

UCLA

UCLA Electronic Theses and Dissertations

Title

Arousal and Associative Processes in Reinstatement of Fear

Permalink

<https://escholarship.org/uc/item/4ff3j24g>

Author

Glenn, Daniel Erik

Publication Date

2014

Peer reviewed|Thesis/dissertation

UNIVERSITY OF CALIFORNIA

Los Angeles

Arousal and Associative Processes in

Reinstatement of Fear

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

Daniel Erik Glenn

2014

© Copyright by

Daniel Erik Glenn

2014

ABSTRACT OF THE DISSERTATION

Arousal and Associative Processes in Reinstatement of Fear

by

Daniel Erik Glenn

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2014

Professor Michelle G. Craske, Chair

This dissertation is a two-paper investigation of the basic associative and arousal-based processes involved in reinstatement of conditional fear. Clarifying the factors involved in reinstatement of fear—a behavioral phenomenon in which the experience of an aversive event following fear extinction produces a return of fear—provides a better understanding of the factors that contribute to the waxing and waning of untreated fear and the return of fear following exposure treatment. Shedding light on the processes involved in reinstatement of fear will contribute to the development of science-based interventions that offset return of fear following fear extinction and improve long term outcomes from exposure therapies for phobias and anxiety disorders.

Study 1 compared the effects of physical pain versus social pain as a reinstating US on reinstatement of conditioned fear, and compared standard reinstatement (same acquisition and reinstatement US) with cross-US reinstatement (different acquisition and reinstatement US). Results indicated that in standard reinstatement procedures social pain produced differential

reinstatement of conditional responding, while physical pain produced non-differential reinstatement (i.e., sensitization) in standard and cross-US reinstatement procedures.

Study 2 aimed to replicate the findings from Study 1, and investigated the role of CS+ belongingness on reinstating of fear. Combining data from both studies, the hypothesis was tested that greater physiological arousal experienced during the reinstatement would predict greater post-reinstatement return of conditional responding. Results replicated Study 1 findings, and found that CS+ belongingness may promote differential return of conditional responding in both standard and cross-US reinstatement. Additionally, findings indicated that higher arousal experienced during reinstatement predicts greater non-differential reinstatement, while concordance between arousal experienced during acquisition and reinstatement does not.

In sum, these studies indicate that both social pain and physical pain can reinstate extinguished fear. Social pain promoted differential return of conditional responding (in standard reinstatement) while physical pain elicited a non-differential increase in responding in both standard and cross-US reinstatement. Unforeseen methodological issues may have muddled the findings regarding cross-US reinstatement with qualitatively distinct USs, while providing tentative evidence that individuals under chronic stress might be at elevated risk of an acute stressor triggering a return of fear generalized beyond the original feared stimulus.

The dissertation of Daniel Erik Glenn is approved.

Michael Fanselow

Bruce Kagan

Thomas Minor

Michelle G. Craske, Committee Chair

University of California, Los Angeles

2014

TABLE OF CONTENTS

I.	Introduction.....	1
II.	Study 1	
	a. Introduction.....	14
	b. Methods.....	19
	c. Results.....	27
	d. Discussion.....	53
III.	Study 2	
	a. Introduction.....	57
	b. Methods.....	59
	c. Results.....	61
	d. Discussion.....	89
IV.	Conclusions.....	94
V.	Appendix 1: Study Stimuli.....	110
VI.	Appendix 2: Study 1 Statistical Output.....	119
VII.	Appendix 3: Study 2 Statistical Output.....	136
VIII.	References.....	150

ACKNOWLEDGEMENTS

The National Institute of Mental Health provided funding for this research through a Ruth L. Kirschstein National Research Service Award (NRSA) for Individual Predoctoral Fellows (1 F31 MH096404-01).

I wish to acknowledge the many individuals who were instrumental in the development and completion of this dissertation. In particular I would like to thank Dr. Michelle Craske, whose encouragement and belief in me has been unwavering since day one. I am so grateful to have you as a mentor and look forward to seeking your counsel and guidance for years to come. You are an inspiration to your students. I am also enormously thankful to the other members of my dissertation committee, all of whom steadfastly supported me in completing this final hurdle in my six-year graduate school journey.

I am deeply grateful for the hard work of my research assistants, Gabriella Imbriano, Jonathan Ebneyamin, and Stephanie Centeno. Your dedication quite literally made my dissertation studies happen through your many hours of recruiting and running participants.

Thank you to my graduate school cohort, who have become some of my closest friends and are some of the most impressive people I know. Thanks in particular to Richard, who became my number one partner-in-crime. To my parents, brother, grandparents, aunts, cousins, and in-laws, I hope you know how much your unconditional love and support has meant to me. To my wife Ashley, thank you for always knowing what I need before I do. I could not have done any of this without you, or anything else for that matter.

VITA

Education

- 2001-2005 Amherst College
B.A., Psychology
- 2008-2014 University of California, Los Angeles
M.A., Clinical Psychology

Professional Employment

- 2013-2014 Psychology Intern
Veterans Affairs Sepulveda Ambulatory Care Center

Academic Honors and Awards

- 2011-2013 Ruth L. Kirschstein National Research Service Award
National Institute of Mental Health, 1 F31 MH096404
- 2009-2010 University Graduate Research Mentorship Award
University of California, Los Angeles
- 2009-2010 Psychology Graduate Research Mentorship Award
University of California, Los Angeles
- 2009 Summer Research Mentorship Award
University of California, Los Angeles
- 2008-2009 University Fellowship
University of California, Los Angeles

Select Publications and Presentation

Glenn D, Golinelli D, Rose RD, Roy-Byrne P, Stein MB, Sullivan G, Bystritsky A, Sherbourne C, Craske MG (2013). Who gets the most out of cognitive-behavioral therapy for anxiety disorders? The role of treatment dose and patient engagement. *Journal of Consulting and Clinical Psychology*, 81, 639-649.

Glenn DE, Minor TR, Vervliet B, Craske MG (2014). The effect of glucose on hippocampal-dependent contextual fear conditioning. *Biological Psychiatry*, 75, 847-854).

Chavira DA, Golinelli D, ... **Glenn D**, Barrios V, Roy-Byrne P, Craske MG (2014). Treatment engagement and response to CBT among Latinos with anxiety disorders in primary care. *Journal of Consulting and Clinical Psychology*, 82, 392-403.

