White, 1994) and the Beck Depression

Inventory (BDI; Beck, Steer, & Brown 1996). The BIS, a 7-item scale, was developed to measure individual differences in sensitivity to one of two general motivational systems which are thought to underlie behavior: a behavioral approach system (BAS) is believed to regulate appetitive motives in which the goal is to move toward something desired while a behavioral avoidance or inhibition system (BIS) is believed to regulate aversive motives in which the goal is to move away from something unpleasant (Carver & White, 1994). The BIS has demonstrated good internal consistency, $\alpha = 0.74$. In a sample of 732 undergraduate students, the mean BIS score was 19.99 with a standard deviation of 3.79.

The Beck Depression Inventory is a widely used screening instrument for depression. It is a 21-item measure where each item is rated on a 4-point Likert-type scale ranging from 0 to 3 with higher scores indicating higher levels of depression. In a sample of 229 young adults recruited from undergraduate courses, the mean score on the BDI was 9.12 with a standard deviation of 8.32 (Segal, Coolidge, Cahill, & O'Riley, 2008). In this sample, the BDI also demonstrated good internal reliability ($\alpha = 0.92$).

Subjective Measures

Across all phases of the experiment (acquisition, extinction, and test), participants were asked to rate their expectancy of the US during CS presentations. They were instructed to immediately record US-expectancy at the onset of each CS presentation. This expectancy was rated on a scale between -6 = "certain no noise" to +6 = "certain noise" with a midpoint of 0 = uncertain.

Participants reported valence ratings for each CS on a scale of -50 = "very unpleasant" to +50 = "very pleasant" with a midpoint of 0 = "neutral." They rated

valence immediately following acquisition, immediately following extinction, at the start of Day 2 (before the spontaneous recovery test), and at the end of Day 2 (following the spontaneous recovery and rapid reacquisition tests).

Physiological Measures

Heart rate (HR) and skin conductance responses (SCRs) were measured using a Biopac MP150 unit running Acqknowledge 4.0 software (Biopac Systems, Inc., Goleta, CA) with one GSR 100C amplifier and one electrocardiogram (ECG) 100C amplifier attached. The GSR amplifier was set to direct current and had a sensitivity of 5 μohm/V, with a 1.0-Hz low-pass filter. Skin conductance responses (SCRs) were measured at the onset of each CS. The ECG amplifier gain was set at 1,000, the R-wave detector was switched on, and the filter was switched off. Data were acquired at 200 samples per second, providing a temporal resolution of 5 ms.

To measure SCRs, two disposable 1 cm diameter AG-AgCl electrodes were placed on the distal phalanx of the index and middle fingers of the non-dominant hand. The magnitude of SCRs was calculated as the difference between the maximum skin conductance level (measured in microsiemens) within 1–6 seconds following CS onset and the mean skin conductance level within the 2-second period prior to CS onset. Amplitudes were range corrected using the largest response elicited by the US for each individual participant. To do this, for each participant, SCRs to each US presentation were calculated as the difference between the maximum skin conductance level within 1-6 seconds following US-onset and the mean skin conductance level within the 2-second period prior to CS-onset (the CS which occurred immediately prior to that particular US presentation). For each participant, all SCRs to CSs were divided by that person's

maximum SCR to the US. These range-corrected responses were then subjected to a square root transformation in order to normalize the distribution prior to statistical analysis. SCRs were rejected for a given CS presentation if behavioral observations indicated movement or behavior such as coughing and sneezing. 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.

To monitor HR, two disposable 1 cm Ag-AgCl ECG electrodes were placed crossing the chest – one 1 inch below the collar bone and one 1 inch above the last rib bone. The former was placed on the same side of the chest as the participant's non-dominant hand (i.e., the hand on which the SCR electrodes were placed) while the latter was placed on the side of the chest of the participant's dominant hand. These two electrodes served as the positive and negative electrodes while one of the SCR electrodes served as the ground to triangulate the ECG signal.

Apparatus and Stimuli

Two neutral faces from the NimStim set of facial expressions (Tottenham, et al., 2009) were used as the conditional stimuli (CSs); one was paired with the US during acquisition training while the other was not. In order to make the facial stimuli as relatable as possible to the majority of research participants at UCLA, the faces were of Asian females. Which of the two faces served as the CS+ versus the CS- was counterbalanced across participants. The unconditional stimulus (US) was a 1-second scream presented binaurally through headphones at 82 decibels. Such auditory stimuli have successfully served as USs in previous studies (e.g., Lau et al., 2008). In fact, auditory USs have been found to demonstrate equivalent or superior conditioning effects

as shock USs (as measured by US-expectancy ratings and skin conductance responses) without the risk of causing pain or excessive anxiety (Neumann & Waters, 2006). Across different auditory US stimuli, a scream-US has been found to produce more robust conditioning effects than a white noise-US (Joos, Vansteenwegen, & Hermans, 2012); thus a scream-US was utilized in the current study. Stimulus delivery was controlled by one computer which presented participants with the CSs and US through E-prime software (Psychology Software Tools, Inc., Pittsburgh, PA, USA). Physiological data acquisition was controlled by a second computer using Acqknowledge software (Biopac Systems, Inc., Goleta, CA, USA).

Procedure

The experiment consisted of several phases that were completed over the course of two days. 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 the BIS and BDI.

Electrodes were placed on participants for recording HR and SCR. Next, psychophysiological measures were recorded for a five-minute resting period during which participants were instructed to "please sit quietly and remain still" and left alone in the room (the researcher monitored physiological data acquisition in a room adjacent to the experimental room; the two rooms were interconnected by a door that remained partially open to allow for communication between the researcher and the participant if necessary). Following the baseline, participants were seated 3 feet in front of a 21" computer monitor placed at eye level which was used to display the CSs. They were asked to put on headphones and told that they may sometimes hear a loud noise through

the headphones. Next participants were trained on the US-expectancy scale and asked to rate their expectancy of hearing the sound as soon as each face appeared on the screen (participants placed their dominant hand on the desk on top of an expectancy ratings sheet and held a pen so they could record their ratings with minimal movement). Participants were instructed to return their attention immediately to the computer screen following each US-expectancy rating. The experimenter then left the room (throughout all experimental phases of the study, the researcher monitored physiological data acquisition from the interconnected room).

On the first day, participants first underwent a *habituation* phase in which they were presented with four 8-second presentations of each CS; the inter-trial interval (ITI) varied across 20, 25, and 30 seconds (mean = 25 seconds). The CSs were presented in random order with the caveat that no more than two trials of each CS were presented sequentially. Immediately following habituation, participants underwent an *acquisition* phase in which they received eight 8-second presentations of each CS using the same randomization procedure and mean ITI. During the last second of each CS+ presentation, the scream-US was presented through the headphones; the CS- was never paired with the US. The acquisition phase was designed to teach participants to associate the CS+ with the aversive scream so that it would elicit a conditional response of fear when presented alone.

Following acquisition, participants were instructed to remove the headphones. They then provided subjective valence ratings for each CS. Participants then put the headphones back on and were asked to continue rating their expectancy of hearing the sound as soon as each face appeared on the screen. Another 5-minute baseline was

recorded prior to commencement of extinction training. During *extinction*, all participants received twenty-four 8-second presentations of the CS+ and twenty-four 8 second-presentations of the CS- (with ITIs ranging across 25, 30, and 35 seconds). For participants in the C group, there were no US presentations during extinction. For participants in the R group, two out of every eight CS+ trials were reinforced (i.e., the US was presented during the last 1-second of the CS presentation). This 2:8 reinforced schedule was chosen given its effectiveness in slowing the rate of rapid reacquisition in rats (Bouton, Woods, & Pineno, 2004). To distribute the reinforced CS+ presentations through extinction, the session was divided into three blocks of eight trials, each of which contained two reinforced trials. Within each block, the reinforced trials occurred either during Trials 2 & 6, 3 and 7, or 4 & 8; two reinforced trials never occurred consecutively. Across both extinction phases, CSs were presented in random order with the caveat that no more than two trials of each CS were presented sequentially. Following extinction training, participants again provided subjective valence ratings for each CS.

Group	Habituation	Conditioning	Extinction
Control	CS+ (4) CS- (4)	CS+ + US (8) CS- (8)	CS+ (24) CS- (24)
Reinforced	CS+ (4) CS- (4)	CS+ + US (8) CS- (8)	CS+ (24)* CS- (24)

*2:8 Partial Reinforcement Schedule, 6 total CS+ + US pairings

On Day 2 one week later, all participants underwent testing for spontaneous recovery and rapid reacquisition. Upon arriving in the laboratory, participants provided subjective valence ratings for each CS. The electrodes were placed just as during Day 1 for psychophysiological recording and they sat in front of the same monitor wearing the same headphones. They were re-familiarized with the expectancy scale and asked to

again record their expectancy of hearing the noise at the onset of each CS presentation. During the *spontaneous recovery* phase, participants were presented with four 8-second presentations of each CS and no US presentations. Immediately following this was the *rapid reacquisition* phase during which participants were presented with four 8-second presentations of the CS+ (all paired with the US) and four 8-second presentations of the CS-. Lastly, immediately following this test of rapid reacquisition, was the *test* phase during which participants were presented with four 8-second presentations of each CS and no US presentations. Throughout these three phases, the CSs were presented in random order with the caveat that no more than two trials of each CS were presented sequentially and with ITIs ranging across 20, 25, and 30 seconds. Upon completion of this phase, participants again provided subjective valence ratings for each CS.

Group	Spontaneous Recovery Test	Reacquisition Test	Test
Control	CS+ (4) CS- (4)	CS+ + US (4) CS- (4)	CSA (4) CS- (4)
Reinforced	CS+ (4) CS- (4)	CS+ + US (4) CS- (4)	CSA (4) CS- (4)

Results

For results of HLM analyses across Acquisition, Extinction, Spontaneous Recovery, Rapid Reacquisition, and Test phases, only significant findings are reported. Statistical values (i.e., F-values, t-ratios, and p-values) for all nonsignificant findings are reported in Appendix B.

Baseline

The mean BIS score in this sample was 23.35 and the mean BDI score was 6.82. Independent samples t-tests revealed no significant differences between the two groups

on the BIS ($t(21) = 0.34$, $p = 0.74$) or the BDI ($t(21) = 1.43$, $p = 0.17$). There were also no significant differences among the groups on age ($t(21) = 1.39$, $p = 0.19$), gender ($\chi^2(1, N = 23) = 1.02$ ($p = 0.31$)), or ethnicity ($\chi^2(4, N = 23) = 2.95$ ($p = 0.57$)).

Acquisition

Skin Conductance Response (SCR)

HLM analyses were conducted for SCRs to each conditional stimulus across the eight acquisition trials. For CS+, the intercept (i.e., the SCR at the first acquisition trial) was significantly different from zero ($b = 0.19$, $t(21) = 3.57$, $p < 0.005$) and the slope of SCRs to CS+ across acquisition was significantly different from zero ($b = 0.07$, $t(159) = 5.02$, $p < 0.001$); indicating that all participants demonstrated a significant increase in SCR to CS+ across acquisition (see Figure 1). For CS-, the intercept (i.e., the SCR at the first acquisition trial) was significantly different from zero ($b = 0.23$, $t(21) = 5.37$, $p < 0.001$) and the slope of SCRs to CS- across acquisition was significantly different from zero ($b = -0.02$, $t(159) = -5.04$, $p < 0.001$) indicating that all participants demonstrated a significant decrease in SCR to CS- across acquisition (see Figure 2).

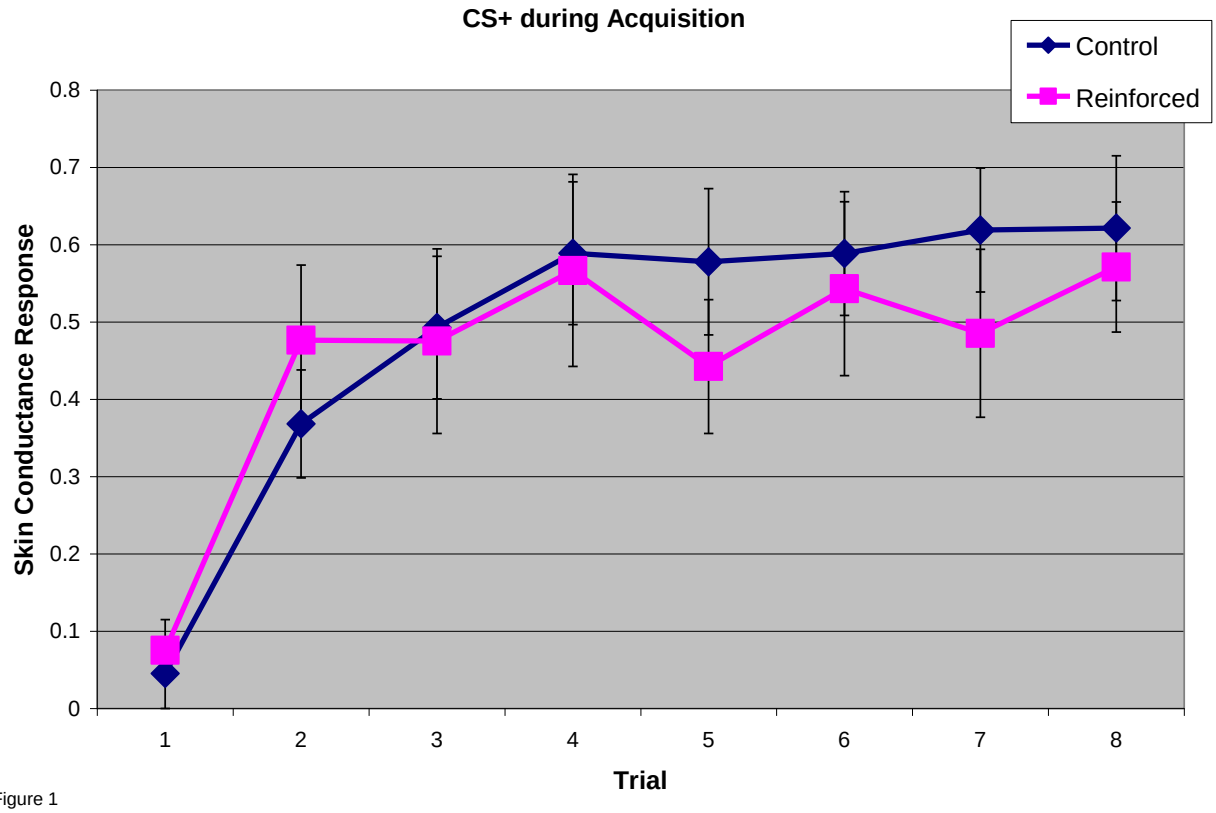


Figure 1

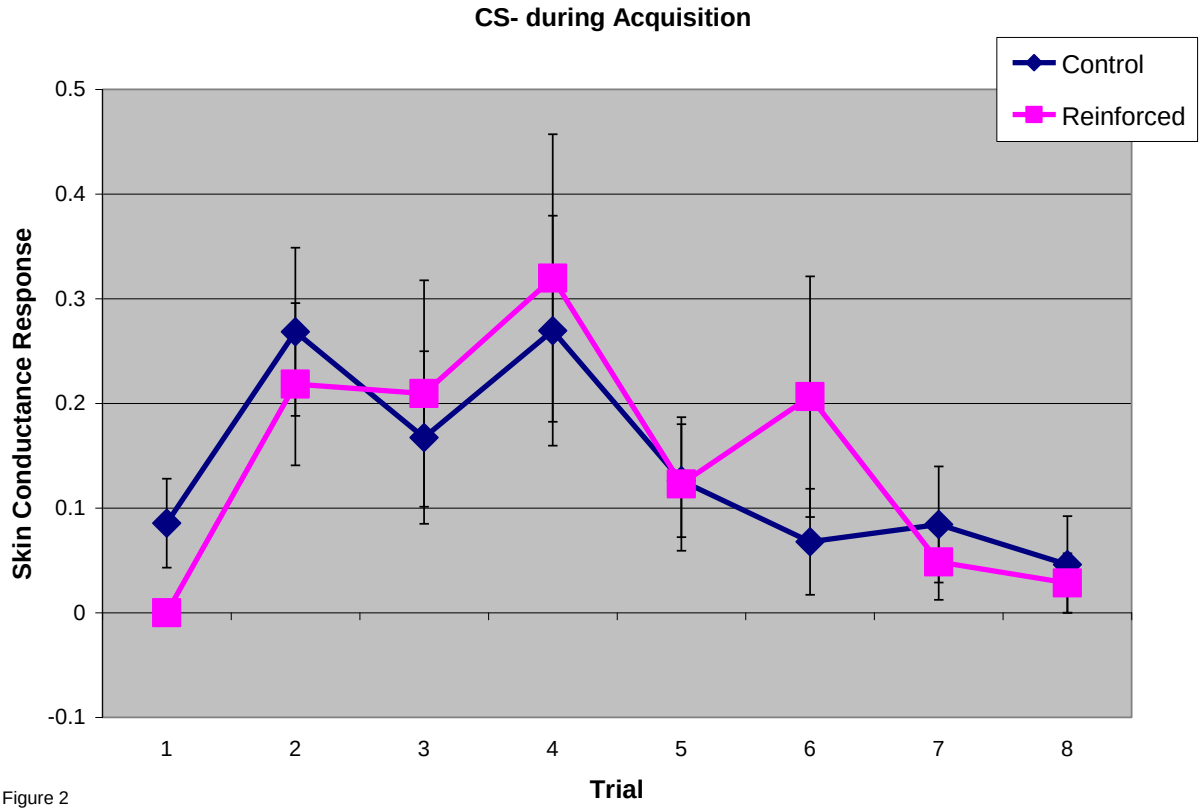


Figure 2

Expectancy Ratings

HLM analyses were conducted for subjective US-expectancy ratings to each conditional stimulus across the eight acquisition trials. For CS+, the slope of expectancy ratings across acquisition was significantly different from zero ($b = 1.01$, $t(159) = 7.22$, $p < 0.001$) indicating that all participants demonstrated a significant increase in US-expectancy ratings to CS+ across acquisition (see Figure 3). For CS-, the slope of expectancy ratings across acquisition was different from zero ($b = -0.34$, $t(159) = -2.31$, $p < 0.05$) indicating that all participants demonstrated a significant decrease in US-expectancy ratings to CS- across acquisition (see Figure 4).