- Brown LA, **Glenn DE**, Rose RD, Stein MB, Sullivan G, Sherbourne C, Bystritsky A, Roy-Byrne P, Craske MG (2013). The Effects of Therapist Competence and Adherence On Novice Therapists' CBT For Anxiety. *Depression and Anxiety*, 30, 97-115.
- Rose RD, Buckley Jr. JC, Zbozinek TD, Motivala SJ, **Glenn DE**, Cartreine JA, Craske MG (2013). A randomized controlled trial of a self-guided, multimedia, stress management and resilience training program. *Behaviour Research and Therapy*, 51, 106-112.
- LeBeau R, **Glenn D**, Hanover L, Beesdo-Baum K, Wittchen H, Craske MG (2012). A Dimensional Approach to Measuring Anxiety for DSM-5. *International Journal of Methods in Psychiatric Research*, 21, 258-272.
- Niles AN, Lebeau R, Liao B, **Glenn DE**, Craske MG (2011). Dimensional indicators of Generalized Anxiety Disorder severity for DSM-V. *Journal of Anxiety Disorders*, 26, 279-286.
- Glenn D**, Craske MG, Minor T. (2011). *The Effect of Glucose on Contextual versus Cued Conditioning*. Symposium presentation at the Anxiety Disorders Association of America, New Orleans, LA, March, 2011.
- LeBeau R, **Glenn D**, Liao B, Wittchen H-U, Beesdo-Baum K, Ollendick T, Craske MG (2010). Specific Phobia: A review of DSM-IV Specific Phobia and preliminary recommendations for DSM-V. *Depression and Anxiety*, 0, 1-20.
- Glenn D**, Rose R, Craske MG (2010). *The CALM Study: Computer Assisted CBT Dose and Engagement on Outcome for Anxiety Disorders in Primary Care*. Symposium presentation at the Association for Behavioral and Cognitive Therapies 44th Annual Convention, San Francisco, CA, November, 2010.

INTRODUCTION

Anxiety disorders represent the most common category of psychopathology, estimated to affect roughly 20% of individuals (Pine & Klein, 2008). While there has been significant progress towards understanding the etiology of anxiety (Mineka & Zinbarg, 2006) and demonstrating the efficacy of exposure based treatments for anxiety disorders (e.g., Norton & Price, 2007; Tolin, 2010), many patients who experience a substantial reduction in fear during treatment subsequently experience a return of fear (Craske & Mystkowski, 2006; Craske & Rachman, 1987; Rachman, 1989; Rose & McGlynn, 1997). Factors that contribute to the post-treatment return of fear represent significant limitations to the effectiveness of exposure therapy for anxiety disorders and phobias. The broad goal of this research was to clarify the basic associative and arousal-based processes involved in reinstatement of fear in order to provide a better understanding of the factors that may contribute to return of fear following successful exposure treatment of anxiety disorders and phobias. Clarifying the processes involved in fear reinstatement represents the translation of basic scientific phenomena into valuable clinical application which may inform more effective and robust interventions and improve etiological understanding of anxiety.

Acquisition of fear

Pavlovian learning is believed to contribute to the acquisition of emotional fear responses, a core element of anxiety disorders (e.g., Bouton, Mineka, & Barlow, 2001; Mineka & Zinbarg, 2006). In Pavlovian fear learning, an initially neutral conditional stimulus (CS) does not elicit a fear response when presented alone. During acquisition of fear, the CS is paired with an unconditional stimulus (US), an aversive stimulus that naturally elicits an unlearned response, called the unconditioned response (UR). By pairing the CS with the US, the CS comes to predict

the US and therefore elicits a conditional response (CR), even when the CS is subsequently encountered in the absence of the US. In anxiety disorders, this conditioned relationship is often the result of a traumatic or highly aversive event through which a formerly neutral object or situation becomes associated with the unpleasant US. For example, a person may initially have no fear of a dog (CS), but after experiencing a dog bite (US) which naturally causes intense pain and physiological arousal (UR), the person subsequently experiences a fear response (CR) when encountering a dog. Through Pavlovian acquisition of fear, an excitatory association is learned between CS and US such that the CS predicts the onset of the US. Pavlovian primary conditioning serves as the basis for secondary conditioning (higher-order conditioning), which serves as the basis for the complex cognitive processes of fear-based psychopathologies (Blaisdell, 2009).

Extinction and the return of fear

Pavlovian extinction of fear is the process of extinguishing conditioned fear through repeated exposure to the CS in the absence of the US, which diminishes the CR (for review see Hermans et al., 2006). Many regard the extinction of conditioned fear as a laboratory analogue for exposure therapy for human fear and anxiety (Craske & Myskowski, 2006; Hermans et al., 2006). For example, after being bitten by a dog and acquiring a fear of dogs, repeatedly encountering dogs without being bitten will likely lead to an extinguished fear response to dogs. Although there is a small amount of evidence that extinction trials which are conducted immediately following acquisition may lead to unlearning of the CS-US relationship, (Myers et al., 2006), the overwhelming majority of research indicates that the excitatory relationship between CS and US learned during acquisition of fear is not forgotten or unlearned as a result of extinction that is conducted after fear acquisition (Bouton, 1993; Bouton & King 1983). Instead,

there is substantial evidence that during extinction a new inhibitory association is learned between CS and US, such that the CS is no longer predictive of the US. Following extinction, the CS has acquired an ambiguous relationship with the US; an excitatory CS-US association competes with an inhibitory CS-no US association.

Though the inhibitory CS-no US relationship appears to be more readily retrievable immediately following extinction as indicated by the diminished CR, numerous behavioral phenomena may contribute to the return of fear to the CS following extinction (e.g., Bouton et al., 2001; Hermans et al., 2006). The passage of time following extinction may result in the spontaneous recovery of the fearful CR (Baum, 1988). Contextual renewal of fear occurs when the extinguished CS is encountered in a different context from the extinction context (e.g., Bouton, 1993). Rapid reacquisition of fear following extinction occurs from additional pairings of the CS with the US (Kehoe & Macrae, 1997). Reinstatement of fear is a behavioral phenomenon in which the presentation of a US alone following extinction produces a subsequent return of the CR (e.g., Pavlov, 1927; Rescora & Heth, 1975).

Clinical relevance and understanding of fear reinstatement

Of the different behavioral phenomena that contribute to the return of fear following extinction, reinstatement may be of particular clinical relevance to the natural remission of, and the exposure treatment for, fears and phobias (Jacobs & Nadel, 1985). It is a truism that encountering some level of unpredictable life stress is inevitable, and such aversive experiences may serve as unsignaled USs which reinstate previously extinguished fear (Rachman, 1989), contributing to the chronic waxing and waning of untreated anxiety and to the return of fear following exposure therapy. Clinical research indicates that interpersonal stress during treatment for anxiety disorders may contribute to poorer treatment outcomes (Steketee et al., 2007), and

several studies (Steketee, 1993; Wade et al., 1993) and individual case examples (i.e. Rachman, 1979) suggest that the occurrence of stressful events post-treatment is associated with greater return of fear.

Reinstatement of an extinguished CR has been demonstrated experimentally with appetitive conditioning (Bouton & Peck, 1989) as well as with conditioned taste aversion (Schachtman, Brown, & Miller, 1985), though most research has been conducted on reinstatement of conditioned fear. Though fear reinstatement has been well demonstrated in animal (Bouton & Bolles 1979; Delamater 1997; Rescorla & Heth 1975) and human laboratory studies (Dirikx et al., 2004; Dirikx et al., 2007; Hermans et al., 2005; Neumann, 2008; Sokol & Lovibond, 2012), there is a dearth of clinical research on reinstatement (Boschen et al., 2009). One of the only experimental clinical studies conducted failed to demonstrate reinstatement of fear following treatment (Rachman & Whittal, 1989). After patients completed exposure therapy treatment for snake and spider phobias, they were presented with unpaired USs (electrical shocks) prior to being exposed again to their phobic stimuli. No reinstatement of fear was observed after US presentation, perhaps because the reinstating US (shock) differed from the original US experienced during fear acquisition.

The qualities of a US that are necessary to produce reinstatement remain unknown. There is ample experimental laboratory evidence demonstrating that unpaired USs contribute to reinstatement of an extinguished fear response, as well as clinical evidence and observation that stressful events often lead to a return of fear, but it remains unclear which kind of stressors are capable of reinstating fear, and why. For example, it is unclear if a successfully treated phobia of dogs would have a similar return of fear after being bitten by a cat, involved in an automobile accident, or socially humiliated.

Reinstatement: Associative process?

One explanation of reinstatement proposes that the return of the CR to the extinguished CS is mediated by associative processes involving excitatory context conditioning (Bouton et al., 2006). This theory contends that reinstatement relies on excitatory contextual conditioning, and that the return of the CR occurs only if the reinstating US is presented in the same context as the retest context (Bouton, 1984; Bouton & King, 1983; Frohardt et al., 2000, Wilson et al., 1995). Unpaired US presentations elicit contextual conditioning (e.g., Fanselow, 1980; Rescorla & Wagner, 1972; Vansteenwegen et al., 2008), so it is proposed that an excitatory association is learned between the US and context during reinstatement. According to this associative account, following reinstatement the excitatory reinstating context activates a mental representation of the US, so subsequently encountering the CS in this context reinstates the original CS-US association, causing a return of the CR (Bouton et al., 2006). For example, after extinguishing a fear of dogs, being bitten by a cat in a particular context should lead to associating that context with being bitten, so the next time a dog is encountered in this context (which now brings to mind the experience of being bitten) there will be a return of fear to the dog. An associative account of reinstatement suggests that only a US that is similar to the original US should reinstate CR to the CS. For example, an extinguished fear of dogs should likely be reinstated after being bitten by a cat but not after being socially humiliated.

Reinstatement: Arousal-based process?

There are several alternative accounts of arousal-based processes driving reinstatement. One arousal-based model of reinstatement comes from the drug-relapse literature (Stewart, 2000, 2008). There are clear differences between the motivational processes involved in drug-related behavior and fear-learning, most basically perhaps the appetitive versus aversive nature of the

USs involved. However, there are important similarities as well, including acquisition, extinction and post-extinction return of strong affective responses to CSs. An extensive body of clinical and experimental research demonstrates that following extinction of drug-craving and drug-seeking behavior, the experience of a discrete stressor will reinstate drug-seeking behavior (for review see Stewart, 2000 and 2008). For example, in rats trained to self-administer an addictive drug and then given extinction training, being exposed to unpredicted foot-shocks leads to reinstatement of drug-seeking behavior (e.g., Shaham, Erb & Stewart 2000). Stewart argues that discrete stressors induce reinstatement of drug-craving by renewing the salience of drug-related cues and stimuli, a process mediated by neural activity of corticotropin-releasing factor (CRF) and noradrenaline. The implications of these findings on reinstatement drug-seeking behavior for reinstatement of fear are that stressful experiences may reinstate fear by renewing the salience of fear-related stimuli, a process which may be driven by physiological arousal and neuroendocrine activity. One additional, potentially relevant finding regarding reinstatement of drug-seeking behavior is that certain distinctly different types of stressors (discrete foot-shock, food deprivation, CRF injection) produce similar reinstatement (Shaham et al., 2000). The implication of this finding is that distinctly different types of USs may be capable of reinstating fear if they produce sufficiently high arousal and neuroendocrine response. In contrast to the associative model of reinstatement, this non-specific arousal-based account of reinstatement stemming from the drug-relapse literature suggests that any unpaired US that produces a UR consisting of sufficient arousal and neuroendocrine response should be adequate to reinstate the CR. For example, an extinguished fear of dogs (originally acquired following a dog bite) should be reinstated by any stressor that produces a high level of arousal.

Other theories of reinstatement suggests that the physiological arousal produced by

unpaired US presentations reinstates the fearful CR to the CS by acting as a reminder of the internal state of arousal experienced during fear acquisition (Hourutunian & Riccio, 1979; Hermans et al., 2005; Jacobs & Nadel, 1985). Acute stressors (such as unpredictable USs) typically produce heightened autonomic activity, including increased heart rate and blood pressure, as well as neuroendocrine activity (elevations in “stress hormones” such as cortisol, epinephrine, and norepinephrine; Johnson et al., 1992). Neuroendocrine responses to stressors may vary significantly based on subjective perceptions (Biondi & Picardi, 1999), allowing for a wide range of physiological arousal in response to stress. This similar-arousal-based theory of fear reinstatement contends that a stressor eliciting physiological arousal similar to the arousal experienced during fear acquisition should act as a reminder of the internal state experienced during fear acquisition, thereby serving as an internal retrieval cue of the excitatory CS-US association (Hourutunian & Riccio, 1979; Jacobs & Nadel, 1985).