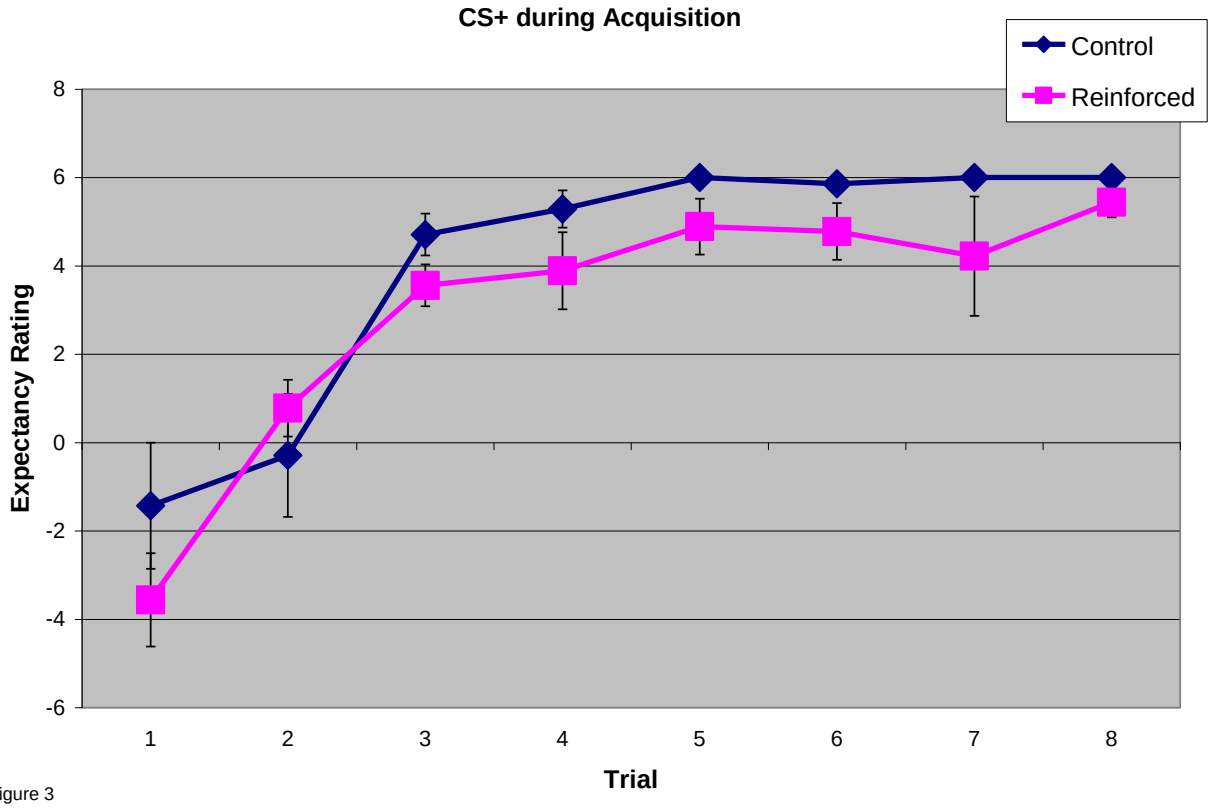


Figure 3

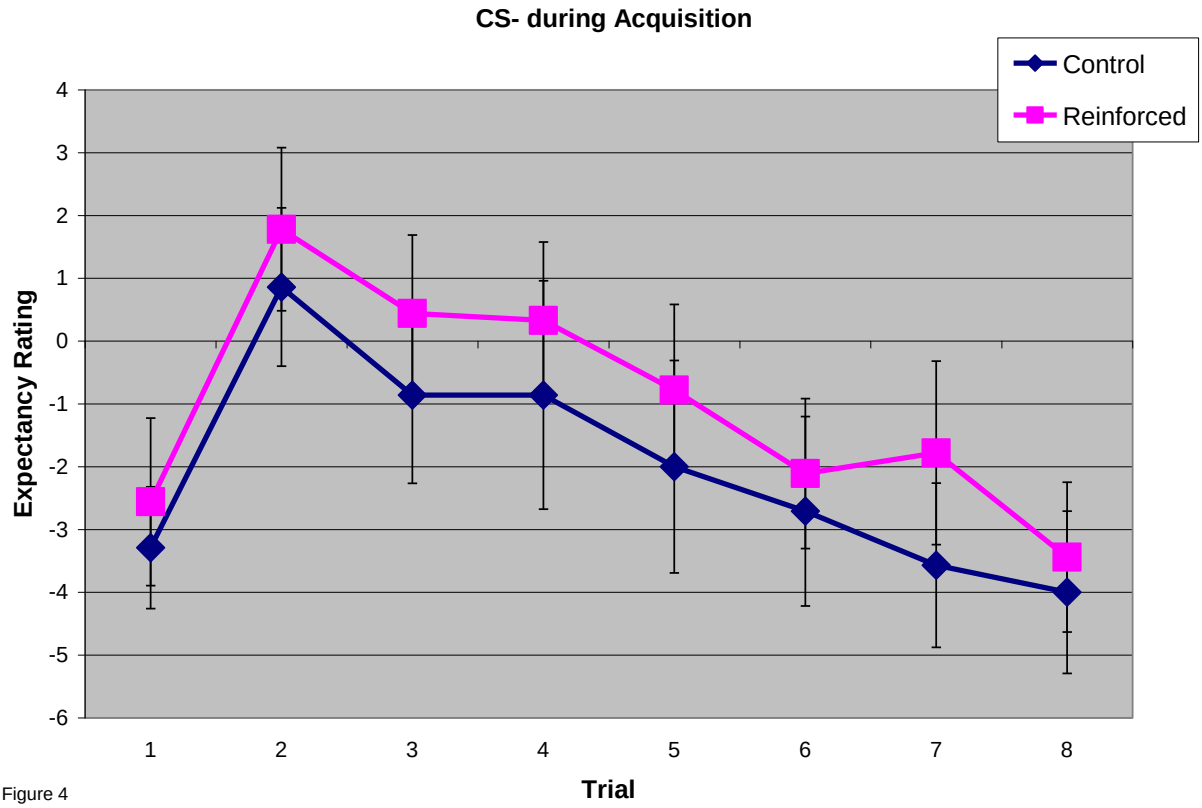


Figure 4

Heart Rate

HLM analyses were conducted for mean heart rate during each conditional stimulus across the eight acquisition trials. For each conditional stimulus, the only significant finding was that the intercept (i.e., mean heart rate during the first trial of acquisition) was significantly different from zero (CS+: $b = 82.52$, $t(21) = 23.62$, $p < 0.001$; CS-: $b = 79.16$, $t(21) = 24.48$, $p < 0.001$; see Figures 5-6).

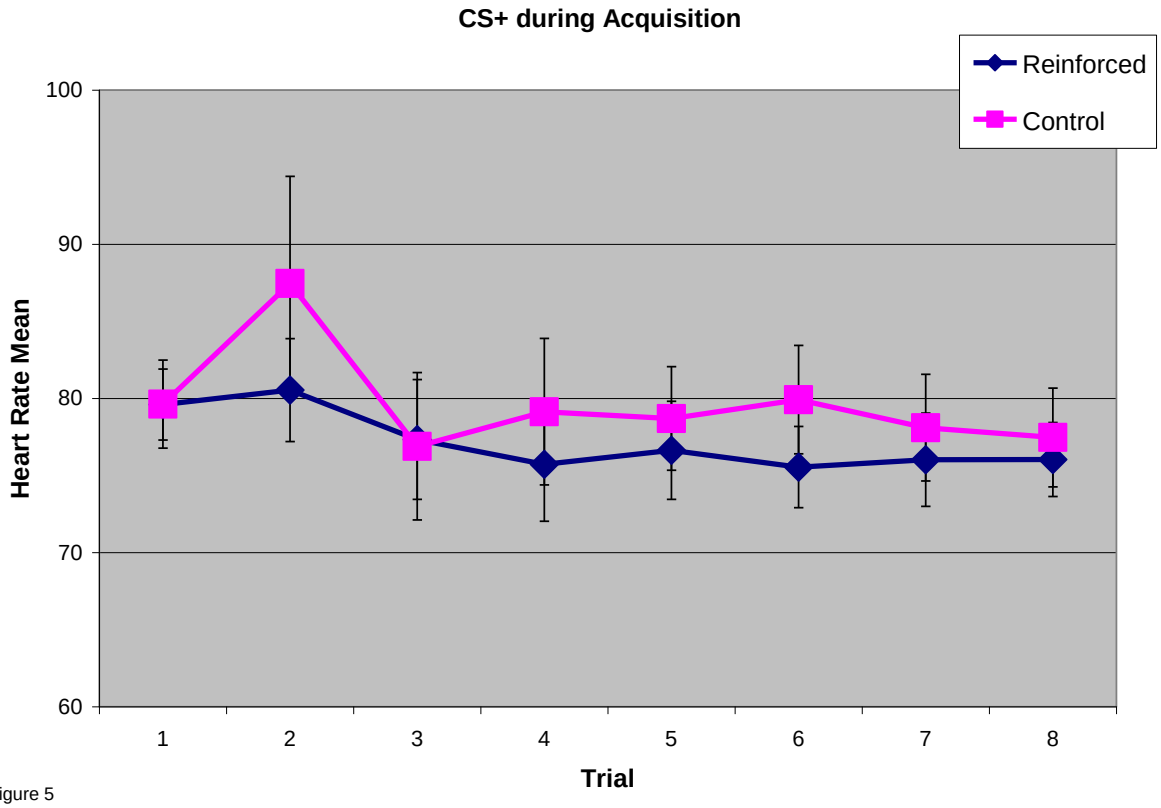


Figure 5

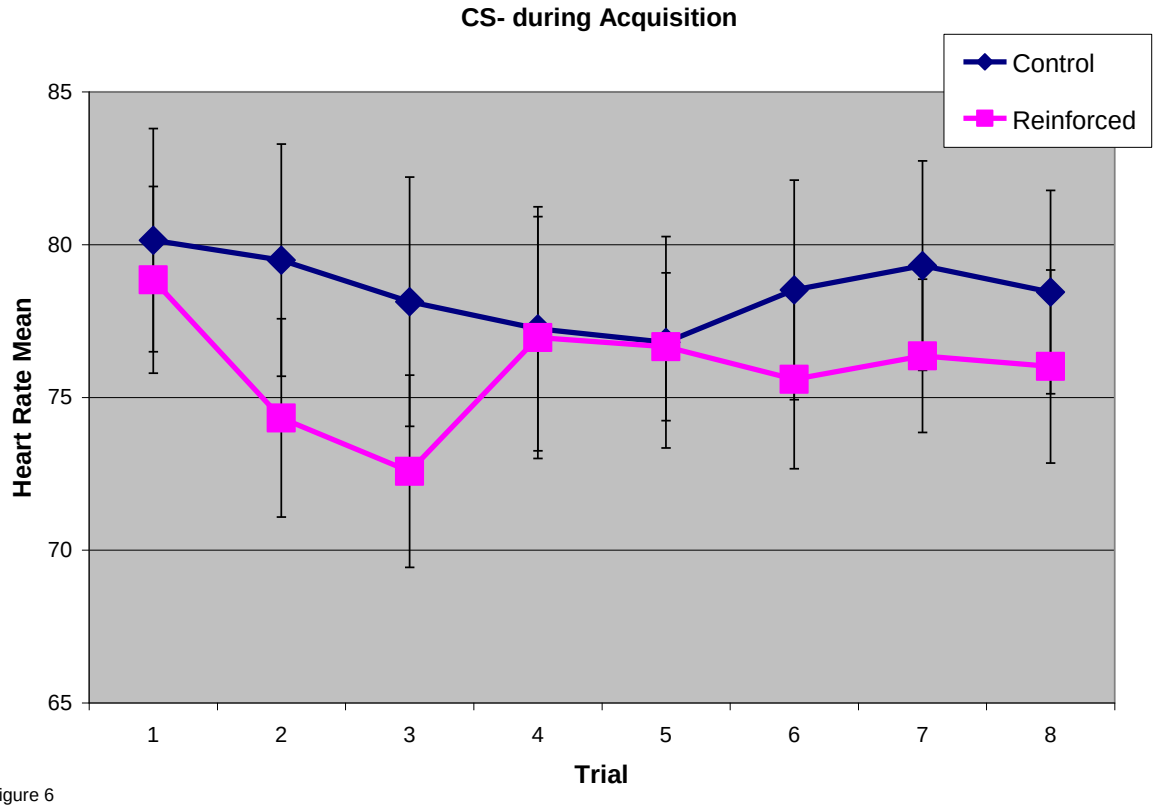


Figure 6

Extinction

Skin Conductance Response (SCR)

HLM analyses were conducted for SCRs to each conditional stimulus across the twenty-four extinction trials. For CS+, the intercept (i.e., SCR at the first trial of extinction) was significantly different from zero ($b = 0.41$, $t(21) = 7.56$, $p < 0.001$). The slope was also significantly different from zero ($b = -0.02$, $t(527) = -7.23$, $p < 0.001$) and the group slopes were significantly different from each other ($b = 0.02$, $t(527) = 5.04$, $p < 0.001$) indicating that group R had a flatter slope than group C (see Figure 7).

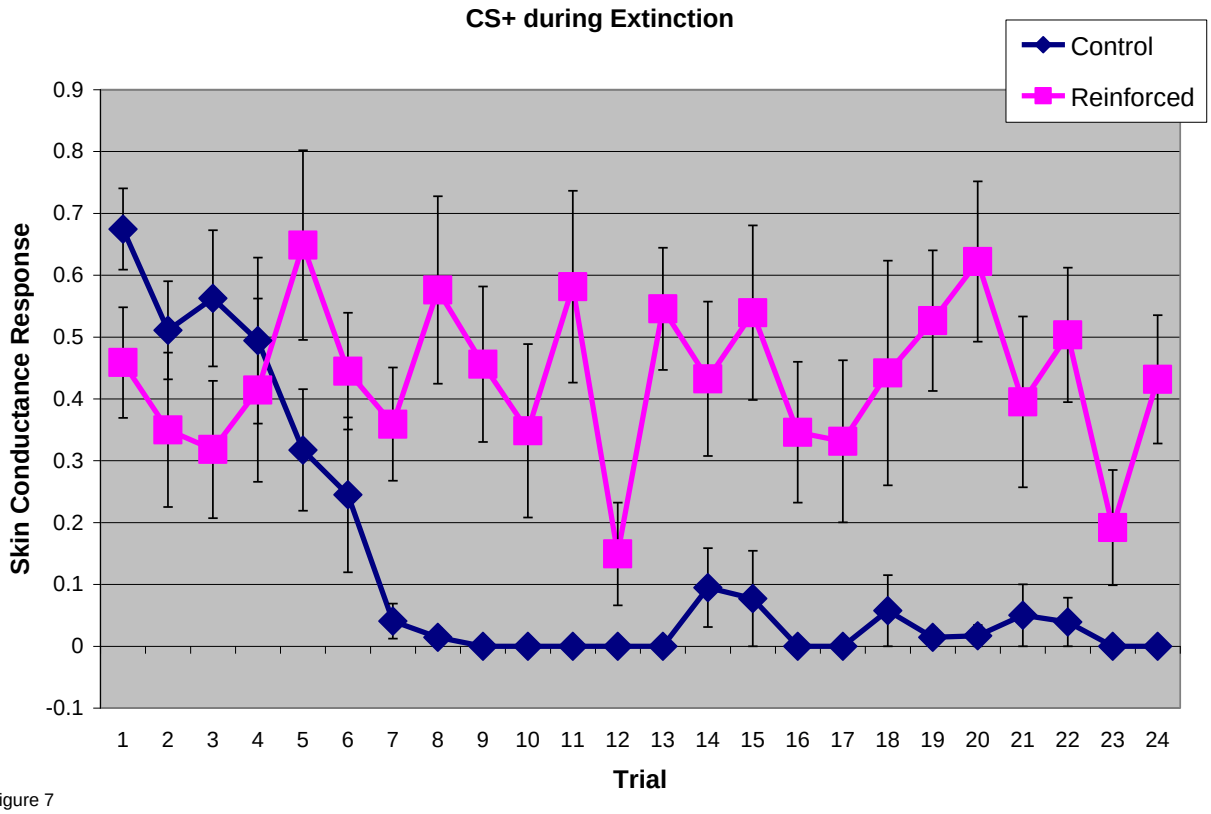


Figure 7

For CS-, the intercept (i.e., SCR at the first extinction trial) was significantly different from zero ($b = 0.10$, $t(21) = 3.75$, $p < 0.005$) and the slope of SCR to CS- was significantly different from zero ($b = -0.01$, $t(527) = -3.00$, $p < 0.005$) indicating that all participants demonstrated a significant decrease in SCRs to CS- across extinction (see Figure 8).

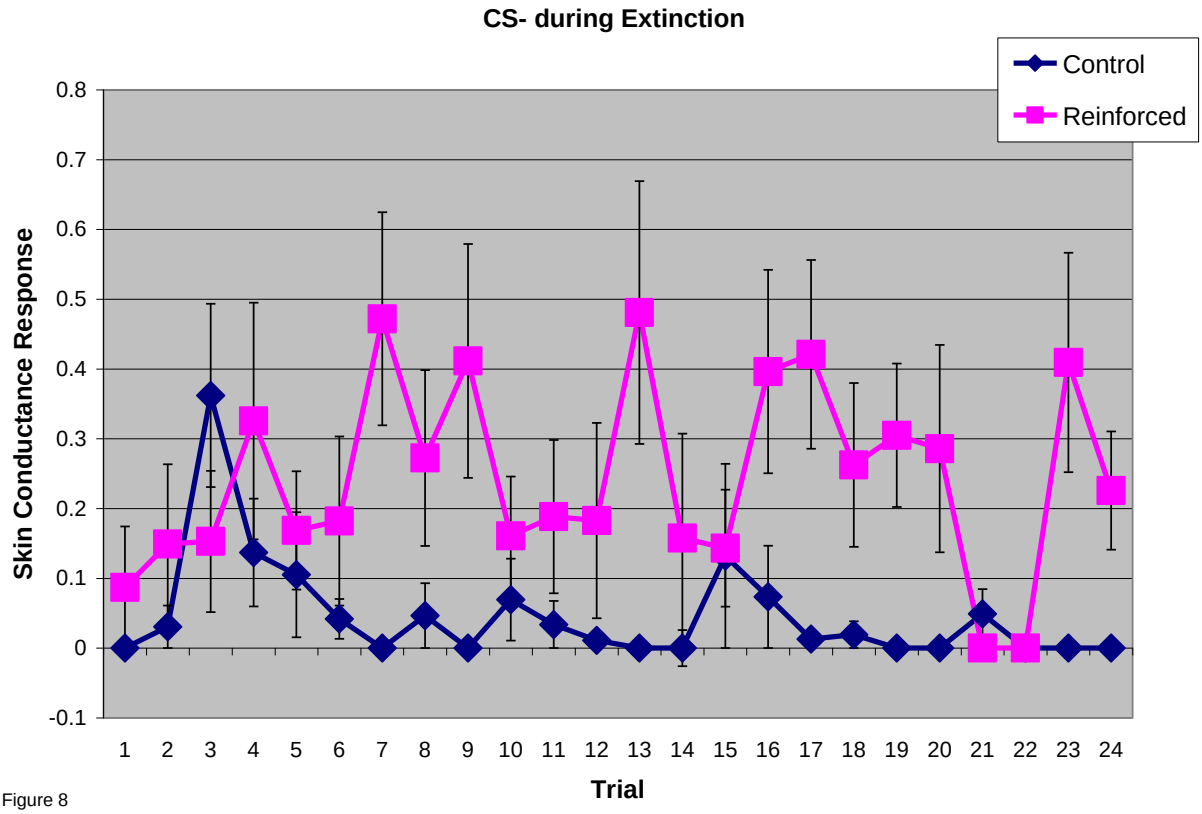


Figure 8

Expectancy Ratings

For CS+, the intercept (i.e., US-expectancy rating at the first trial of extinction) was significantly different from zero ($b = 2.68$, $t(21) = 3.64$, $p < 0.005$). The slope of expectancy ratings to CS+ was also significantly different from zero ($b = -0.30$, $t(527) = -6.29$, $p < 0.001$) and there were significant group slope differences ($b = 0.33$, $t(527) =$

6.01, $p < 0.001$) indicating that group R demonstrated a significantly flatter slope than group C (see Figure 9).

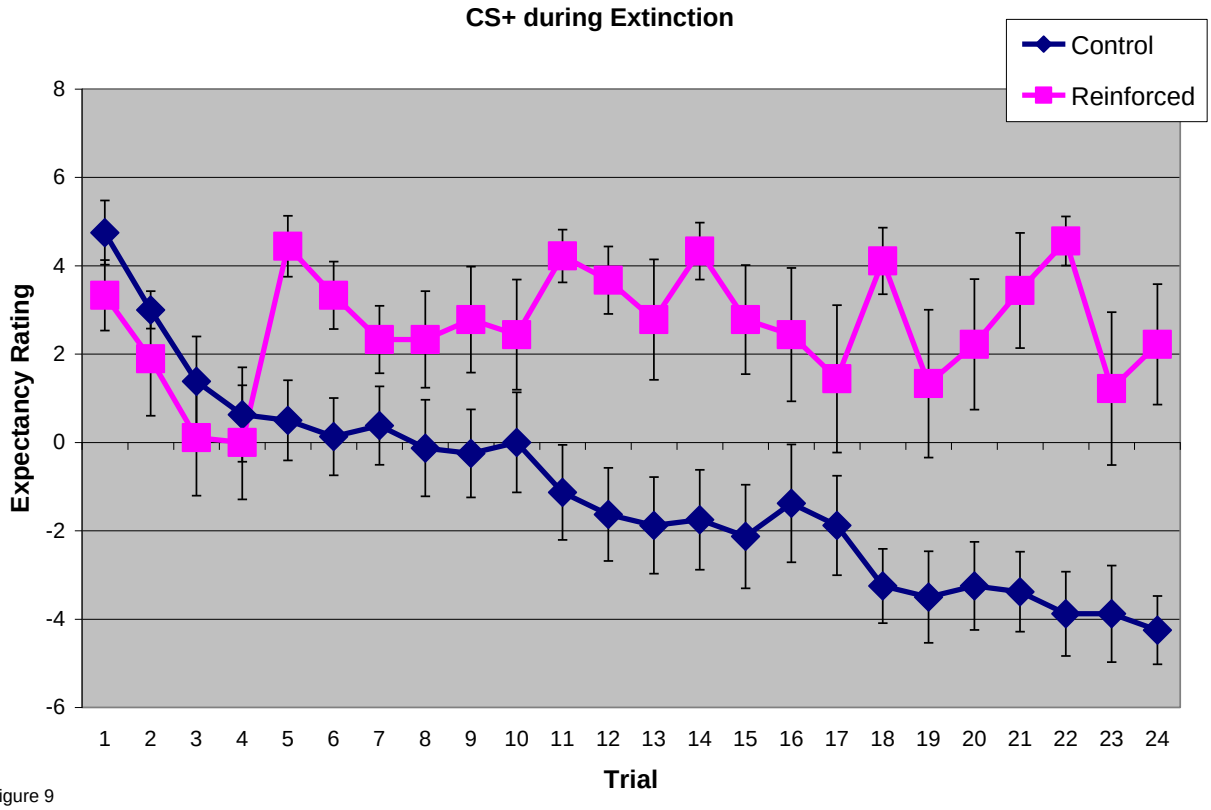


Figure 9

For CS-, the slope of expectancy ratings was significantly different from zero ($b = -0.14$, $t(527) = -6.78$, $p < 0.001$) indicating that all participants demonstrated a significant decrease in US-expectancy ratings to CS- across extinction (see Figure 10).

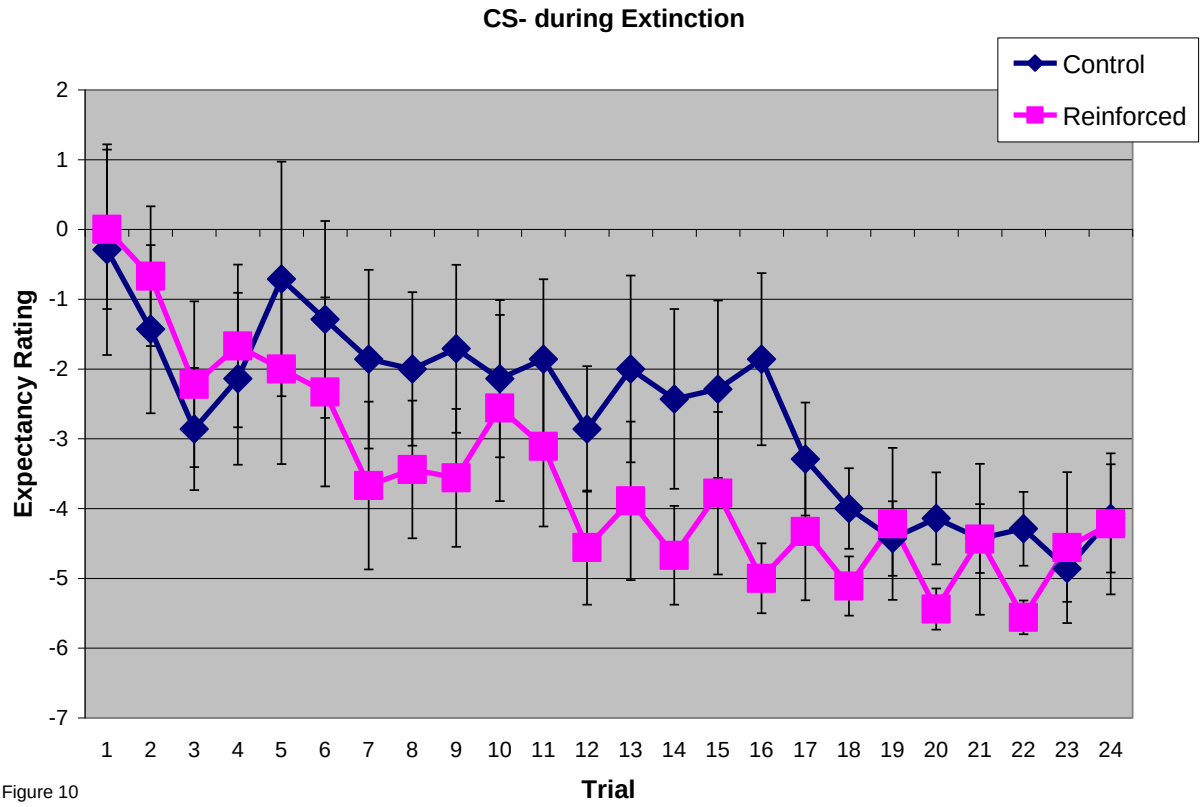


Figure 10

Heart Rate

For each conditional stimulus, the only significant finding was that the intercept (i.e., mean heart rate during the first trial of extinction) was significantly different from zero (CS+: $b = 77.35$, $t(21) = 26.41$, $p < 0.001$; CS-: $b = 77.97$, $t(21) = 27.25$, $p < 0.001$; see Figures 11-12).

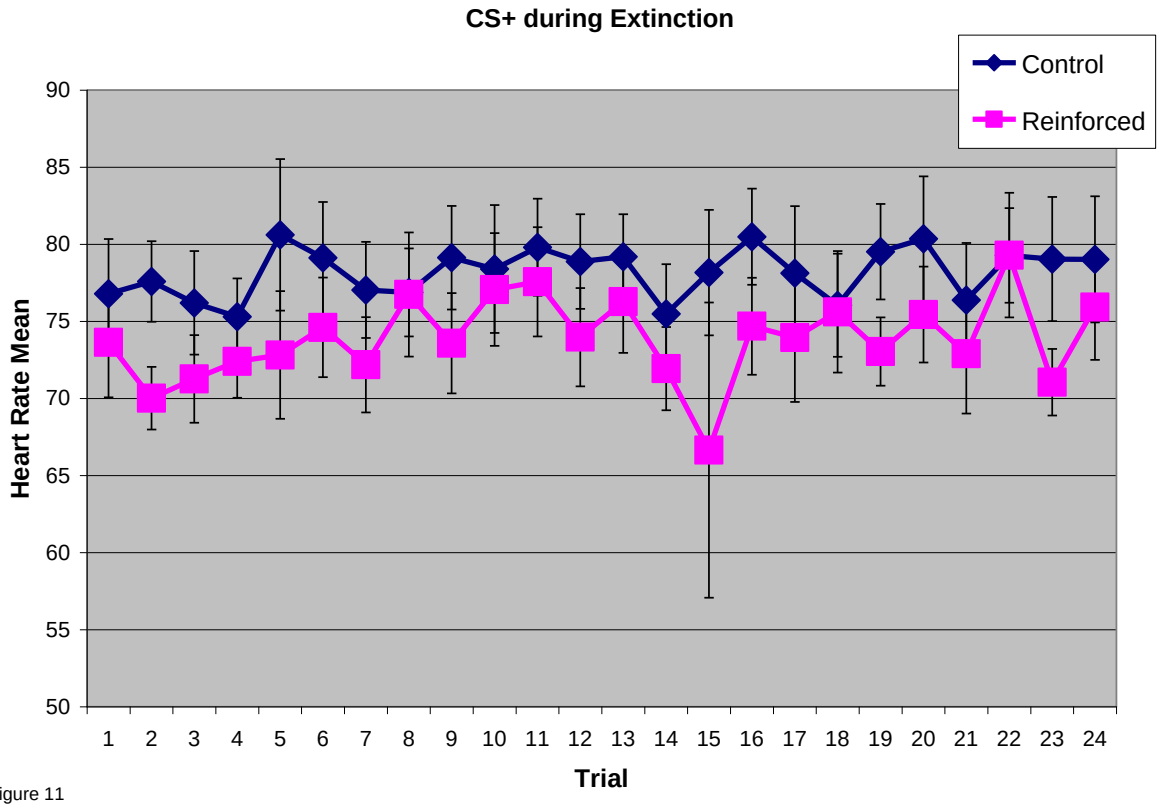


Figure 11

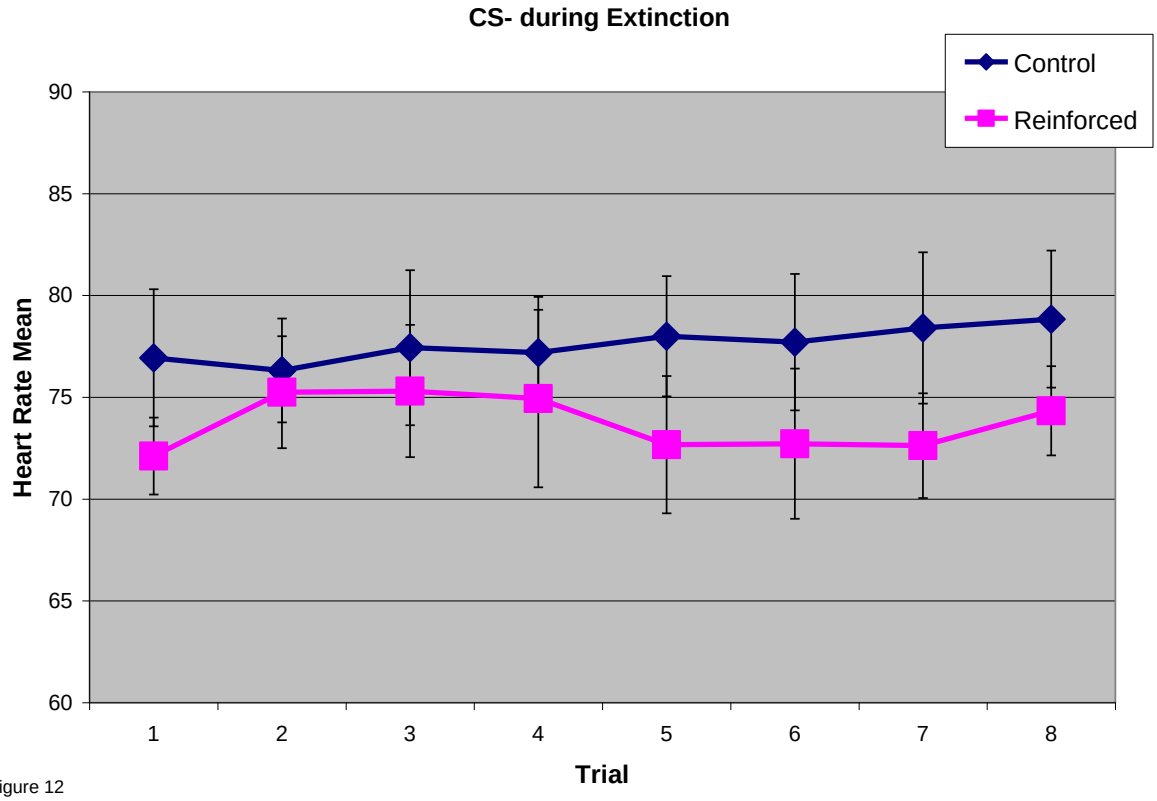


Figure 12

Post-Acquisition to Post-Extinction

Valence Ratings

A 2 (Group: Control versus Reinforced) x 2 (Time: post-Acquisition, post-Extinction) repeated measures ANOVA revealed a significant effect of Time ($F(1,21) = 7.60$, $p < 0.05$, $\eta^2 = 0.34$) and a significant Time x Group interaction effect ($F(1,21) = 22.16$, $p < 0.001$, $\eta^2 = 0.60$). Tests of simple effects examined this interaction and revealed that participants in the Control group demonstrated a significant increase in valence ratings to CS+ from post-Acquisition to post-Extinction ($F(1,21) = 26.31$, $p < 0.001$, $\eta^2 = 0.64$) while participants in the Reinforced group demonstrated no significant change in valence ratings to the CS+ from post-Acquisition to post-Extinction. For CS-, there were no significant effects of Time, Group, or their interaction (see Figure 14).

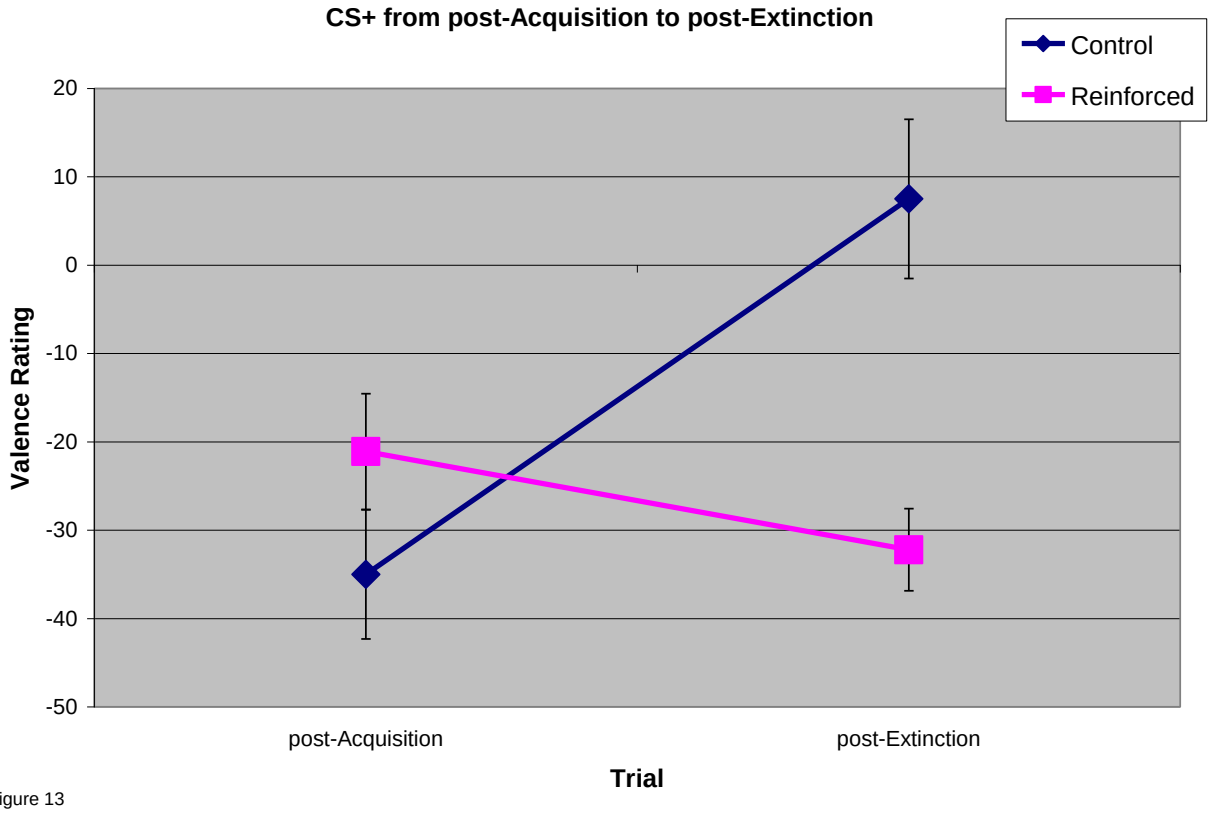


Figure 13

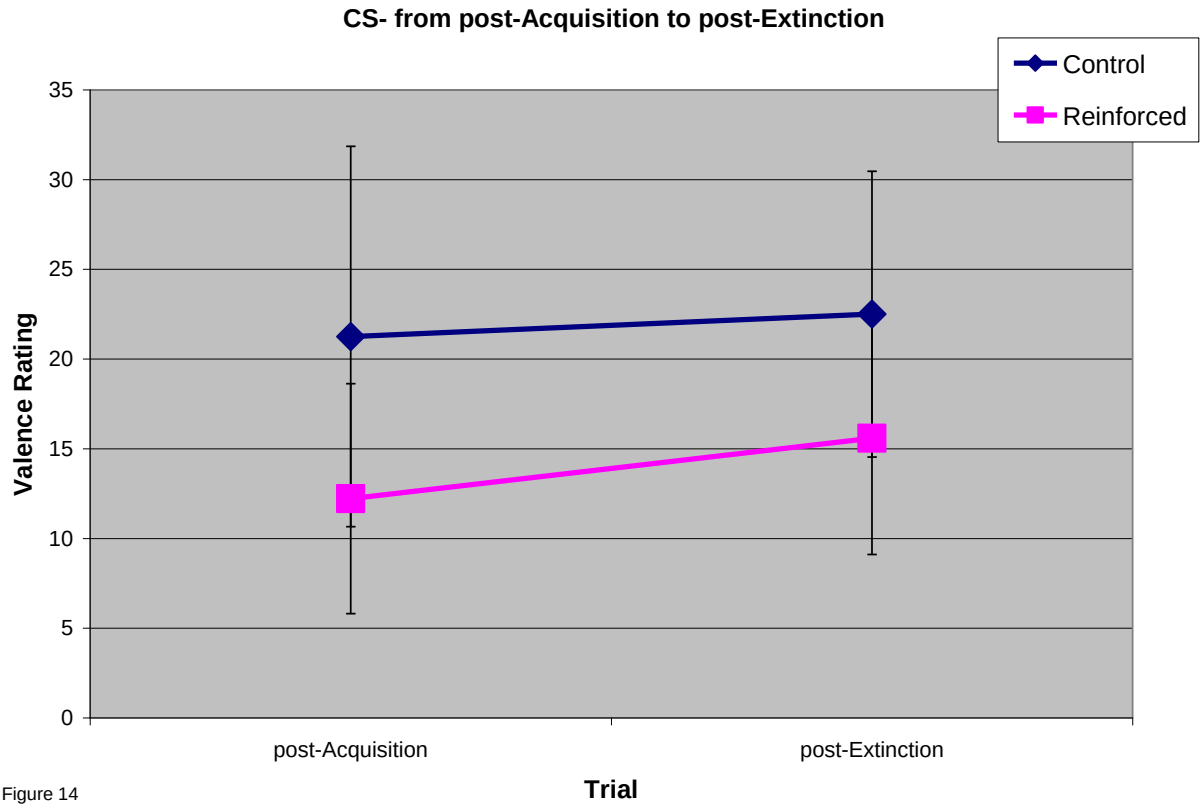


Figure 14

Spontaneous Recovery Test

Change from Endpoint of Extinction to Spontaneous Recovery Test

Skin Conductance Response (SCR)

A 2 (Group: Control versus Reinforced) x 2 (Time: End of Extinction, Spontaneous Recovery Test) repeated measures ANOVA revealed a significant interaction effect ($F(1,21) = 10.11$, $p < 0.01$, $\eta^2 = 0.44$). Tests of simple effects examined this interaction and revealed that participants in the Control group demonstrated a significant increase in skin conductance responses to CS+ from the End of Extinction to the Spontaneous Recovery Test ($F(1,21) = 10.63$, $p < 0.01$, $\eta^2 = 0.45$) whereas participants in the Reinforced group demonstrated no significant change in skin

conductance responses to CS+; see Figure 15). For CS-, there were no significant effects of time, group, or their interaction (see Figure 16).