In one study, immature rats (21 days old) completed a discriminative fear conditioning procedure (shock paired with one compartment of a shuttle box, no shock paired with the other compartment of the shuttle box), completed a reinstatement procedure one week later, and then one week later completed a passive place avoidance test as a measure of fear (Hourutunian & Riccio, 1979). Reinstatement of fear was achieved by simultaneous exposure to the feared CS along with administration of adrenocorticotropic hormone (ACTH; a stress hormone released as part of the hypothalamic-pituitary-adrenal axis, administered via injections at levels equivalent to those that rats release in response to shock), but no reinstatement occurred from simultaneous fear cue exposure along with amphetamine or strychnine administration (stimulants of the central nervous system). In a similar study, exposure to a fear cue and administration of epinephrine (a catecholamine stress hormone) reinstated a fear CR (Hourutunian & Riccio, 1977). These

findings did not examine reinstatement of extinguished fear (no extinction trials were conducted, though immature rats demonstrate rapid memory loss of initial fear acquisition), and the procedures used a CS in addition to induced arousal as the reinstating stimuli rather than using an unlearned US. Nevertheless, they suggest that in order for arousal to reinstate conditioned fear, neuroendocrine activity during reinstatement must be similar to neuroendocrine activity experienced during original fear acquisition.

It has been posited that fear is a combination of high arousal and negative valence (Lang et al., 1990). During acquisition of fear the CS comes to have a predictive association with the US, and elicits arousal as well as negative valence (Baeyens et al., 1988; Hermans et al., 2002). While extinction is effective at reducing the excitatory predictive value of the CS and arousal in response to the CS, the negative valence of the CS often survives extinction (De Houwer et al. 2001). A recent account of fear reinstatement contends that the presentation of reinstating unpaired USs leads to the context eliciting high arousal (excitatory context conditioning during reinstatement; Bouton, 1991). When an extinguished CS is encountered following reinstatement, the return of the fearful CR may result from residual negative valence (from the extinguished CS) being recombined with arousal (from the now excitatory context) (Hermans et al., 2005). Several studies have replicated the finding that following extinction higher residual negative valence of a CS, though not fear or US-expectancy ratings, predicts greater return of fear following reinstatement (Dirikx et al., 2004; Dirikx et al., 2007; Hermans et al., 2005). However, these studies have not specifically measured the role of arousal in reinstatement.

Hourutunian and Riccio's findings (1977, 1979) suggest that in order for arousal to reinstate fear it must be sufficiently similar to the arousal experienced during acquisition. Thus, not every stressful or arousal experience would be expected to reinstate fear. For example,

interpersonal stress and physical exercise both produce high levels of arousal, but each elicits distinct patterns of physiological changes (e.g., Dimsdale & Moss, 1980), and interpersonal stress is believed to be capable of reinstating fear while physical exercise is not (Steketee, 1993). In contrast to the associative model of reinstatement and the non-specific arousal-based account of reinstatement, this similar-arousal-based account of fear reinstatement suggests that any unpaired US that produces a UR adequately similar to the internal state of arousal experienced during initial US-CS pairings should be sufficient to reinstate the CR. For example, an extinguished fear of dogs (originally acquired following a dog bite) should be reinstated by any stressor that produces similar physiological arousal to the arousal experienced following the initial dog bite.

Review of research about which types of USs produce reinstatement

In animal research, one study found that cross-US reinstatement (using different acquisition and reinstatement USs) produced a return of fear following presentation of either shock or loud noise (Rescorla and Heth, 1975). Recent research has demonstrated successful cross-US reinstatement of fear in humans with shock and loud noise as the two USs (Sokol & Lovibond, 2012). However, an appetitive conditioning study with rats found that presentation of a different appetitive US (pellet or sucrose) from the US used to train bar-pressing response to a CS was insufficient to produce reinstatement of responding (Delamater, 1997). Aside from one study where no reinstatement effects were observed in a clinical sample (Rachman & Whittal, 1989) clinical evidence is qualitative only, without any direct experimental evaluation. Specifically, clinical research suggests that various forms of life-stress may contribute to the onset of anxiety disorders (Blazer et al., 1987; Brook & Schmidt, 2008; Gothelf et al., 2004; Klauke et al., 2010), and that interpersonal stress during treatment may contribute to poorer

treatment outcomes for anxiety disorders (Steketee et al., 2007). Several studies (Steketee, 1993; Wade et al., 1993). Individual case examples (i.e. Rachman, 1979) suggest that the occurrence of stressful events post-treatment is associated with greater return of fear, but there is a lack of well-controlled research in general, let alone research on which types of events reinstate which types of fear, and through which processes they do so.

Predictions of associative, non-specific arousal, and similar arousal-based theories of reinstatement

Associative accounts of reinstatement suggest that only a US that is similar to the original US should reinstate the CR to the CS. In contrast to associative models of reinstatement, arousal-based accounts of reinstatement suggest that reinstatement of the CR depends on the internal state of arousal experienced during reinstatement. A non-specific-arousal based account of reinstatement (based on extensive drug-relapse research) suggests that any unpaired US that produces a UR consisting of a sufficiently large increase in arousal and neuroendocrine response should reinstate the CR. Similar-arousal-based accounts of fear reinstatement suggest that reinstatement depends on an unpaired US producing a UR that is similar to the internal state of arousal experienced during initial US-CS pairings.

Both associative (i.e. Bouton et al., 2006) and arousal-based accounts (Hermans et al., 2005) suggest that in order for reinstatement to occur, the unpredictable US must encountered in the test context or return of fear to the CS will not occur. The role of contextual learning in reinstatement is an important issue, but it was not the focus of this research. As described later, both studies utilized the same experimental context during all experimental procedures.

Associative learning theories relevant to qualitatively different USs in reinstatement

One useful approach to considering the distinctly different predictions made regarding

the effects of associative and arousal-based factors on reinstatement of fear may be to look more broadly to prominent theories of associative learning. In recent years, the most commonly utilized associative learning theory in human fear conditioning research has been the Rescorla-Wagner (R-W) model (Rescorla & Wagner, 1972) which has been used to generate a plethora of valuable predictions relevant to acquisition and extinction of fear, as well as associative learning process more broadly (for review see Miller et al., 1995). R-W has been applied to understanding the role of context conditioning in reinstatement (i.e., Bouton & Bolles, 1979), but overall as a model R-W has been less successful in dealing with return of fear phenomena following extinction, including reinstatement (for review see Miller et al., 1995). The primary problem with R-W in terms of return of fear phenomena the model's assumption that following extinction the associative value of the CS has been erased. Moreover, in regards to the current research it is unclear how R-W would be useful in making predictions about the effect of qualitatively distinct aversive USs in reinstatement.