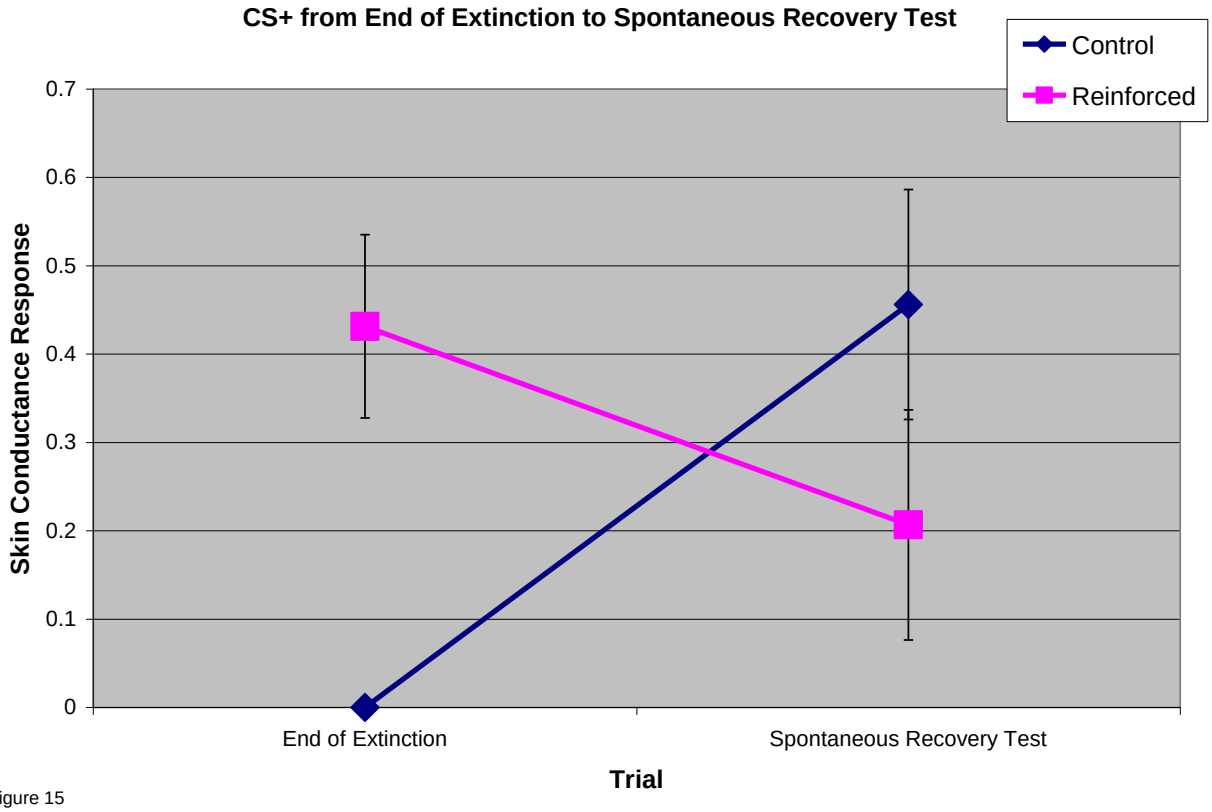


Figure 15

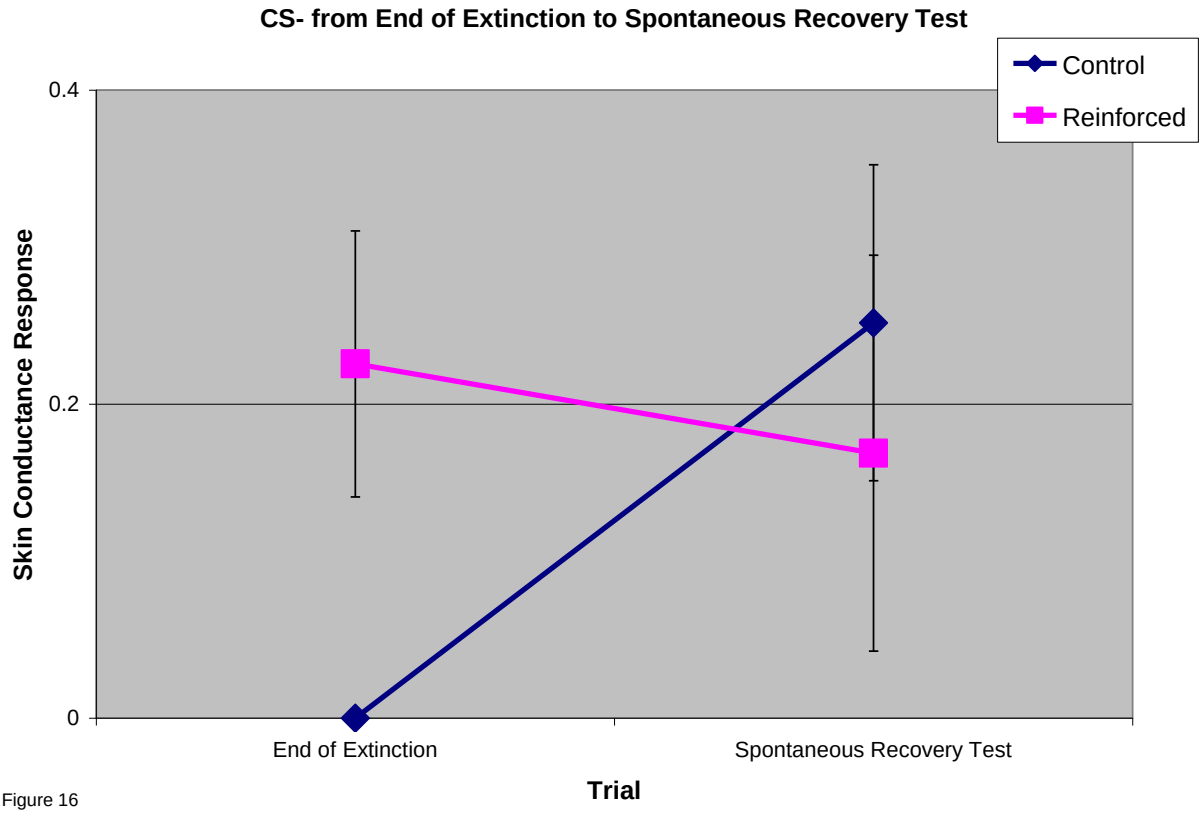


Figure 16

Expectancy Ratings

A 2 (Group: Control versus Reinforced) x 2 (Time: End of Extinction, Spontaneous Recovery Test) repeated measures ANOVA revealed a significant interaction effect ($F(1,21) = 7.44$, $p < 0.05$, $\eta^2 = 0.33$). Tests of simple effects examined this interaction and revealed that participants in the Control group demonstrated a significant increase in US-expectancy ratings to CS+ from the End of Extinction to the Spontaneous Recovery ($F(1,21) = 8.59$, $p = 0.01$, $\eta^2 = 0.36$) whereas participants in the Reinforced group demonstrated no significant change in US-expectancy ratings to CS+ (see Figure 17).

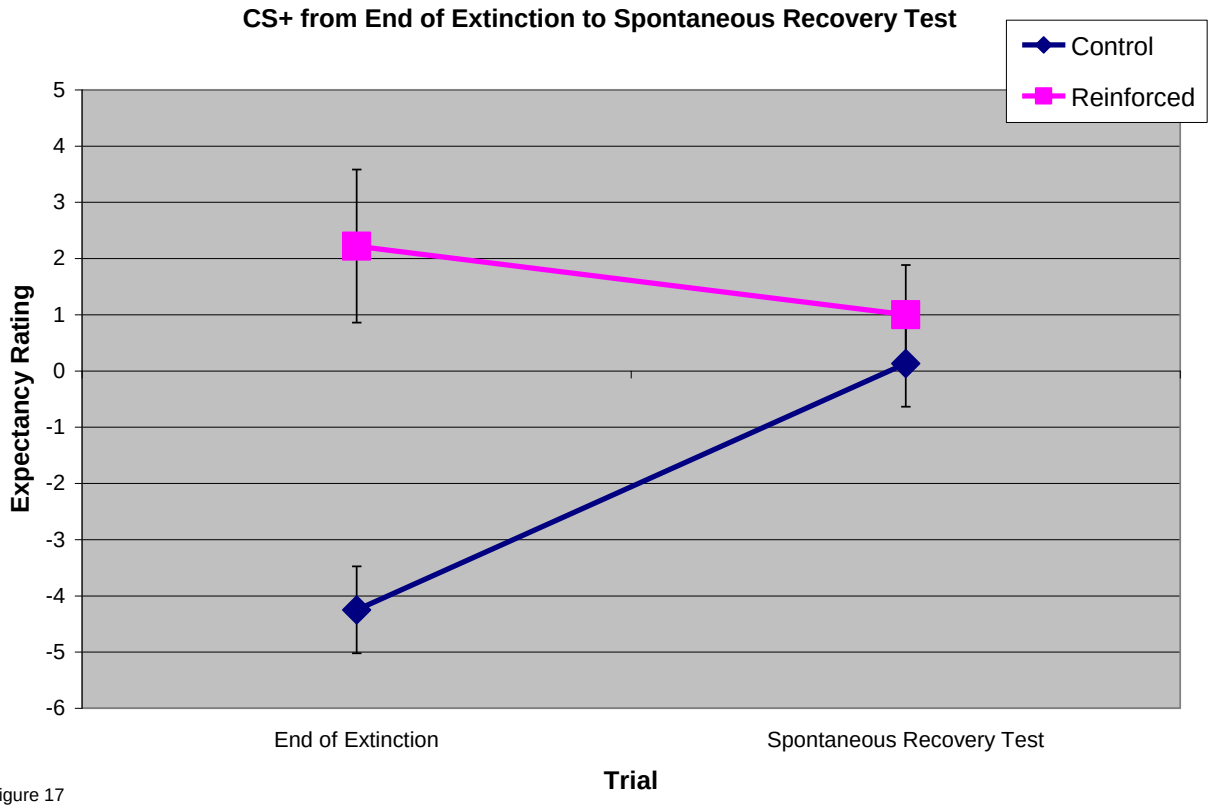


Figure 17

For CS-, a 2 (Group: Control versus Reinforced) x 2 (Time: End of Extinction, Spontaneous Recovery Test) repeated measures ANOVA revealed a significant interaction effect ($F(1,21) = 6.17$, $p < 0.05$, $\eta^2 = 0.31$). Tests of simple effects examined this interaction and revealed that participants in the Control group demonstrated a significant increase in US-expectancy ratings to CS- from the End of Extinction to the Spontaneous Recovery ($F(1,21) = 6.63$, p , 0.05, $\eta^2 = 0.32$) whereas participants in the Reinforced group demonstrated no significant change in US-expectancy ratings to CS- (see Figure 18).

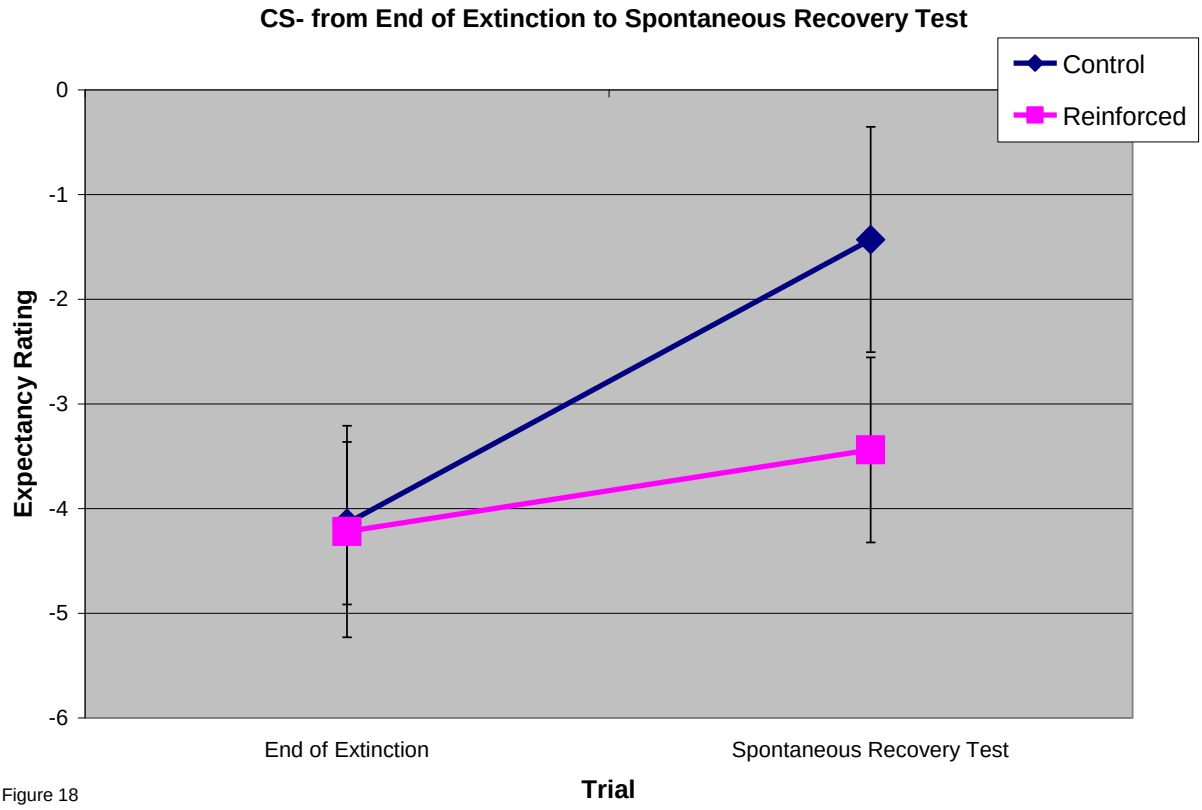


Figure 18

Heart Rate

For both CS+ and CS-, there were no significant findings of time, group, or their interaction on heart rate means from the end of extinction to the spontaneous recovery test.

Valence Ratings

A 2 (Group: Control versus Reinforced) x 2 (Time: End of Extinction, Spontaneous Recovery Test) repeated measures ANOVA revealed a significant effect of Group ($F(1,21) = 11.16$, $p < 0.004$, $\eta^2 = 0.43$) and a significant Time x Group interaction effect ($F(1,21) = 5.94$, $p < 0.05$, $\eta^2 = 0.28$). Tests of simple effects examined this interaction and revealed that participants in the Reinforced group demonstrated a significant increase in valence ratings to CS+ from the End of Extinction to the

Spontaneous Recovery ($F(1,21) = 5.17$, $p < 0.05$, $\eta^2 = 0.26$) whereas participants in the Control group demonstrated no significant change in valence ratings to CS+ from the End of Extinction to the Spontaneous Recovery Test ($F(1,21) = 1.45$, $p = 0.25$, $\eta^2 = 0.09$; see Figure 19).

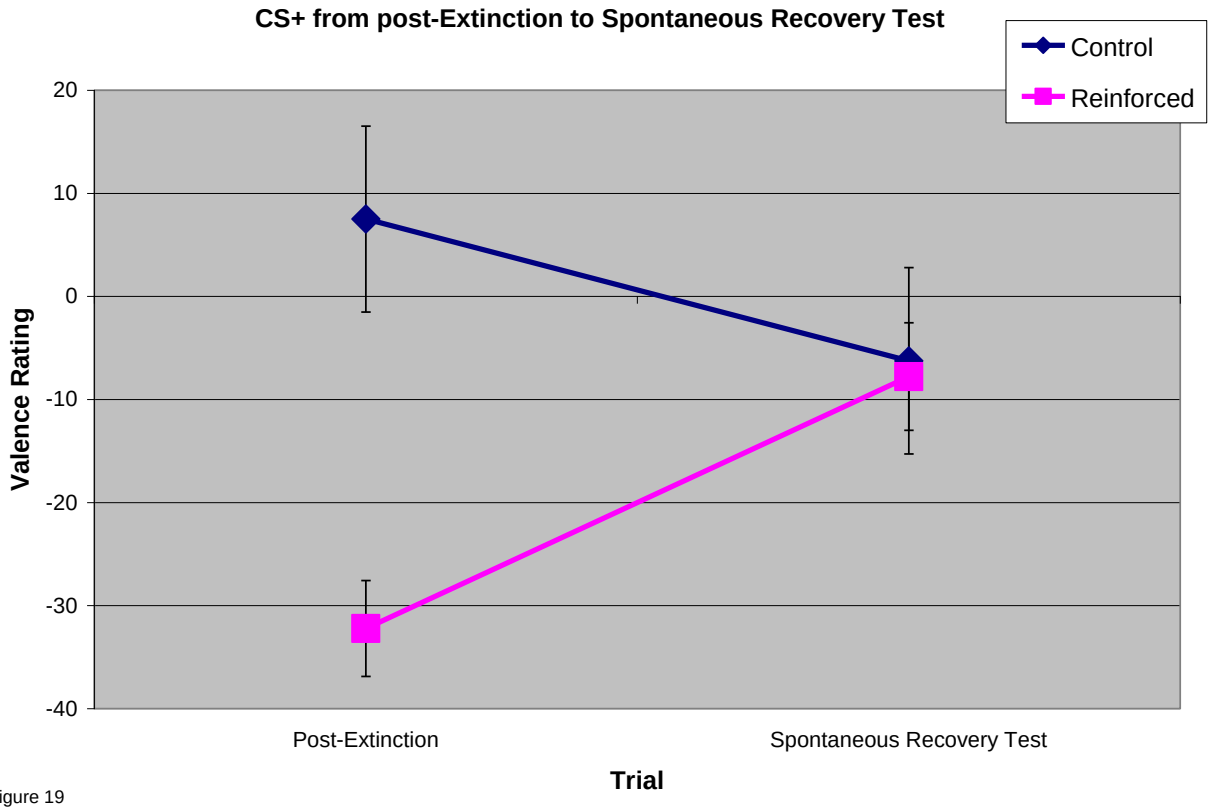


Figure 19

For CS-, a 2 (Group: Control versus Reinforced) x 2 (Time: End of Extinction, Spontaneous Recovery Test) repeated measures ANOVA revealed a significant effect of time ($F(1,21) = 6.28$, $p < 0.05$, $\eta^2 = 0.30$), indicating that all participants demonstrated a significant decrease in valence ratings to CS- from the end of extinction to the one-week follow-up test (see Figure 20).

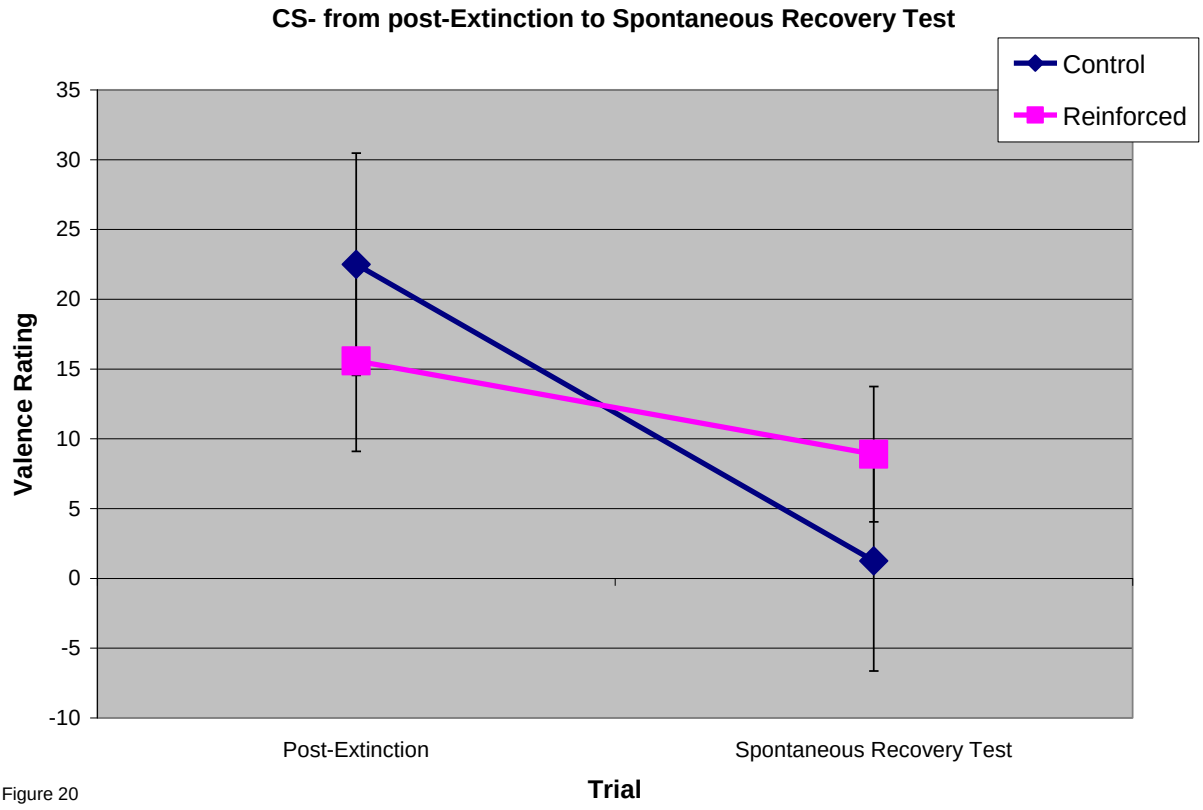


Figure 20

Rapid Reacquisition Test

Skin Conductance Response (SCR)

HLM analyses were conducted across the four reacquisition trials at Day 2. For CS+, the slope was significantly different from zero (b = 0.16, t(67) = 3.50, p = 0.001 and there was a nonsignificant trend towards a group slope difference (b = -0.11, t(67) = -1.82, p = 0.07) indicating that group R demonstrated a flatter slope than group C (see Figure 21). For CS-, there were no significant findings (see Figure 22).

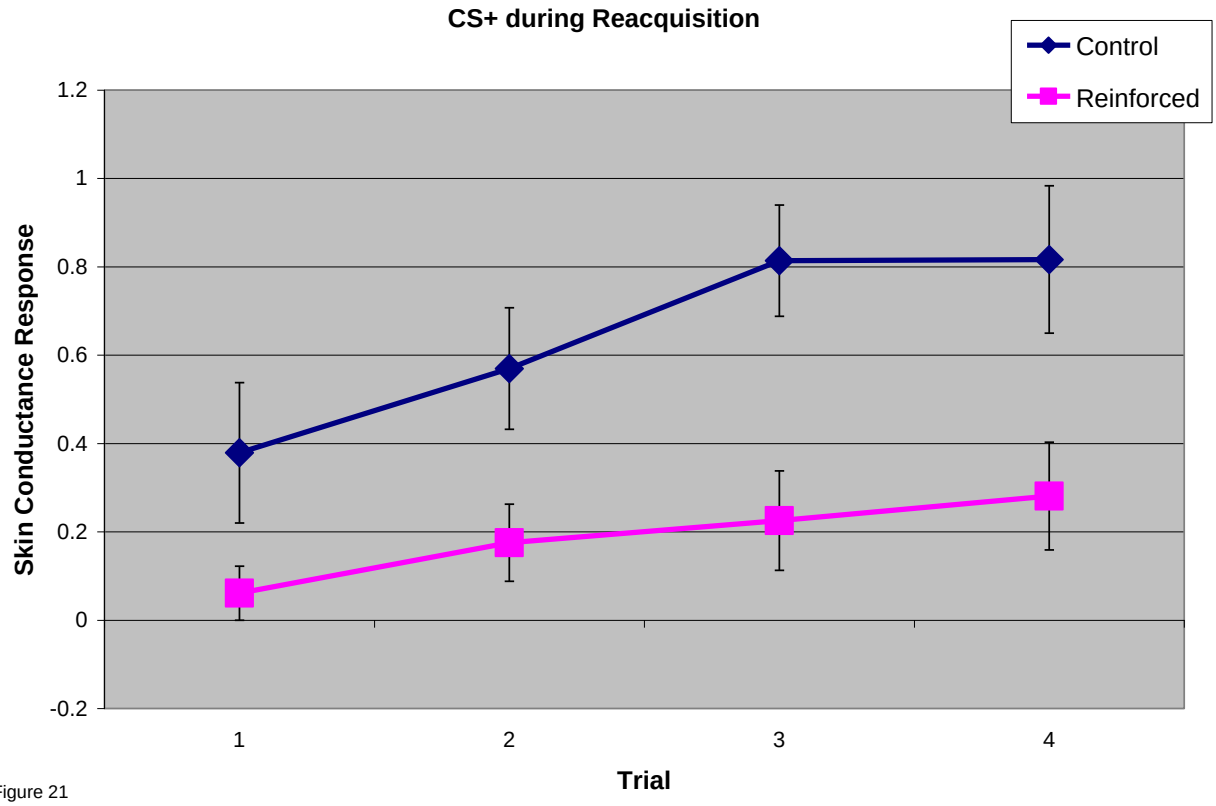


Figure 21

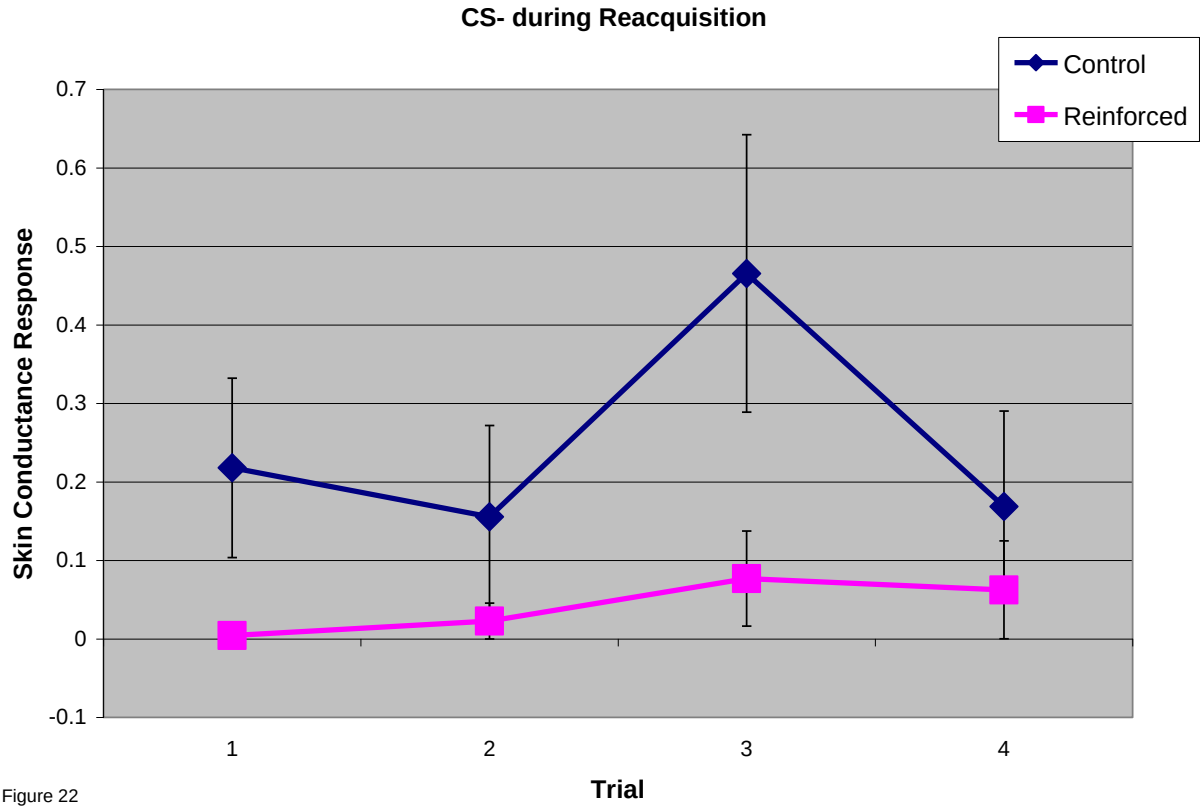


Figure 22

Expectancy Ratings

For CS+, the intercept was significantly different from zero (b = -6.01, t(21) = -3.05, $p < 0.01$) and the slope was significantly different from zero (b = 1.49, t(67) = 5.23, $p < 0.001$) indicating that all participants demonstrated US-expectancy ratings higher than zero at the first trial of reacquisition and all participants demonstrated a significant increase in US-expectancy ratings across the four reacquisition trials (see Figure 23).

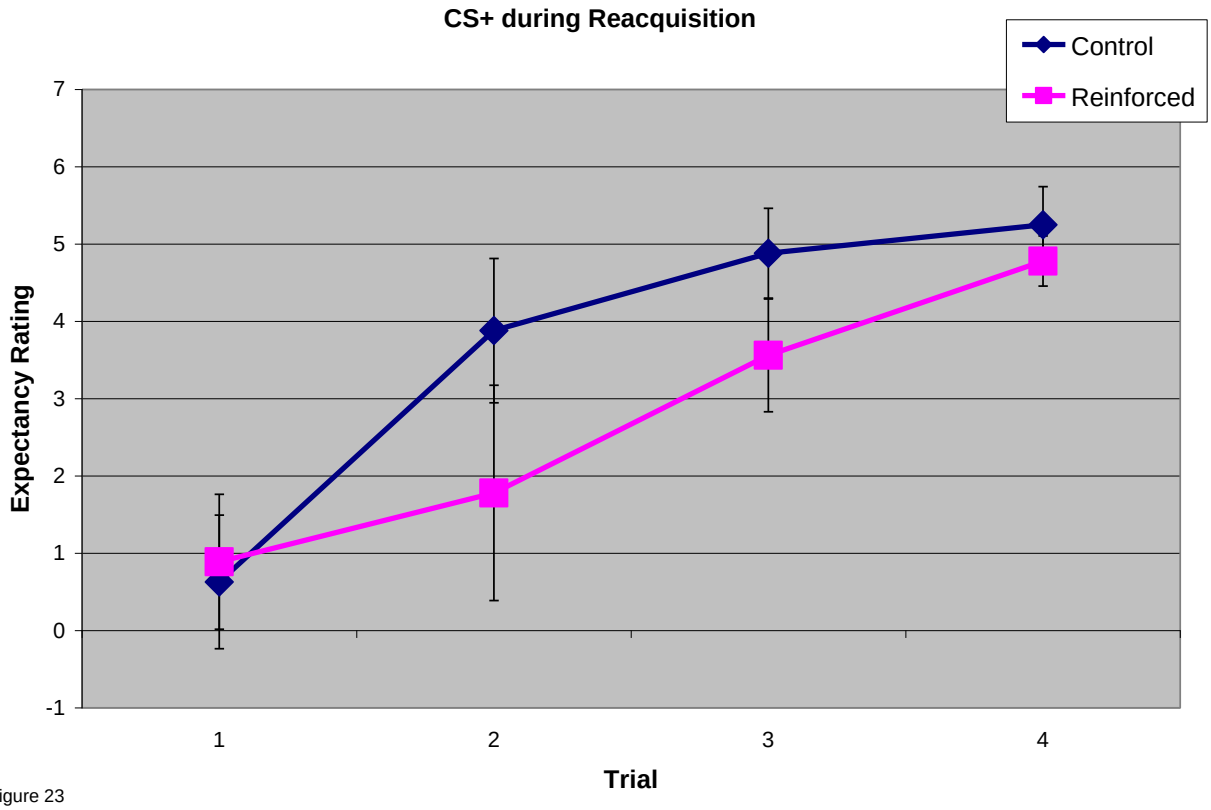


Figure 23

For CS-, there were significant group differences at the intercept (b = -4.76, t(21) = -2.34, $p < 0.05$) indicating that participants in the R group demonstrated significantly lower US-expectancy ratings than participants in the C group at the first trial of reacquisition. And the slope was significantly different from zero (b = -0.49, t(67) = -

2.45, $p < 0.05$) indicating that all participants demonstrated a decrease in US-expectancy ratings to CS- across the four reacquisition trials (see Figure 24).

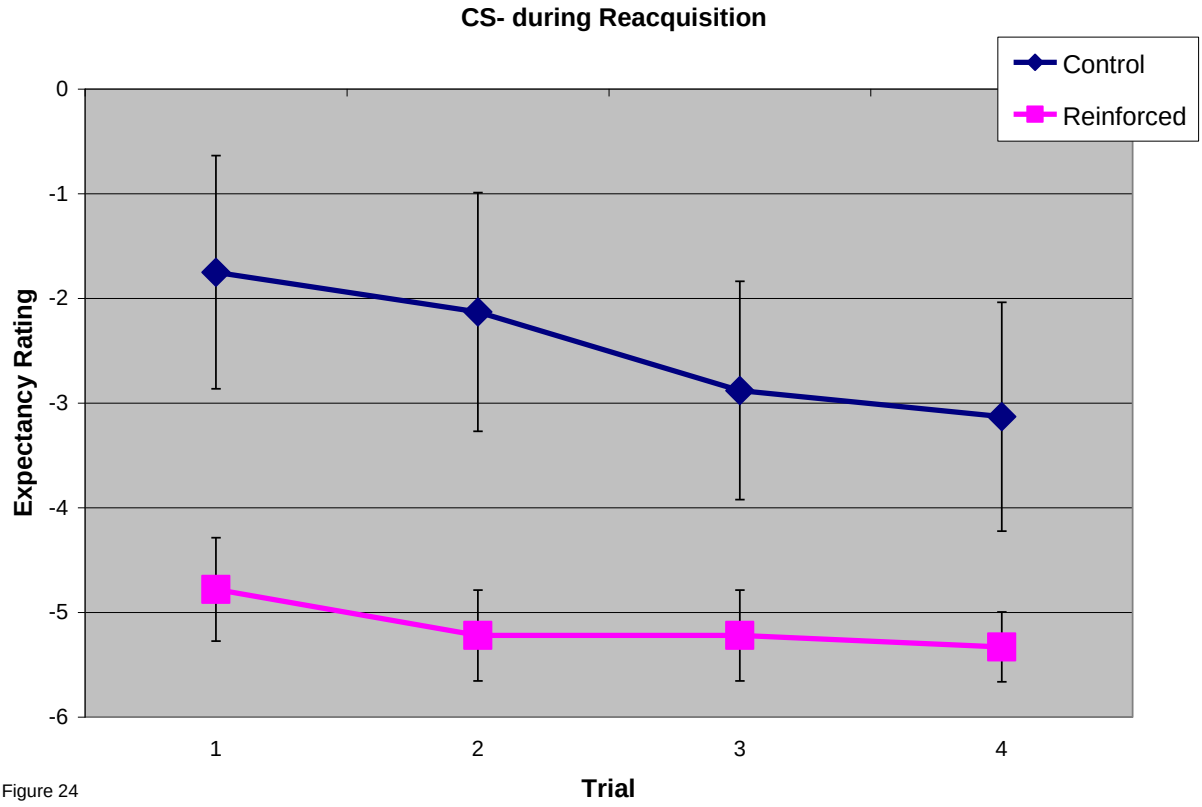


Figure 24

Heart Rate

For each conditional stimulus, the only significant finding was that the intercept (i.e., mean heart rate during the first trial of reacquisition) was significantly different from zero ($b = 82.26$, CS+: $t(21) = 15.56$, $p < 0.001$; CS-: $b = 77.58$, $t(21) = 15.90$, $p < 0.001$).