One associative learning theory that might prove useful for considering the current research question is the Affective Extension of the Sometimes-Opponent Process (AESOP) theory (Wagner & Brandon, 1989). In order to apply the model to the current research a brief overview will be provided, but for a more detailed explanation see Wagner and Brandon (1989). The AESOP model assumes that stimulus presentation (CS or US) elicits representational nodes which at any time can be in one of three states of attention: A1 (primary focus of attention), A2 (periphery of attention), and I (inactive/long-term memory). When a stimulus is first encountered it enters into the A1 state, then over time decays into the A2 state (based on a time-dependent decay parameter, which varies by stimulus), and then into the I state (based on a time-dependent decay parameter). A node in A2 cannot directly move into A1, but rather must decay into the I

state before entering A1. In this model excitatory and inhibitory learning between stimuli occurs based on the states elements of different stimuli co-occur in: elements which co-occur both in A1 or both in A2 will develop excitatory associations, while elements which co-occur in different states (one in A1 and another in A2) will develop inhibitory associations. Once an excitatory association has been formed between stimuli, presentation of one stimulus will bring the other stimulus into the A2 state.

The key addition to the AESOP model building on the previously posited Sometimes-Opponent Process model (SOP) is that stimuli have both sensory and emotional elements that each proceed through the A1-A2-I stages, and that emotional elements are likely to have a slower decay rate than sensory elements. Before conditioning a CS is assumed to elicit a very weak emotional node, so emotional activity is generally ignored for CSs before learning, although following associative learning a CS+ may elicit emotional responding by activating the A2 emotional state of the US. The emotional responses in the A1 and A2 states may be identical, similar, or distinctly different, a parameter which varies by stimulus.

The basic assumption of the AESOP model that different types of USs produce distinct URs, and that the unique temporal and emotional qualities of the UR play a central role in determining the form of conditional responding, make it well suited for considering the associative and arousal-based factors that influence reinstatement. This assumption is particularly relevant to the current research given that the UR to physical threat/pain (i.e., vigorous escape and avoidance behaviors, elevated sympathetic nervous system activity) is believed to differ in important ways from the UR to social threat/pain (i.e., submissive gestures, such as blushing and lowering of one's head, less significant sympathetic nervous system activation; Ohman, 1985). Moreover, this model's distinction between sensory and emotional

elements is helpful for separately considering both perceptual differences between stimuli and affective/arousal-inducing differences.

According to the AESOP model, we would expect stronger standard than cross-US reinstatement. AESOP treats CSs and USs similarly with regards to associative learning processes, so following excitatory learning between CS+ and US during acquisition, subsequent presentation of the same US should selectively recall previously learned excitatory associations with the CS+. The sensory and emotional nodes of a standard reinstating US should selectively recall previously learned excitatory associations with the CS+, while this recalling of excitatory CS+ associations should not occur with a qualitatively different US in cross-US reinstatement. An additional tentative prediction of the AESOP model would be greater Social/Physical reinstatement than Physical/Social reinstatement. The rationale behind this prediction is that reinstatement may be expected to the extent that emotional nodes of the reinstating US recall previously learned excitatory associations with the CS+. Sympathetic activation is part of the emotional response to both P-US and S-US, although it may be higher in response to P-US (Öhman, 1986). Thus, it would be predicted that emotional elements (i.e. high sympathetic activation) triggered during P-US reinstatement would be more likely to act as reminders of S-US acquisition emotional elements (low to moderate sympathetic activation) than would S-US reinstatement elements recall P-US acquisition elements.

STUDY 1

Introduction

The most commonly used US in human fear conditioning research is electrical shock, which effectively serves as a physically painful US (P-US). Physical pain (an unlearned US) is believed to activate a pain motivation system, which promotes recuperative behaviors as part of the UR. Anticipation of physical pain (i.e. an excitatory CS signaling P-US onset) activates a fear motivation system, which promotes defensive behaviors as part of the CR (Bolles & Fanselow, 1980). Social pain (i.e. perceived or actual social rejection) is a highly aversive but distinct experience from physical pain or fear of physical harm. Perceived or actual threats of rejection from a group lead to considerable distress for most people, likely as a result of an evolutionary adaptation to ensure the maintenance of social bonds (Herman & Panksepp, 1978; Thornhill & Thornhill, 1989). Experimentally induced social pain has been demonstrated to produce deleterious short-term changes in neuroendocrine, cardiovascular, and immune system functioning (Dickerson, 2011). Research in humans and animals suggests that the social pain system built off the already existing physical pain system, and as a result there is significant overlap between the two systems (for review see MacDonald & Leary, 2005). Recent neuroimaging research demonstrates that neural areas central to physical pain responses, such as the dorsal anterior cingulate cortex (dACC) and periaqueductal grey (PAG), are also central to social pain responses (Eisenberger, 2011, 2012). Recent research has also demonstrated that a socially painful US (S-US) may successfully engender aversive conditioning in socially anxious individuals (Lissek et al., 2008).

While physical and social pain share similarities, there are hypothesized to be qualitative differences between the UR to a P-US and S-US. Cues eliciting anticipation of physical pain

(e.g., pain, loud noises, loud scream, signals of physical danger) elicit a response consistent with acute fear (escape and avoidance behaviors, elevated heart rate, shortness of breath, and elevated sympathetic nervous system activation), while the threat of social pain is believed to elicit responses serving to avert attack or rejection from dominant conspecifics (submissive gestures, blushing and lowering of the head, and moderate sympathetic activation; Öhman, 1986). Yet, the relationship between P-USs and S-USs in fear learning processes, including reinstatement, remains unclear. For example, it is unclear if an extinguished phobia of dogs (initially acquired following a physically painful dog bite) would be reinstated through social humiliation, or if a successfully treated social phobia (initially acquired following a socially painful experience) would be reinstated after being in an automobile accident.