Discussion

This study tested the hypotheses that occasional reinforced trials during extinction training would lead to less recovery of fear at the spontaneous recovery and rapid reacquisition tests one week later. The results of the study provide strong evidence for the study hypotheses.

As measured by subjective US-expectancy ratings and skin conductance responses to the CS+, there were significant group differences in the change from the end of the extinction to the spontaneous recovery test. Skin conductance responses to CS+ were significantly higher in the Reinforced group than in the Control at the endpoint of extinction; however, the Reinforced group demonstrated no significant change from the end of extinction to the spontaneous recovery test one week later whereas the Control group demonstrated a significant increase. Thus, participants in the Control group demonstrated significant spontaneous recovery of physiological fear responding to the CS+ whereas participants in the Reinforced group did not. Similarly, US-expectancy ratings to CS+ were significantly higher in the Reinforced group than in the Control group at the endpoint of extinction. However, participants in the Control group demonstrated a significant increase from extinction to test whereas participants in the Reinforced group demonstrated no significant change. Thus, participants in the Control group demonstrated significant spontaneous recovery of subjective fear responding to the CS+ whereas participants in the Reinforced group did not. Lastly, participants in the Reinforced group demonstrated a significant *increase* in valence ratings to CS+ from extinction to test (i.e., they rated the CS+ as significantly more "pleasant" at the spontaneous recovery than at the end of extinction). Thus, neither group demonstrated

spontaneous recovery effects in valence ratings; furthermore, the Reinforced group actually demonstrated a significant change in the opposite direction to that predicted by spontaneous recovery.

These results are exciting and important; however the clinical significance remains a bit uncertain. Even though participants in the Reinforced group demonstrated no spontaneous recovery effects in physiological or subjective fear responding to the fearful stimulus, they also demonstrated significantly higher fear responding throughout extinction; thus, the absolute level of fear responding at the spontaneous recovery test was not different in the two groups. It remains to be seen whether longer intervals between extinction and test would lead to significant group differences in fear responding. Since the Control group demonstrated a significant increase in fear responding from extinction to test, it is conceivable that the two groups would have diverged in absolute fear responding had the spontaneous recovery test occurred two weeks later rather than one week later. In fact, data in animal models indicate that longer intervals between extinction and test lead to greater spontaneous recovery effects (e.g., Quirk, 2002).

The results of the rapid reacquisition test provide further evidence for the effectiveness of occasional reinforced trials in protecting against fear recovery. Skin conductance responses across the four reacquisition trials indicated that all participants demonstrated an increase in physiological fear responding to the CS+ when it was again paired with the US. However, participants in the Reinforced group showed a flatter slope than participants in the Control group (although this group slope difference did not quite reach significance).

Rapid reacquisition is an especially problematic phenomenon in the treatment of anxiety disorders, especially those disorders in which the feared catastrophe (i.e., the US) is likely to reoccur following successful treatment. For example, in the treatment of social anxiety disorder, typical procedures involve repeated exposure to the conditional stimuli (social situations) in the absence of the unconditional stimulus (social rejection or negative evaluation). During treatment, conditional fear responding to social situations decreases as the individual learns that rejection and negative evaluation do not occur. However, occasional negative evaluations and social rejection are unavoidable in life. Thus, for the socially anxious individual who successfully undergoes exposure treatment and is then faced with rejection or negative evaluation at some point in the future (i.e., the CS and US occur together again), rapid reacquisition predicts that this individual's fear of social situations will quickly recover. In other words, he or she will experience a relapse of their social anxiety disorder. The results of this study indicate that building in some CS-US pairings during extinction may protect against this potential for relapse. This would entail the individual experiencing some negative evaluations and/or some experiences of social rejection during exposure treatment.

Similarly, for individuals with panic disorder, the feared catastrophe (i.e., US) is the panic attack. Exposure therapy for panic disorder involves repeated exposure to both internal and external cues of panic attacks (i.e., CSs) in the absence of a panic attack. This allows individuals to learn that they can experience internal cues of a panic attack (e.g., a slight increase in heart rate) and external cues of a panic attack (e.g., being in a crowd) without actually experiencing a panic attack; thus, conditional fear responding to these cues decrease. However, the rapid reacquisition phenomenon predicts that fear

responding to these cues will recover quickly if the individual again experiences a panic attack following successful exposure therapy. Incorporating CS-US pairings during exposure treatment for panic disorder would entail purposely inducing occasional panic attacks during exposure. The current results suggest that doing so may protect the individual from relapse in the future.

Of course, there are ethical limitations to utilizing these procedures in the clinical treatment of anxiety disorders. In the treatment of post-traumatic stress disorder, for instance, it is not ethical to expose the individual to the US during exposure treatment since the US in this situation is a trauma. Or, in the treatment of specific phobias, the feared catastrophe may be receiving a bite from an animal or insect or being in a crash while driving or flying. In such cases, it is obviously not feasible to incorporate CS-US pairings in exposure treatment. However, when treating anxiety disorders in which the US is most likely to reoccur following treatment (such as in the case of social rejection for example) and it is ethical as well as feasible to include some CS-US pairings during exposure, doing so may protect the individual from future relapse.

STUDY 3

SUSTAINED AROUSAL AND EMOTIONAL VARIABILITY DURING EXPOSURE ATTENUATES RECOVERY OF FEAR AT FOLLOW-UP TESTING[1]

Introduction

A large body of literature supports the effectiveness of exposure therapy for the treatment of anxiety disorders and phobias (e.g., Norton & Price, 2007). Anxiety disorders are characterized by avoidance of feared stimuli and this avoidance perpetuates the fear: each time the individual avoids the fearful stimulus, he or she reinforces the belief that avoidance is the key to remaining safe. Exposure exercises force the individual to test the hypothesis that facing the fearful stimuli will *not* lead to catastrophic results. During exposure therapy, the individual learns that nothing aversive occurs while in the presence of the fearful stimuli; therefore, fear of the stimuli decreases. Thus, extinction learning is believed to be one mechanism underlying the effectiveness of exposure therapy (Eelen & Vervliet, 2006).

However, not every recipient of exposure therapy benefits (non-response rates roughly vary from 10 to 30% depending on the anxiety disorder; see Craske, 1999). In addition, even for those who do benefit, fear responding can sometimes return (Craske & Mystkowski, 2006). In fact, it is now well understood that extinction does not involve the unlearning of the previous fear association but, rather, new learning. That is, rather

[1] A version of this paper has been published: Culver, N., Stoyanova, M.S., & Craske, M.G. (2012). Emotional variability and sustained arousal during exposure. *Journal of Behavior Therapy and Experimental Psychiatry, 43(2),* 787-793.

than being a passive process, extinction is an inhibitory but active form of learning (Bouton, 1993). Thus, even though extinction can eliminate fearful responding, the original fear learning is intact and retrievable as demonstrated by various procedures including changing the test context (renewal; Bouton, 1993), presenting unsignaled USs (reinstatement; Rescorla & Heth, 1975), or simply allowing time to pass (spontaneous recovery; Baum, 1988). The fact that the fear memory appears to remain intact even after extinction learning occurs can be problematic for treatment. Individuals may successfully complete exposure therapy only to experience subsequent return of fear. Thus, investigations aimed at finding methods for enhancing exposure-based interventions are warranted in order to increase response rates and decrease the likelihood of return of fear following successful treatment.

Investigations using animal models to explore methods for enhancing extinction learning may provide insight into methods for increasing the effectiveness of exposure therapy (Hermans, Craske, Mineka, & Lovibond, 2006). In nonprimate samples, Rescorla (2000) demonstrated that extinction of a conditional stimulus (CS) in the presence of additional excitatory stimuli was superior to extinction alone. Although fear responding during extinction was greater when the CS underwent extinction training in the presence of an additional excitatory stimulus, these rats demonstrated significantly less fear responding to that CS at follow-up testing. Thus, additional excitatory stimuli during extinction training increase fear responding during extinction but also facilitate long-term fear reduction. Rescorla (2000) posits potential mechanisms for these results including: 1) that when the CS is presented with another excitatory stimulus, the discrepancy between expectation (i.e., of the US) and reality (i.e., no US) is maximized,

which facilitates learning according to error correction models (e.g., Rescorla & Wagner, 1972) and 2) when the CS is presented with another excitatory stimulus, fear responding increases and this itself might generate more extinction learning.

There is evidence that increased responding alone may optimize learning during extinction. This evidence comes from investigations involving adrenergic neurotransmission. The adrenergic system has been implicated in the acquisition and consolidation of emotional memories in animals (Cahill, Pham, & Setlow, 2000) and humans (Nielson & Jensen 1994). In fact, adrenergic hyperactivity has been implicated in anxiety disorders (e.g., Southwick, Morgan, Charney, & High, 1999). Thus, Cain and colleagues (2004) hypothesized that enhanced adrenergic transmission would retard extinction learning. In other words, they predicted that enhanced arousal and elevated fear responding during extinction would lead to less learning during extinction. To test this, they administered yohimbine (which blocks autoinhibitory α_2 adrenergic receptors and is anxiogenic in humans) prior to extinction. Their results indicated the opposite effect – administration of yohimbine *facilitated* extinction learning. The authors posited several possible mechanisms for these results including that increased adrenergic activity promotes consolidation of emotional memories (Cahill, Prins, Weber, & McGaugh, 1994; Cahill et al., 2000; Nielson & Jensen, 1994) so it is possible that administration of yohimbine facilitated consolidation of extinction memories. Thus, enhanced arousal and increased fear responding during extinction may itself enhance extinction learning.

In fact, some studies have demonstrated that procedures designed to maintain arousal throughout exposure may facilitate long-term fear reduction. For example, pharmacological procedures which inhibit arousal, if administered during exposure, have

been shown to impede extinction learning in rats (Berman & Dudai, 2001). The findings of Cain et al. (2004), taken together with those presented by Rescorla (2000), suggest that greater arousal and elevated fear responding may actually facilitate learning during exposure therapy. The association between elevated fear responding during extinction training and greater fear reduction at retest is at odds with the tenets of Emotional Processing Theory (EPT; Foa & Kozak, 1986; Foa & McNally, 1996), a widely used model for exposure therapy. EPT posits that both within-session habituation and between-session habituation of fear are necessary for long-term fear reduction. However, these premises of EPT have not been well supported by the evidence (for review, see Craske et al., 2008).

The goal of the present study was to directly investigate whether the presence of additional excitatory and arousing stimuli throughout exposure facilitates long-term fear reduction. It was expected that the presence of such stimuli would lead to elevated fear responding and enhanced arousal throughout exposure, thus providing a test of whether maintaining elevated fear throughout exposure facilitates long-term fear reduction. This investigation utilized a clinical analog sample of participants highly fearful of public speaking. Two excitatory stimuli were chosen to be presented during exposure exercises; participants were exposed to both of the excitatory stimuli concurrently throughout exposure, both of the excitatory stimuli but sequentially throughout exposure, or none of the excitatory stimuli. Based on the findings of Rescorla (2000) and Cain et al. (2004), participants exposed to no excitors were hypothesized to demonstrate more return of fear at follow-up testing as compared with those participants who were presented with excitors (concurrently or sequentially) during exposure. Additionally, participants

presented with the two excitors sequentially during exposure were hypothesized to demonstrate less return of fear at follow-up testing as compared with participants presented with the two excitors concurrently. This is due to the expectation that the novelty of adding an excitor will cause an increase in arousal; thus, participants in the sequential excitors group will experience more increases in arousal since the second excitor is added in halfway through each speech versus those in the concurrent excitor group who are presented with both excitors throughout each exposure exercise.

Method

Design

Participants fearful of public speaking were randomly assigned to one of three experimental groups: Concurrent [C], n = 21; Sequential [S], n = 20; or No [N], n = 18 excitors during exposure. These three groups were compared across three time points: Baseline (prior to exposure), Test-1 (immediately following exposure), and Test-2 (one week following exposure).

Participants

Fifty-nine participants (44 females, 15 males), highly fearful of public speaking, with a mean age of 19.5 (range 18 to 25), were recruited through mass testing sessions from several Introduction to Psychology classes. Participants were from a variety of ethnic backgrounds: 35.6% Asian, 25.4% Caucasian, 15.3% Latino, 10.1% Pacific Islander, 5.1% Black/African-American, 5.1% Biracial, and 3.4% Other. Participants were screened using two items, assessing how anxious they would feel giving a formal speech before a live audience and how likely they would be to avoid taking a class that required an oral presentation, each rated on a 0-8 point scale where 0 = none/never and 8

= extremely/always. Participants were recruited if they scored 6 or higher (strongly to extremely) in response to both items. This 2-question survey has been used previously to recruit participants highly fearful of public speaking (Tsao & Craske, 2000). Exclusion criteria for study participation included any heart, respiratory, or neurological problems, as well as scoring above 18 on the Beck Depression Inventory (BDI; Beck, Steer, & Garbin, 1988).

Measures

Self Report Questionnaires

Two self report measures were completed at Baseline to assess group differences: the Self-Statements during Public Speaking Scale (SSPS; Hofmann & DiBartolo, 2000) and the Personal Report of Confidence as a Speaker (PRCS; Paul, 1966). The SSPS is a 10-item questionnaire that asks respondents to imagine how they feel in public speaking situations. It is comprised of five positive self-statements, summed to obtain the positive subscale score (SSPS-P), and five negative self-statements, summed to obtain the negative subscale score (SSPS-N). Each subscale has demonstrated high internal consistency, SSPS-P = 0.84, SSPS-N = 0.83 and good test-retest reliability (r = 0.78 and r = 0.80; Hofmann & DiBartolo, 2000). In a sample of 100 undergraduate students ranging in age from 17 to 23 years, means on the SSPS-P and SSPS-N were 15.4 and 7.9, respectively; in a sample of 41 outpatients ranging in age from 19 to 50 diagnosed with Social Phobia using the Anxiety Disorders Interview Schedule for DSM-IV, means on the SSPS-P and SSPS-N were 13.4 and 12.3, respectively (Hofmann & Dibartolo, 2000).

The PRCS assesses confidence in speaking ability and has demonstrated high internal consistency (r = 0.91) and good convergent validity, correlating positively with a

number of other measures of public speaking and social anxiety, r = 0.52-0.97 (Daly, 1978). Paul (1966) recommended using 16 as the clinical cutoff score based on a mean of 20.6 in a clinical sample and 11.6 in a nonclinical sample. However, a more recent report suggests that a score of 17 or 18 may be a more appropriate clinical cutoff for the PRCS in a sample of undergraduate students (Phillips, Jones, Rieger, & Snell, 1997).

Behavioral Avoidance Test

A Behavioral Avoidance Test (BAT) was completed at Baseline, Test-1, and Test-2. In each case, participants gave a speech with ongoing measurement of physiology (heart rate) and subjective fear recorded at 0, 1, and 2 minutes. The speech was up to two minutes in length; participants were instructed to speak for as long as they could but to stop when they wanted to end the speech, thus providing a behavioral index of duration. Participants drew two topics at random (out of a bag of topics) and selected one topic to present; then, they stood in front of the experimenter and began the speech.

Subjective fear was monitored with the Subjective Units of Distress scale (SUDS; Wolpe, 1973); a 100-point scale, where 0 = no fear, 25 = mild fear, 50 = moderate fear, 75 = severe fear, and 100 = very severe fear. Participants were asked for their SUDS rating at 0, 1, and 2 minutes during each BAT. If participants chose to speak for less than two minutes, they were asked to provide a SUDS rating at the completion of their speech. These values were used to calculate mean-SUDS (average across all ratings) and max-SUDS (highest of all ratings) for each BAT.

Heart rate (beats per minute) was recorded continuously throughout the BAT using a Polar S610i heart rate monitor. This monitor provides wireless heart rate monitoring with ECG-accurate continuous measurement, sampling once every five

seconds. It consists of an elastic belt that attaches around the chest of the participant and a wrist-watch receiver that records the heart rate data. A five-minute resting heart rate was used to calculate difference scores (i.e., the difference in mean heart rate during resting and mean heart rate during the BAT).

Exposure Process Measures

SUDS ratings were recorded at 0, 1, 2, 3, 4, and 5 minutes of each of the four exposure speeches given during a single exposure day. Also, heart rate was recorded continuously throughout exposure; heart rate change scores were calculated by taking the difference between mean heart rate during each exposure and mean heart rate at resting.

Procedure

The study consisted of two sessions. At the start of Session 1, participants completed the informed consent form. Next, participants were given the first set of questionnaires (BDI, SSPS, and PRCS). Participants were familiarized with the 0-100 point SUD scale and then fitted with the heart rate monitor. Next, heart rate was recorded for a five-minute resting period during which participants were instructed to "please sit quietly and remain still" and left alone in the room. Upon returning to the room, the experimenter gave instructions for the Baseline BAT. After the BAT, participants were given a two-minute rest period.

For the exposure phase, the experimenter explained to participants that they would be giving speeches to the experimenter. Participants completed four five-minute exposure speeches with two-minute inter-trial intervals. For participants in the C group, two excitors were presented throughout each speech: 1) participants wore a tightened workman's belt designed to elevate arousal by causing a sensation of tightness around the

abdomen[2] and 2) participants completed the speeches in front of a video camera which was connected to a monitor on which the participants viewed themselves and were told that their speeches were being recorded so that observers could later rate how well they did. Participants in the S group began each speech with one of these excitors present (the belt or video camera) and, halfway through each speech, the second excitor was introduced. Order was counterbalanced such that, for half of the participants in the S group, the video camera was present during the first half of speeches 1 and 3 whereas, for the other half, the video camera was present during the first half of speeches 2 and 4. Participants in the N group completed all exposure speeches with no excitors present.

For each speech, participants selected three topics out of a bag (using a different pool of topics than for the BAT speeches) and chose one to present. Then, participants stood and gave a five-minute speech; participants who stopped prematurely were prompted by experimenters to continue speaking, even if that meant repeating what had already been said. Participants provided SUDS ratings at the start of each exposure speech and at one-minute intervals throughout each speech. (The experimenter prompted the participant to make these ratings by asking "SUDS?" and participants were told ahead of time to respond quickly with their SUDS rating each time the experimenter asked and then to continue speaking). At the conclusion of each speech, participants were given a one-minute break before selecting another topic and completing the next speech.

[2] The belt was designed to increase interoceptive awareness as participants felt pressure around the abdomen and experienced difficulty taking a deep breath. There is evidence that socially anxious individuals perceive physical arousal accurately (Stevens et al., 2011) and that they exhibit an attentional bias toward cues of internal sources of potential threat (Pineles & Mineka, 2005). Thus, by increasing interoceptive awareness, it was expected that participants would accurately perceive heightened physiological arousal and that this arousal would provide evidence for a threat which would lead to heightened anxiety during the exposure task.

Following completion of exposure, participants were instructed to sit and relax for one minute.

Participants then completed the Test-1 BAT. The procedure was identical to the Baseline BAT, except that the topic chosen for the Baseline BAT speech was excluded from the topics bag. Upon completion of the Test-1 BAT, the heart rate monitor was removed and Test-2 was scheduled, 6 to 8 days following Test-1.

For Test-2, the experimenter escorted participants to the same laboratory room where they completed the second set of questionnaires (SSPS and PRCS). Next, participants were fitted with the heart rate monitor and another five-minute resting period was recorded before they were re-familiarized with the SUD scale. The Test-2 BAT was identical to the previous two BATs except that topics chosen in prior BATs were removed.

Results

Baseline

Means on both subscales of the SSPS confirmed that the screening measure was effective in selecting a sample of participants highly fearful of public speaking (SSPS-P = 12.93; SSPS-N = 12.19), although the mean on the PRCS (7.38) was not above the clinical cutoff score. Participants were only included if they reported a fear level of at least 40 on the SUD scale during the Baseline BAT; the average maximum SUDS rating during the Baseline BAT was 59.78 (on a 0-100 scale). Mean heart rate at resting prior to the Baseline BAT (77.15 bpm) was significantly lower than mean heart rate during the Baseline BAT (95.39 bpm) across all groups ($F(1,49) = 174.68$, $p < 0.001$, $\eta = 0.78$).

One-way ANOVAs revealed no significant differences among the three groups on the BDI ($F(2,56) = 1.69$, $p = 0.20$) or the negative subscale of the SSPS ($F(2,56) = 0.18$, $p = 0.84$). However, there were significant group differences on the PRCS ($F(2,56) = 3.59$, $p < 0.05$) and the positive subscale of the SSPS ($F(2,56) = 3.24$, $p < 0.05$). A series of independent-samples t-tests indicated that, on the PRCS, the concurrent excitors [C] group scored significantly higher than the sequential excitors [S] group ($t(55) = 2.71$, $p = .01$) and, on the SSPS-P, the no excitors [N] group scored significantly higher than the sequential excitors [S] group ($t(55) = 2.87$, $p < .01$). Therefore, scores on the SSPS-P and PRCS were entered as covariates in all subsequent analyses.

There were no significant differences among the groups at Baseline in self-reported or physiological fear (mean-SUDS: $F(2, 58) = 0.94$, $p = 0.40$; max-SUDS: $F(2,58) = 0.31$, $p = 0.74$; heart rate: $F(2,35) = 0.06$, $p = 0.94$).

Subjective Measures

A 3 (Group: C, S, N) x 3 (Time: Baseline, Test-1, Test-2) repeated measures ANOVA for max BAT SUDS[3] scores revealed a significant effect of Time ($F(2,56) = 23.55$, $p < 0.001$, $\eta^2 = 0.30$) but no significant effect for Group ($F(2,56) = 1.62$, $p = 0.21$, $\eta^2 = 0.06$) or Group x Time interaction ($F(4,56) = 0.75$, $p = 0.56$, $\eta^2 = 0.03$). Examination of means indicated max-SUDS scores decreased from Baseline to Test-1, ($F(1,56) = 24.23$, $p < 0.001$, $\eta^2 = 0.30$) but remained unchanged from Test-1 to Test-2, ($F(1,56) = .18$, $p = 0.68$, $\eta^2 = 0.00$; see Table 1).

[3] Because max-SUDS was highly correlated with mean-SUDS for all BAT speeches (BAT 1: $r(57) = 0.95$, $p < 0.000$; BAT 2: $r(57) = 0.97$, $p < 0.000$; BAT 3: $r(57) = 0.98$, $p < 0.000$) and because all results obtained with max-SUDS were also obtained with mean-SUDS, only results of max-SUDS analyses are reported.

Behavioral Measures

A 3 (Group: C, S, N) x 3 (Time: Baseline, Test-1, Test-2) repeated measures ANOVA for BAT durations revealed a significant effect of Time ($F(2,56) = 42.94$, $p < 0.001$, $\eta^2 = 0.43$) but no significant effect for Group ($F(2,56) = 0.18$, $p = 0.84$, $\eta^2 = 0.001$) or Group x Time interaction ($F(4,56) = 0.53$, $p = 0.59$, $\eta^2 = 0.02$). Examination of means indicated BAT durations increased from Baseline to Test-1, $F(1,56) = 74.10$, $p < 0.001$, $\eta^2 = 0.57$), and decreased from Test-1 to Test-2, $F(1,56) = 10.89$, $p < 0.01$, $\eta^2 = 0.16$; see Table 1.

Physiological Measures

A 3 (Group: C, S, N) x 3 (Time: Baseline, Test-1, Test-2) repeated measures ANOVA for heart rate change scores revealed a significant effect of Time ($F(2,30) = 30.99$, $p < 0.001$, $\eta^2 = 0.51$) but no significant effect for Group ($F(2,30) = 0.19$, $p = 0.83$, $\eta^2 = 0.01$) or Group x Time interaction ($F(4,30) = 1.39$, $p = 0.27$, $\eta^2 = 0.09$; Table 1).

		Group		
		C	**S**	**N**
Baseline:	**Mean SUDS**	50.59 (12.35)	46.73 (14.12)	52.69 (14.66)
	Max SUDS	59.52 (13.31)	58.20 (14.92)	61.83 (14.91)
	Heart Rate Change	17.73 (6.20)	19.00 (9.34)	19.77 (14.02)
	Duration (sec)	91.67 (28.08)	88.05 (20.82)	88.50 (28.54)
Test-1:	**Mean SUDS**	41.97 (17.78)	33.84 (20.51)	42.98 (20.64)
	Max SUDS	48.67 (20.86)	38.45 (22.16)	49.89 (22.41)
	Heart Rate Change	8.81 (4.21)	10.56 (7.75)	8.48 (6.24)
	Duration (sec)	116.95 (9.85)	120 (0)	120 (0)
Test-2:	**Mean SUDS**	39.33 (39.33)	33.60 (19.13)	42.87 (17.91)
	Max SUDS	45.67 (14.86)	39.20 (21.45)	49.72 (20.25)
	Heart Rate Change	19.96 (5.19)	18.11 (5.59)	14.45 (7.48)
	Duration (sec)	106.24 (25.06)	109.75 (17.70)	113.89 (18.52)

Table 1: Mean subjective fear, physiological fear (change scores in heart rate from resting period), and BAT speech duration in each group during Baseline, Test-1, and Test-2 BAT tasks. Standard deviations are included in parentheses.

Examination of means indicated heart rate change scores decreased from Baseline to Test-1, $F(1,30) = 49.87$, $p < 0.001$, $\eta^2 = 0.52$), and increased from Test-1 to Test-2, $F(1,30) = 9.47$, $p < 0.01$, $\eta^2 = 0.17$).

Exposure Process Measures

One-way ANOVAs revealed no significant differences among the three groups on subjective ($F(2,56) = 0.24$, $p = 0.79$) or physiological ($F(2,30) = 0.75$, $p = 0.48$) fear during the exposure speeches.

		Group		
		C	**S**	**N**
Exposure Speech 1:	**Mean SUDS**	54.18 (17.78)	50.23 (13.45)	45.08 (19.63)
	Max SUDS	55.58 (24.57)	60.18 (21.09)	47.86 (23.04)
	Heart Rate Change	11.82 (5.33)	13.70 (8.52)	12.88 (8.51)
Exposure Speech 2:	**Mean SUDS**	42.40 (19.15)	39.12 (19.62)	43.76 (20.96)
	Max SUDS	45.45 (19.66)	45.00 (22.11)	44.00 (23.10)
	Heart Rate Change	10.63 (5.02)	12.41 (8.22)	12.90 (8.42)
Exposure Speech 3:	**Mean SUDS**	41.47 (19.29)	34.44 (16.26)	44.85 (21.14)
	Max SUDS	43.16 (22.25)	41.54 (19.30)	42.39 (24.32)
	Heart Rate Change	10.17 (5.23)	10.79 (8.36)	11.67 (8.21)
Exposure Speech 4:	**Mean SUDS**	40.96 (19.46)	35.22 (22.09)	40.37 (20.60)
	Max SUDS	43.00 (22.76)	40.04 (23.08)	40.71 (21.69)
	Heart Rate Change	9.78 (5.48)	11.19 (9.00)	10.75 (8.51)

Table 2: Mean subjective fear and physiological fear (change scores in heart rate from resting period), during each of the exposure speeches.

The relationship between exposure process variables and dependent measures at Test-2 was examined using a stepwise multiple regression model. Thus, these analyses allowed evaluation of the degree to which outcomes were predicted by subjective and physiological exposure process measures above and beyond the effects of baseline values and group status. The first step entered the baseline value of each dependent measure and baseline scores on the SSPS-N and PRCS. The second step entered Group. The third

step contained the exposure process variables using forward selection: 1) initial fear activation (max-SUDS during the first exposure speech), 2) within-session habituation (max-SUDS during the first exposure speech minus the SUDS rating at end of last exposure speech), 3) fear level at the end of exposure (SUDS rating at minute 5 of Exposure Speech 4), and 4) emotional variability (the standard deviation of SUDS ratings across all four exposure speeches; which was found to be a significant predictor of outcome in previous investigations, Kircanski et al., in submission). A second set of regression analyses was then conducted utilizing the same method but investigating the relationship between physiological exposure process variables and dependent measures at Test-2. All methods were identical as above; however, in these analyses, initial fear activation was operationalized as the maximum heart rate score during the first exposure speech; within-session habitation was operationalized as the difference between the maximum heart rate score during the first exposure speech and the last heart rate score; ending fear level was operationalized as the last heart rate score during the fourth exposure speech, and emotional variability was operationalized as the standard deviation of all heart rate scores throughout all four exposure speeches.

Self-Report Measures

No exposure process variables were significant predictor of SSPS-P or SSPS-N at Test-2. For Test-2 PRCS, higher heart rate scores at the end of exposure predicted lower scores, R^2 change = 0.11, standardized $\beta = -0.48$, $p < 0.05$. No other exposure process variables were significant predictors of post-PRCS scores.

BAT Subjective Measures

Greater emotional variability throughout exposure predicted lower mean-SUDS at Test-2, R^2 change = 0.17, standardized β = -0.42, $p < 0.001$. (Please see Figure 1 for an illustration of high emotional variability during exposure versus low emotional variability during exposure). No other exposure process variables were significant predictors of mean-SUDS at Test-2.

Similarly, for Test-2 max-SUDS, greater emotional variability throughout exposure predicted lower scores, R^2 change = 0.19, standardized β = -0.46, $p < 0.001$. No other exposure process variables were significant predictors of max-SUDS at Test-2.

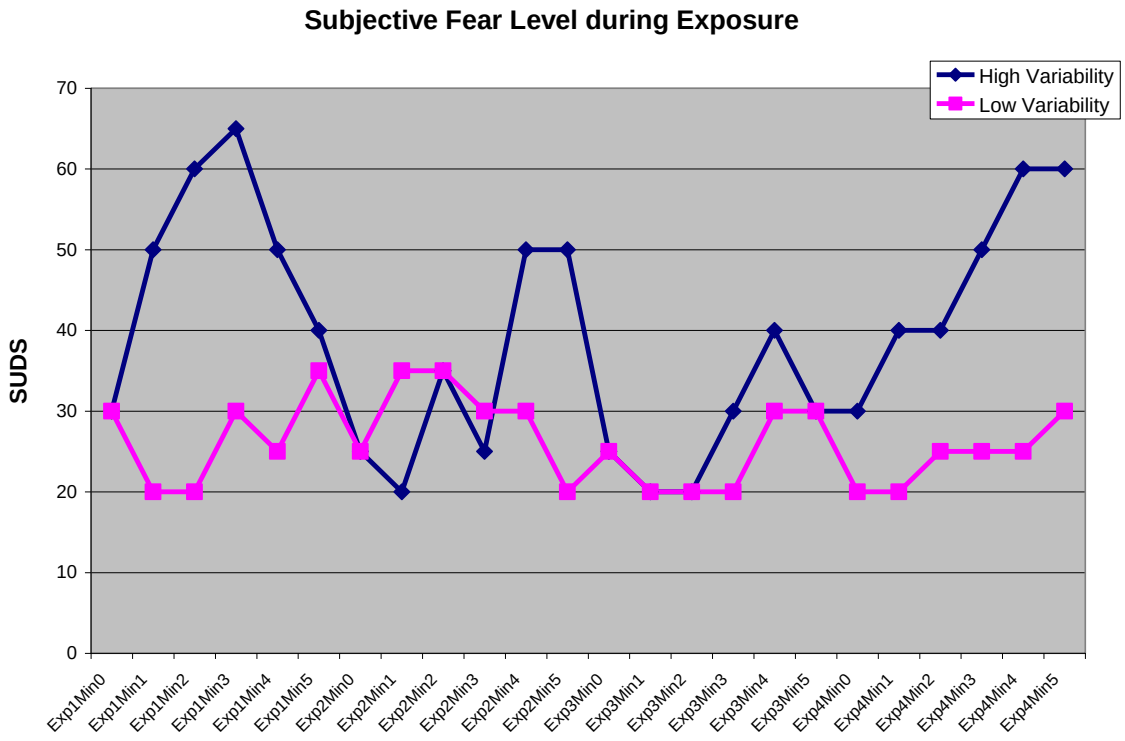


Figure 1: Subjective fear responding during exposure exercises for a participant who demonstrated high emotional variability versus a participant who demonstrated low emotional variability.

BAT Behavioral Measures

Less within-session habituation (the difference between SUDS rating at Minute 0 of Exposure Speech 1 and SUDS rating at Minute 5 of Exposure Speech 4) predicted higher Test-2 durations, R^2 change = 0.06, standardized $\beta = -0.26$, $p < 0.05$. No other exposure process variables were significant predictors of speech duration at Test-2.

BAT Physiological Measures

No exposure process variables were significant predictors of physiological fear responding at Test-2.

Discussion

These results partially supported the study hypotheses. The excitatory stimuli used in the current design did not produce distinct groups in regards to fear arousal throughout exposure. We had predicted that the participants presented with no excitors would demonstrate the lowest level of fear responding during exposure, the group presented with concurrent excitors would demonstrate a significantly higher level of fear responding during exposure, and that the group presented with sequential excitors would demonstrate the significantly highest level of fear responding during exposure. The excitors used in this study failed to produce such groups; in other words, we were unable to manipulate level of fear responding and arousal during exposure. However, some participants did demonstrate sustained fear throughout exposure; in other words, fear responding did not habituate across exposure. And this lack of habituation *facilitated* learning during exposure. In addition, some participants demonstrated higher emotional variability in fear responding during exposure and this variability also facilitated long-term fear reduction.

There are several possibilities for why the excitatory stimuli used in the current design failed to enhance arousal. First, the stimuli used (the video camera and workman's belt) may not have been sufficiently excitatory. Indeed, there were no group differences in mean SUDS or heart rate during exposure speeches suggesting that the excitatory stimuli failed to produce the intended effects. Second, in the present study, the excitatory stimuli were designed to elevate arousal but not necessarily expectancy of the US. If the excitatory stimuli had had associative value with the US, they would have presumably increased US-expectancy thereby maximizing discrepancy between expectation and reality to enhance learning. Third, it is possible that the exposures did not maximize fear levels. The screening measure assessed participants' fear of giving speeches in front of a live audience. However, the exposures and BATs in this study entailed giving a speech in front of the experimenter. This may have lead to moderate levels of fear during exposure speeches. Mean SUDS during the four exposure speeches, which ranged from 38.83 to 50.07 representing only up to moderate levels of fear, provide some support for this possibility. On the other hand, physiological fear was elevated throughout exposure speeches, as reflected by mean heart rate change scores ranging from 10.62 to 12.87. Had additional audience members been present, the exposures may have been more challenging leading to a broader range in fear responses which would have allowed the excitatory stimuli to produce the intended effects. Lastly, this study was limited to all exposures on the same day. Multiple exposure days would have allowed for analysis of both within-session and between-session changes in fear responding.

On the other hand, the analyses of predictors of outcome from exposure process variables suggest that emotional variability during exposure exercises and lack of habituation throughout exposure may actually facilitate long-term fear reduction. Since baseline values, group status, and all exposure process variables were entered into the regression analyses, this was an especially stringent test. Thus, the fact that emotional variability and less within-session habituation predicted better outcomes provides strong evidence that these factors are important in successful exposure therapy.

In general, the results of this experiment did not support the tenets of emotion processing theory (EPT). Initial fear activation was not a predictor of outcome in any of the analyses. Whether measured subjectively or physiologically, initial fear activation did not predict subjective, physiological, or behavioral fear at Test-2. Within-session habituation was not a predictor of outcome with regards to subjective or physiological measures of fear. Within-session habituation was a significant predictor of outcome in regards to the behavioral measure of fear (BAT duration); however, in the opposite direction of that predicted by EPT. Less within-session habituation predicted *longer* BAT durations at Test-2. Also, higher heart rate at the end point of exposure significantly predicted *lower* scores on the Test-2 PRCS completed one week later.

The fact that elevated fear levels at the end of exposure predicted less fear at follow-up testing is in line with a body of literature demonstrating the effectiveness of exposure therapy when each exposure trial is terminated at the point of elevated or unduly high anxiety (e.g., Emmelkamp & Mersch, 1982; Rachman, Craske, Tallman, & Solyom, 1986). In addition, several studies have shown that lower fear at the completion of exposure does not predict long-term fear reduction. For example, Rachman, Robinson,

& Lopatka (1987) found that those who continued a single exposure trial until fear reduced by 100% from baseline showed more return of fear four weeks later than those whose exposure was discontinued when fear had reduced by 50% from baseline. The present results provide further evidence that habituation across exposure and low levels of fear at the end of exposure are not crucial for long-term fear reduction. In fact, as posited by Cain et al. (2004), enhanced arousal may actually facilitate the formation of memories thereby enhancing learning during exposure. However, recent findings in animal models have not supported the idea that enhanced arousal may facilitate extinction learning. Some investigators have been unable to replicate the Cain et al. (2004) findings (e.g., Mueller, Olivera-Figueroa, Pine, & Quirk, 2009) while others have replicated it but found that the yohimbine effect actually depended on subtle context learning (Morris & Bouton, 2007). For a full review of the current literature regarding noradrenergic effects of fear extinction, please see Mueller and Cahill, 2010. In addition, other recent animal extinction experiments have found that high levels of responding during extinction predict increased rates of renewal and spontaneous recovery (for a full review, see Bouton & Woods, 2008). Thus, the current research in animal models indicates that enhanced fear responding may not facilitate extinction learning which is somewhat at odds with the present results indicating that sustained fear responding across exposure may facilitate extinction learning. Clearly, more research is needed on this topic; however, the present results do provide evidence that, at the least, habituation during exposure exercises is not crucial for extinction learning to occur as was posited by EPT.