Research has demonstrated that the simultaneous presentation of a picture of an angry face and a non-personally-relevant insult may function as an S-US capable of eliciting fear conditioning in social anxious individuals (Lissek et al., 2008). The use of such socially painful stimuli, however, is not ideal for comparison with a P-US because unlike physical pain which is both aversive and personally relevant, such social pain is aversive but not personally relevant. The Trier Social Stress Test (TSST) is a widely used laboratory task which reliably elicits social stress and a physiological stress response (i.e. changes in ACTH and cortisol) by providing participants with personally relevant negative feedback while they are delivering a speech and performing mental arithmetic in front of an audience (Kirschbaum, Pirke, & Hellhammer, 1993). Yet, as the TSST takes 20 minutes and is completed continuously, it is not an ideal task for presenting discrete S-USs which are similar in presentation and duration to P-US. Therefore, novel social pain stimuli were developed as part of this research, building on previous methodology which utilized S-US in conditioning (simultaneous presentation of angry face plus

auditory insult; Lissek et al., 2008) and a personally-relevant social pain manipulation (Eisenberger, 2012).

Photographs of male actors expressing a range of different emotions were taken from a large database of videos that were created for a prior study in the laboratory (see Niles et al., 2013 for full review of methodology). 28 pictures of faces (4 happy, 8 neutral, and 16 angry) and 40 adjectives (7 positive, 4 neutral, and 29 negative) were pilot tested for ratings of valence, distress, and rejection. Participants were asked to separately rate each face on the following 1 (“not at all”) to 7 (“completely”) likert scales: 1) *How negative is this facial expression?* 2) *How distressed would you feel if somebody made this facial expression at you?* 3) *How rejected would you feel if somebody made this facial expression at you?* Participants were asked to separately rate each adjective on the following 1 to 7 likert scales: 1) *How negative is this word?* 2) *How distressed would you feel if somebody used this word to describe you?* 3) *How rejected would you feel if somebody used this word to describe you?* For both faces and adjectives composite scores were calculated for the average of the distress and rejection ratings. Four words were chosen for use as part of the S-US, all with composite scores over 5.5 out of 7: *unlikeable*, *dumb*, *annoying*, and *forgettable*. Four angry faces were chosen, two each from two different actors, all with composite scores over 4 out of 7.

It is noteworthy that even the most negatively rated faces were given only moderate ratings on the distress and rejection scales; the highest composite score for any face was 4.76 out of 7. This finding is not entirely surprising, given that during pilot testing participants may have habituated to the negative faces while viewing 28 pictures consecutively, and given that the negative facial expressions were not presented as being personally relevant. It was anticipated that in this research the negative faces would be experienced as more highly aversive due to

being presented to social anxious individuals as personally relevant, and in conjunction with personally relevant auditory negative feedback.

The social pain manipulation developed for this research involved deceiving participants. Participants were told that they would be watching and rating another participant's videotaped interview, before completing a similar videotaped interview and subsequently being rated themselves by another participant. Participants were seated in front of a computer, shown a live video of themselves being recorded on the computer by a webcam, and told to speak into the webcam so the reviewer could see and hear them clearly. Next, participants were shown a video of another participant being interviewed (actually a standard video made with a confederate actor). Participants were told that while watching the video their own reactions to the watching the interview would be recorded through the webcam. Participants were given a list of 30 positive, negative, and neutral adjectives and told to rate the other participant's performance using up to 10 words from the list (list included in appendix 1). After participants completed watching the interview, they were asked to read the adjectives they selected aloud into the webcam. Next, participants were informed that they would now complete a similar interview, comprised of 7 personal questions (i.e. "*What do you see yourself doing in five years?*" "*What is your favorite hobby?*") as well as intellectually-oriented questions (i.e. "*What are the biggest challenges facing the United States?*" "*What are your thoughts and predictions about the 2012 presidential election?*"). Participants were told that just as they had rated another person's interview performance, another participant would be watching their video and choosing adjectives from the list to describe their impressions of the participant based on the their personality and intelligence demonstrated during the interview. Participants were informed that later during the study they might receive feedback based on the reviewer's evaluation of their

performance which would consist of two elements: a picture of the reviewer's face taken at some point while they were watching the video, and simultaneously the voice of the reviewer speaking one of the rating words that they used to describe the interview performance. Participants were then shown several examples on the computer of what this feedback might look like, with several presentations of neutral or smiling faces and neutral and positive words. The faces used as examples were from different actors than the one used as the actual study reviewer. Finally, participants were shown a smiling picture of the person who would be rating their interview. Later, during the appropriate study phase, the S-US was comprised of the simultaneous presentation of an angry face of "the reviewer" and an auditory presentation of one of the negative adjectives. For each S-US presentation a different negative adjective was used. All social pain stimuli (angry faces and the negative adjectives they are presented along with) are included in the Appendix 1.

Though most experimental studies of standard fear reinstatement have used the same US (shock) during both acquisition and reinstatement, the same exact US may not necessarily occur in the natural environment, and thus it is important to understand cross-US reinstatement with qualitatively different US. There is some evidence for cross-US differential fear reinstatement in humans, although this research used USs qualitatively similar to each other in eliciting physical threat CRs (Sokol & Lovibond, 2012). Recent studies with S-USs highlight the importance of utilizing disorder-relevant USs to better understand conditioned fear (Lissek et al., 2008).

Study 1 compared four different reinstatement procedures: acquisition and reinstatement both with P-US (Physical/Physical), acquisition and reinstatement both with S-US (Social/Social), P-US acquisition with S-US reinstatement (Physical/Social), and S-US acquisition with P-US reinstatement (Social/Physical). Indices of conditional responding through

all experimental phases included self-report measures (P-US expectancy, S-US expectancy, valence and arousal ratings) and physiological measures (acoustic startle response, skin conductance response, photoplethysmography). Several hypotheses were tested in Study 1. First, it was hypothesized that both P-US and S-US would support acquisition of conditional responding to the CS+ relative to the CS-. Second, it was hypothesized that reinstatement with both the P-US and S-US would produce a significant differential increase in responding to the CS+ relative to the CS- from the end of extinction to post-reinstatement test trials. Third, it was hypothesized that standard reinstatement (Physical/Physical, Social/Social) procedures would produce greater differential fear reinstatement than cross-US reinstatement (Physical/Social, Social/Physical).

Methods

Overview of design

Participants were randomly assigned to one of four experimental groups according to a 2 (Acquisition US: *Physical* or *Social*) x 2 (Reinstatement US: *Physical* or *Social*) design. The distribution of participants across the groups was as follows: Physical/Physical=17, Social/Social=13, Physical/Social=14, Social/Physical=13.

Participants

Fifty-seven participants (37 females, 20 males), with a mean age of 22.01 ($SD=5.52$) were recruited from undergraduate introductory psychology courses and through posting recruitment flyers on a university campus. Study participants received either course credit or \$30 for 2 hours of study participation. The ethnic distribution of the sample was 24.6% Caucasian, 38.6% Asian, 22.8% Hispanic, 5.3% African American, and 8.8% who classified themselves as multiethnic or other. Participants were recruited if they met the following eligibility criteria: a)

age 18 years or older; b) English speaking; c) score 6 or higher on the Mini Social Phobia Inventory (Mini-SPIN). Exclusion criteria for study participation included: a) physician recommended to stay away from stressful situations; b) serious respiratory, cardiovascular, pulmonary, or neurological condition, c) hearing impaired, d) pregnant.

Measures

Questionnaires

Mini-Social Phobia Inventory (Mini-SPIN; Connor et al., 2001) is a 3-item brief screening questionnaire assessing fear and avoidance of embarrassment, which was developed from the longer 17-item *Social Phobia Inventory* (SPIN; Connor et al., 2000) and has demonstrated reliability and validity. The Beck Depression Inventory (BDI; Beck et al., 1961) is a widely used 21-item questionnaire that assesses symptoms of negative mood. State anxiety was measured by the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1970). The Anxiety Sensitivity Index (ASI; Peterson & Heilbrunner, 1987) was used to measure anxiety sensitivity, defined as the fear of anxiety-related symptoms, which is another important variable that has been demonstrated as a cognitive risk factor in the development and maintenance of anxiety disorders. Trait mindfulness was measured by the Mindfulness Attention Awareness Scale (MAAS; Brown & Ryan, 2003). Mindfulness, described as a non-judgmental present-centered awareness, may predispose individuals to be less likely to respond fearfully during conditioning.

Indices of fear responding: Physiological arousal

Physiological arousal was recorded continuously using a psychophysiology data acquisition system (Biopac; Pflanzner, 1998) which measured skin conductance response and acoustic startle response. It has long been argued that the different fear response channels exhibit differential forms of reactivity to fear learning and extinction (Rachman, 1978; Grey et al., 1979).

Using multiple indices of fear responding allows measurement of general arousal (SCR) as well as potentiated fear (ASR).

Skin Conductance Response (SCR): SCRs were measured using a Biopac MP150 unit running Acqknowledge 4.0 software with a GSR100C amplifier set to direct current and had a sensitivity of 5 $\mu\text{ohm/V}$, with a 1.0-Hz low-pass filter. SCRs were measured at the onset of each CS and each US.

Skin conductance signals were acquired using two disposable 1 cm diameter AG-AgCl electrodes 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 in microsiemens (μS) within 1–6 seconds following CS onset and the mean skin conductance level within the 2-second period prior to CS onset. Skin conductance amplitude was recorded to the nearest μS throughout each trial and each ITI, as well as throughout the reinstatement phase. 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 US-onset. Amplitudes were range corrected using the largest response elicited by the any acquisition or reinstatement US presentation for each individual participant. For each participant, all SCRs to CSs were divided by the participant'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. Outliers at each time point were transformed through winsorizing (Wilcox & Keselman, 2003).

Acoustic Startle Response (ASR): Fear potentiated ASR (shown to be potentiated by aversive conditioning; Lipp, Sheridan & Siddle, 1994), was measured by exposing subjects to

brief white noise presented through headphones and then measuring the movement of muscles around the eye in response to this noise. Surface electromyogram data were acquired with a Biopac MP150 unit using two electrodes (Ag/AgCl, 11 mm outer diameter, 4 mm inner diameter contact surface) filled with electrode gel and fitted with adhesive collars. The first electrode was placed directly underneath the left eye, and the second was placed approximately 1 cm to the left below the outside corner of the left eye. Raw voltages were filtered via hardware (BIOPAC model EMG100C) and software (AcqKnowledge version 4.0, BIOPAC Systems, Inc.) producing a 10 - 500 Hz passband. Data were sampled at 2000 Hz, and rectified and averaged over 10 milliseconds. Subjects received auditory startle probes (105 dB, zero rise time, 50 ms bursts of white noise) binaurally via headphones. One startle probe was presented during every CS presentation and every ITI, but not during the reinstatement phase. Probes were presented 5.5 to 7 seconds after CS onset and 20 to 30 seconds after ITI onset. Probe presentation varied within the given timeframes in one of two predetermined orders, counterbalanced across trials, stimuli, and groups. ASRs during CSs and ITIs were calculated as the difference between mean EMG amplitude during the 20 ms prior to onset and the maximum amplitude during the 52 to 65 ms following probe onset. Outliers at each time point were transformed through winsorizing (Wilcox & Keselman, 2003), and then ASR scores were standardized using a z-transformation.

Photoplethysmography (PPG): PPG detects changes in infrared reflectance resulting from varying blood flow to the face, and was used to measure blushing response. PPG has previously been used to demonstrate experimentally induced blushing to social embarrassment (Shearn et al., 1990, 1992). Though PPG has never been previously measured in conditioning research, it was included as a measure with the potential to capture the blushing response, believed to be a unique form of responding to social but not physical threat (Öhman, 1986). PPG

was measured via a matched infrared emitter and photo diode attached to the forehead of the participant. Placement on the forehead is based on findings that such placement yields the same results as cheek placement and is less sensitive to facial movement (Cooper & Gerlach, 2013). PPG responses were calculated for CS, and for the entirety of the reinstatement phase. PPG responses to CSs were calculated by taking the difference between the mean PPG level for the 5 seconds prior to CS onset and maximum PPG level in the 33 seconds following CS onset. PPG response during the reinstatement phase was calculated by taking the difference between the mean PPG level for the 5 seconds prior to the first reinstating US onset and maximum PPG level in the 90 seconds following US onset. The comparatively long window of measurement for PPG is based on prior research suggesting that the blushing response may take significantly longer to reach its peak than other measures of autonomic activity (Cooper & Gerlach, 2013). Outliers at each time point were transformed through winsorizing (Wilcox & Keselman, 2003), and then PPG scores were standardized using a z-transformation.

Indices of fear responding: Self-Report

Valence and Arousal ratings: Before and after study phases participants rated current response to each CS on two 0 to 10-point scales (0=not at all, 5=moderately, 10=very much). Participants shown a picture of each CS separately, and were asked to rate current valence (“How negatively do you feel about this stimulus?”) and arousal (“How fearful do you feel about this stimulus?”)