The present results also indicate the potential importance of variability in subjective fear during exposure in facilitating learning in the long-term. Several possible

mechanisms may account for this. One possibility is greater emotional variability during exposure allows for a variety of internal contexts to become associated with extinction learning. Mystkowski, Mineka, Vernon, & Zinbarg (2003) have demonstrated that internal states can serve as exposure contexts. Thus, if multiple internal states are associated with the learning that occurs during exposure, retrievability of extinction learning can be facilitated thereby reducing the possibility of return of fear. Similarly, the multiple internal contexts may function as retrieval cues. As posited by Bouton (1993), extinction involves new inhibitory learning (CS-no UCS) which then competes with the original CS-UCS memory. When the CS occurs in a context other than the extinction context, retrieval of the original CS-UCS memory may cause return of fear; retrieval cues associated with the extinction context can offset such return of fear. If internal states can function as retrieval cues, participants who experienced greater emotional variability throughout exposure would have more retrieval cues associated with the extinction context than participants who did not experience as much emotional variability during exposure; thus making the CS-no UCS association more easily retrievable and reducing the likelihood of return of fear.

Another mechanism by which emotional variability may enhance exposure learning is by providing more opportunities for corrective learning to occur. According to the Rescorla-Wagner model (Rescorla & Wagner, 1972), learning is proportional to how surprised the individual is (i.e., the discrepancy between expectation and reality) and there is recent evidence to support this error-correction model of extinction learning (e.g., Rescorla, 2006). During the first few trials of extinction, this discrepancy is high, which facilitates learning. However, this learning in turn decreases expectation of the negative

event, thereby reducing the surprise factor. Once individuals no longer expect an adverse event, they are no longer surprised when the adverse event does not occur and, therefore, no longer learn anything new about the CS. Perhaps those participants who experienced greater emotional variability during exposure were actually demonstrating fluctuations in their expectancies of a negative event. If this were the case, these individuals were giving themselves repeated opportunities to learn about the CS. Although we cannot be certain that this occurred, it is a possible mechanism by which emotional variability may enhance exposure learning. In future investigations, it will be important to examine whether the presence of excitatory stimuli which are associated with the US enhance learning during extinction.

This study had several limitations including the use of a clinical analog sample, although participants demonstrated subjective and physiological fear comparable to clinical samples. However, the results do indicate that some participants maintained elevated fear throughout exposure whereas others did not and that some participants experienced more emotional variability throughout exposure than did others. Although we do not know what caused these differences, we can conclude that sustained fear throughout exposure and elevated fear at the end of exposure is not detrimental to, and may actually enhance, long-term fear reduction. In addition, the present results indicate that greater emotional variability throughout exposure enhances the long-term effects of exposure therapy.

DISCUSSION

Summary of Results

This set of studies provides important information regarding methods for enhancing learning during extinction training. The results of Study 1 indicate that compound stimulus presentations during extinction can enhance learning and provide protection against spontaneous recovery and reinstatement effects. In addition, the results provide support for the hypothesis that the mechanism by which compound extinction trials enhance learning is most likely enhanced associative change rather than enhanced responding alone. Participants who ingested caffeine prior to extinction training and did not receive compound extinction trials did show some protection against spontaneous recovery effects indicating that enhanced responding alone may enhance extinction learning to some degree. However, caffeine ingestion prior to extinction training provided no protection against reinstatement effects whereas compound extinction trials *did*. Thus, enhanced responding alone during extinction did not predict attenuation of reinstatement whereas enhanced associative change (experienced by participants presented with compound extinction trials) did.

The results of Study 2 provide evidence that occasional reinforced trials during extinction protect against spontaneous recovery and rapid reacquisition effects. Participants who experienced some CS-US pairings during extinction demonstrated elevated fear responding throughout extinction but no significant changes in physiological or subjective fear responding across the one week period between extinction and test. In contrast, participants who experienced no CS-US pairings during extinction demonstrated a decline in fear responding across extinction and low levels of

fear at the end of extinction but significant *increases* in physiological and subjective fear responding across the one week period between extinction and test. In other words, participants who experienced no CS-US pairings during extinction demonstrated significant spontaneous recovery effects while participants who experienced occasional CS-US pairings during extinction did not. In addition, occasional reinforced trials during extinction predicted significantly slower rates of reacquisition.

Lastly, the results of Study 3 indicate that sustained fear responding throughout exposure exercises provides protection against spontaneous recovery effects. This finding is consistent with the finding in Study 1 that caffeine ingestion elevated fear responding during extinction training but also provided some protection against spontaneous recovery. Thus, it does seem that enhanced responding alone can facilitate learning during extinction or exposure exercises to some degree. In addition, Study 3 provides evidence that high emotional variability during exposure provides protection against spontaneous recovery effects.

Enhanced Associative Change

The results of Study 1 provide strong support for the prediction made by the Rescorla-Wagner model (Rescorla & Wagner, 1972) that elevating US-expectation during extinction training will maximize surprise and enhance learning. Two CSs each underwent some extinction training on their own and were then paired together for compound extinction trials. When the two CSs were first paired together, subjective and physiological fear responding significantly increased confirming the predictions of the Rescorla-Wagner model. This compound stimulus then underwent further extinction training. Although physiological fear responding remained elevated in the compound

extinction groups, both compound groups showed significantly less physiological fear responding at spontaneous recovery one week later compared with participants in the Single Placebo group. The reinstatement test revealed that the Compound Placebo group demonstrated significantly less physiological fear responding and significantly lower US-expectancy ratings to CSA than participant in the Single Placebo group.

These results indicate the importance of enhanced associative change. Caffeine provided some protection from spontaneous recovery effects but it did not provide protection against reinstatement effects and, as predicted, ingestion of caffeine did not elevate US-expectancy ratings during extinction training. Caffeine did, however, elevate skin conductance responses during extinction. Thus, caffeine elevated physiological fear responding without increasing expectation of the US. The finding that compound trials predict lower fear responding following a test of reinstatement compared with caffeine provides strong evidence for the role of enhanced associative change in maximizing learning during extinction.

The results of Study 3 may provide further evidence for the importance of enhancing discrepancy between expectation and reality during extinction training. One of the findings of this study was that high emotional variability predicted less fear responding at spontaneous recovery. It is not known what led to fluctuations in subjective fear and why some participants demonstrated more fluctuations (i.e., higher emotional variability) than others. However, one possibility is that this variability in participants' subjective fear responding actually reflected fluctuations in their expectancy of a negative outcome. For example, if participants somehow detected an increased risk for a negative evaluation, this would represent an increase in US-expectancy (since the

US in this case is negative evaluation, rejection, or judgment). Then, when the US did not occur, surprise was maximized and learning enhanced.

Although we cannot be certain this occurred, there is evidence that individuals with high social anxiety more accurately indentify facial expressions (irrespective of valence or race) than individuals with low social anxiety (Hunter, Buckner, & Schmidt, 2009). These findings are in line with a body of literature demonstrating that social anxiety is associated with selective attention toward social anxiety cues (e.g., Rapee & Heimberg, 1997). For example, in the face-in-the-crowd paradigm where individuals are asked to detect an angry, happy, neutral, or disgust target face in a crowd of twelve distracter photographs, those with social anxiety disorder exhibited greater attentional biases for angry than for happy faces in a neutral crowd (Gilboa-Schechtman, Foa, & Amir, 1999) compared with nonanxious controls. In addition, individuals with social anxiety were more slowed down in their performance by happy and angry versus neutral distracter photographs whereas nonanxious controls did not exhibit this sensitivity.

Thus, it is conceivable that participants in Study 3 selectively attended to social anxiety cues (e.g., subtle changes in facial expression exhibited by the experimenter) and accurately detected any signs of disapproval or judgment by the experimenter. Of course, the experimenter in Study 3 attempted to maintain a completely neutral facial expression throughout the exposure speeches. However, it is certainly possible that the experimenter sometimes reacted to the participant's speech, especially since the content of many of the topics were somewhat emotionally charged. In addition, participants who stopped prematurely (prior to the end of the five-minute interval for each exposure speech) were given a standardized prompt ("Please continue speaking and I will let you know when to

stop"). If participants stated they had nothing more to say on the topic, they were instructed to repeat what they had already said. If participants deviated from the topic at any point during an exposure speech, experimenters were instructed to say "Please do not deviate from the topic; you may repeat what you have already said but you must remain on the topic." Such verbal feedback and/or inadvertent feedback in the form of subtle changes in experimenters' facial expressions during the speeches may have increased participants' expectation of a negative evaluation (e.g., the experimenter saying "Your speech is not very good" or "I really do not like what you are saying") and, when that did not happen (i.e., the US did not occur), surprise was maximized and learning enhanced. Unfortunately, experimenters in Study 3 were not asked to record the number of times they had to prompt a participant to continue the speech and/or stay on topic. If this data were available, the relationship between such promptings and participants' level of emotional variability could have been examined.

Lastly, the results of Study 2 may also provide support for the effectiveness of elevating US-expectancies during extinction even though the Rescorla-Wagner model does not predict that extinction learning will be optimized with occasional CS-US pairings. The results of Study 2 did indicate that participants in the Reinforced group demonstrated significantly higher US-expectancy ratings throughout extinction than participants in the Control group. Thus, expectancy ratings fluctuated throughout extinction and remained elevated which, according to basic tenet of the Rescorla-Wagner model, should optimize learning by enhancing surprise. However, the Rescorla-Wagner model also predicts that associative strength between the CS and US will be strengthened during each CS-US pairing and, therefore, the overall decrement in predictive value of

the CS will be no different in the Control versus Reinforced group. In other words, even though US-expectancies remain elevated, the fact that the US does sometimes occur following the CS will not enhance overall inhibitory CS-US learning. One model that may predict enhanced associative change utilizing this paradigm is Wagner's SOP model (Wagner, 1981). Similar to the CS preexposure effect, if memory representations of the CS remained in A2 when a CS-US presentation occurred, the excitatory relationship between the CS and US would not be enhanced since memory representations cannot be activated from A2 to A1. However, since US-expectancy ratings in humans are thought to be determined both by conscious processes as well as automatic associative processes (McLaren, Green, & Mackintosh, 1994), expectation of the US may have increased following a CS-US pairing even if the associative excitatory CS-US relationship was not enhanced. Therefore, it is possible that occasional reinforced trials during extinction led to elevated expectation of the US throughout extinction without enhanced learning of the CS-US excitatory relationship. If this was the case, occasional reinforced trials during extinction could lead to enhanced associative change (i.e., enhanced inhibitory learning regarding the CS).

Thus, procedures for enhancing expectation of a negative outcome facilitate extinction learning by maximizing surprise between expectation and reality. The resulting enhanced associative change provides protection against future fear recovery effects.

Enhanced Responding during Extinction

Across all three of these studies, elevated fear responding throughout extinction predicted significantly lower recovery of fear at test. In Study 1, both caffeine ingestion

and presentation of compound extinction trials increased fear responding during extinction. At reinstatement, only compound trials predicted less fear indicating the impact of enhanced associative change. However, caffeine ingestion did provide some protection against spontaneous recovery (as measured by heart rate means and subjective valence ratings). Thus, the results of Study 1 indicate that elevated fear responding *alone* during extinction does predict less fear recovery at spontaneous recovery as measured by some, but not all, indices of fear responding. This is consistent with the results of Study 3 which indicated that sustained subjective fear responding throughout exposure (i.e., less change in SUDS ratings from the start of the first exposure to the end of the last exposure) predicted less fear responding at spontaneous recovery (as evidenced by longer speech durations). Lastly, the results of Study 2 cannot rule out the possibility that increased responding enhanced extinction learning. Participants in the reinforced group demonstrated significantly higher subjective and physiological fear responding throughout extinction than those in the Control group. Thus, it is possible that this enhanced responding played a role in why participants in the reinforced group demonstrated significantly less spontaneous recovery and slower rates of reacquisition. It is important to consider the mechanism by which elevated fear responding can enhance extinction learning in order to better understand the results of these studies as well as their clinical implications.

There is some evidence that pharmacological manipulations which increase fear responding may optimize learning if administered prior to extinction (Cain, Blouin, & Barad, 2004). In this study, when rats were administered yohimbine (which blocks autoinhibitory α_2 adrenergic receptors and is anxiogenic in humans) prior to extinction,

learning was facilitated. The authors posited several possible mechanisms for these results including that increased adrenergic activity promotes consolidation of emotional memories (Cahill, Prins, Weber, & McGaugh, 1994; Cahill et al., 2000; Nielson & Jensen, 1994) so it is possible that administration of yohimbine facilitated consolidation of extinction memories. Further evidence for the role of elevated fear responding during extinction comes from the finding that pharmacological procedures which inhibit arousal, if administered during extinction, can impede extinction learning in rats (Berman & Dudai, 2001). Thus, it is possible that elevated fear responding alone facilitates extinction learning and the mechanism by which this occurs is that increased adrenergic activity promotes consolidation of memories encoded during extinction (i.e., the CS-no US relationship).

A possible mechanism for this is that elevated responding during extinction may lead to increased activation of the amygdala. There is extensive evidence in animals of the crucial role that the amygdala plays in the acquisition and extinction of emotional memories (for a review, see LeDoux, 2000). Most of this work has been done in animal models but, recently, there have been many investigations in humans as well; across neuroimaging, neuropsychological, drug and neural stimulation studies, the amygdala has been consistently implicated as playing a key role in enhancing explicit memory for highly emotional stimuli through modulation of encoding and consolidation processes (for a review, see Hamann, 2001). For example, one study examined the difference between healthy normal controls and patients who had undergone a unilateral resection of the anteromedial temporal lobe including the amygdala, hippocampus, and adjacent cortex as a surgical treatment for medically intractable epilepsy (LaBar & Phelps, 1998).

Participants rated emotionally arousing and neutral words on an arousal scale while their skin conductance responses (SCRs) were measured. Recall for the words was assessed immediately and after a 1-hour delay. Although both groups of participants demonstrated elevated SCRs and increased subjective arousal ratings for the arousing words at the time of encoding, only control subjects exhibited an increase in memory for the arousing words at the 1-hour delay test. This provides strong evidence for the role of the amygdala in consolidating memories of arousing events. Thus, procedures which increase fear responding during extinction (e.g., compound stimulus presentations, caffeine ingestion, and occasional reinforced trials) may facilitate learning by activating the amygdala which will enhance consolidation of extinction memories. In contrast, procedures in which fear responding diminishes during extinction and is low at the end of extinction (such as the Single Placebo group in Study 1 and the Control group in Study 2) may trigger reduced activation of the amygdala thereby attenuating consolidation of extinction memories.

If enhanced responding facilities consolidation of extinction memories, this may explain the successes of an arousal-mediated theory of learning which is able to explain some phenomena that the Rescorla-Wagner model cannot (Podlesnik & Sanabria 2011). These authors propose a modified version of the Rescorla-Wagner equation which accounts for arousal elicited by US presentation. They do so by replacing the β term in the Rescorla-Wagner model with *A* (arousal) and allowing it to vary with US density. Thus, when the US is presented frequently (by itself or along with other stimuli), CSs are more rapidly learned, extinguished, and discriminated. As time since the last US elapses, learning progressively slows down. This arousal-mediated model of learning can account

for phenomenon that the original Rescorla-Wagner model cannot; however, it has its own limitations (reviewed in Podlesnik & Sanabria, 2011). Nevertheless, it provides an interesting theoretical model for explaining how enhanced responding during extinction may facilitate extinction.

An especially interesting component of this model is that arousal varies with US density. Thus, the model does not necessarily predict enhanced learning as a result of elevated arousal in general (as in the case of caffeine ingestion) but rather predicts learning will be enhanced if arousal specific to US-presentations occur during extinction. This has important implications for Study 2, indicating that perhaps learning was optimized in the Reinforced group not only because those participants demonstrated enhanced responding throughout extinction but because US-triggered arousal continued to occur throughout extinction. The arousal-mediated model does not specifically address whether using a different US (than the one the CS was initially paired with) would also enhance learning although it does seem to imply that it should be the same US. It is interesting to consider whether presenting a different US during extinction (perhaps during an intertrial interval) than the one the CS was initially paired with elevate US-triggered arousal and facilitate inhibitory learning regarding the CS; this is something future studies can address.

It is also interesting to consider the predictions of the arousal-mediated model for Study 1. Although the US was not presented during extinction training in Study 1, CS presentations during extinction are hypothesized to activate memory of the US (e.g., Wagner, 1981) and it is possible that activation of the US memory representation is much stronger during compound trials than during single extinction trials. If so, US-triggered

arousal would be elevated in the compound extinction groups in Study 1 and the arousal-mediated model of learning would predict enhanced inhibitory learning regarding the CS.

Thus, elevated fear responding during extinction plays an important role in optimizing learning and protecting against future fear recovery effects.

Conclusions and Clinical Implications

The results of this set of studies provide support for several procedures in enhancing the effectiveness of exposure therapy for anxiety disorders: presenting multiple fear-provoking stimuli simultaneously after each has undergone some extinction training on its own, incorporating occasional CS-US pairings (e.g., a speech exposure with a negative evaluation, an external cue of a panic attack with a panic attack), triggering fluctuations in subjective fear responding throughout exposure, and maintaining elevated fear responding throughout exposure.

Underlying these procedures seems to be two mechanisms which facilitate learning during extinction: enhanced associative change and enhanced responding. This set of studies provides evidence for both of these mechanisms in optimizing learning during exposure exercises. However, the findings regarding enhanced responding were limited to tests of spontaneous recovery. In Study 1, caffeine ingestion attenuated spontaneous recovery but not reinstatement. In Study 3, the test of fear recovery was limited to a spontaneous recovery test; thus, it could not be ascertained whether sustained subjective fear responding throughout exposure also predicted attenuated reinstatement, renewal, or rapid reacquisition effects. Enhanced associative change, on the other hand, attenuated multiple fear recovery effects: spontaneous recovery, reinstatement, and rapid reacquisition. This suggests that, although enhanced responding alone may facilitate

inhibitory learning during extinction (i.e., CS-no US), enhancing the discrepancy between expectation and reality will facilitate this learning to a greater degree. The most compelling evidence for this is that, in Study 1, both caffeine and compound trials attenuated spontaneous recovery but only compound trials attenuated reinstatement. Thus, it seems that enhanced associative change is a more powerful mechanism for enhancing the effects of exposure therapy.

Interestingly, procedures which enhanced extinction learning across all three of these studies are at odds with Emotional Processing Theory which emphasizes initial fear activation, within-session habituation (i.e., decline in fear responding), and between-session habituation. In fact, broadly speaking, these studies indicate that exposure therapy will be most effective when exposure sessions are unpredictable, variable, include multiple fear-provoking stimuli, and include aversive events (e.g., social rejection, panic attack) that individuals are likely to experience occasionally following the completion of therapy.

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