Online P-US Expectancy: Participants rated expectancy of muscle stimulation via a dial attached to the arm of their chair (Lovibond et al., 2000), as used in prior studies in our laboratory. Participants continuously rated P-US expectancy on a sliding dial (BIOPAC model TSD115, AcqKnowledge version 4.0; BIOPAC Systems, Inc.). P-US expectancy was calculated

as the mean rating during the 0.5 seconds before acoustic startle probe onset.

Online S-US Expectancy: Participants rated expectancy of reviewer feedback via a dial attached to the arm of their chair (Lovibond et al., 2000), as used in prior studies in our laboratory. Participants continuously rated S-US expectancy on a sliding dial (BIOPAC model TSD115, AcqKnowledge version 4.0; BIOPAC Systems, Inc.). S-US expectancy was calculated as the mean rating during the 0.5 seconds before acoustic startle probe onset.

Conditioning Stimuli

Conditional Stimuli: 2 geometric figures (blue square, yellow circle) were randomized to serve as CS+ and CS-. Pictures of conditional stimuli are included in the Appendix 1.

Unconditional Stimuli: Two US were used. The second was a S-US, which was an angry face paired with an auditory insult. As previous research with S-USs found conditioning effects in social anxious individuals only (Lissek et al., 2008), both proposed studies screened with the Mini-SPIN for participants at least moderately high in social anxiety. The P-US was an electrical stimulation of the bicep muscle. As the S-US was a compound stimulus consisting of both visual (angry face) and auditory (verbal insult) components, the physical pain US was also designed to be a compound stimulus including physical (electrical stimulation) and visual (picture of an electrical spark) components. Pictures of study unconditioned stimuli are included in the Appendix 1.

Procedures

Participants signed informed consent, completed questionnaires, and then were seated two feet in front of a 21" computer monitor placed at eye level which was used to display the stimuli. Experimenters attached electrodes from the Biopac MP-150 system to participants for recording physiological responses and to deliver the uncomfortable bicep muscle stimulation that

served as part of the P-US. Next, participants completed the social pain manipulation, including both rating another person's video and then completing their own five-minute interview. To establish the appropriate level of muscle stimulation, subjects underwent a stimulation workup procedure in which each individual selected a level that they experienced as unpleasant but not painful, as has been done in previous conditioning research (Lipp, 2006). This level was then applied as the P-US muscle stimulation level throughout the experimental phases. Two geometric figures (blue square, yellow circle) were counterbalanced and served as the CS+ and CS-.

Participants were informed that during the study their primary task would be to figure out when it was likely that reviewer feedback or muscle stimulation be presented. Study procedures around explaining expectancy dials were loosely based on procedures used by Sokol and Lovibond (2012). Participants were shown how to use a "Muscle Stimulation Expectancy" dial and a "Reviewer Feedback Expectancy" dial. The P-US expectancy sliding dial ranged from "1=Stimulation Unlikely" to "9=Stimulation Very Likely," with a midpoint anchor of "5=Stimulation Uncertain." The S-US expectancy sliding dial ranged from "1=Feedback Unlikely" to "9=Feedback Very Likely," with a midpoint anchor of "5=Feedback Uncertain." Participants then completed several practice trials using the expectancy dials in response to images and information presented on the computer screen. The experimenter provided corrective feedback as necessary to ensure that participants were correctly utilizing the feedback dials, and by the end of the third and final practice trial all participants demonstrated understanding how to use the expectancy dials.

Participants then completed the *Habituation* phase consisting of 6 trials (3 CS+ and 3 CS-). No US presentations occurred during Habituation. Throughout the study, CS presentations

lasted 8 seconds, and the ITI varied between 35 to 45 seconds. Next, participants completed the *Acquisition* phase, consisting of 12 trials, comprised of 6 CS+ trials that each co-terminate with the US (either all P-US or all S-US, depending on group randomization), and 6 CS- trials with no US presentation. During pilot testing of the experimental procedures multiple participants gave the feedback that receiving exclusively negative feedback about their interview performance during acquisition quickly diminished the believability and potency of the S-US. Thus, to enhance S-US feedback, two unpaired presentations of neutral feedback were added to the acquisition phase. At the start of the acquisition phase participants were presented with a neutral looking picture of the reviewer paired with hearing “decent,” and another unpaired neutral feedback presentation (“adequate”) was presented after one presentation each of the CS+ and CS-. Thus, S-US acquisition participants were presented with a total of 8 pieces of reviewer feedback during acquisition (6 negative feedback paired with CS+, 2 neutral unpaired feedback) while P-US acquisition participants were presented with 2 neutral and unpaired pieces of reviewer feedback during acquisition. The neutral feedback was presented at least 20 seconds apart from the presentation of any other study stimuli. Following acquisition, participants completed the *Extinction* phase, consisting of 12 trials, comprised of 6 CS+ (without US) and 12 CS- trials (without US), again in one of two predetermined random orders.

After a 2-minute break, participants next completed the *Reinstatement* phase during which participants were randomized into one of two reinstatement procedures: *physical pain* or *social pain*. In physical pain and social pain reinstatement groups participants received four US presentations (P-US in physical pain group, S-US in social pain) over a period of 95 seconds. Reinstating US were presented with no prediction after 15, 55, 85, and 95 seconds (Hermans et al., 2005). No CS or startle probes were presented during reinstatement. After a 5-minute rest

period following reinstatement, participants completed a *Test phase*, consisting of 4 trials (2 with CS+, and 2 with CS-), all without US presentation. CS+ and CS- trials during all study phases were presented in one of two predetermined orders. The order of trials were counterbalanced across participants during all phases.

Throughout all study conditioning phases SCR, ASR, and PPG response were monitored, in addition to participants rating online P-US and S-US expectancy. Acoustic startle probes were not presented during the reinstatement phase, so ASR was not measure during this phase. Valence and arousal ratings in response to each CS were collected before and after all study phases. After completing all experimental phases participants completed questionnaires about their experiences in the study, including perceived distress related to P-US, S-US, and startle probes, as well as believability of the negative feedback. Upon completing the study participants were informed about the deception involved in the social pain manipulation.

Table 1.
Study 1 Conditioning Procedures Flow Chart

Group	Habituation	Acquisition	Extinction	Reinstatement	Test Phase
Physical/ Physical	CS+ (3) CS- (3)	CS+ / P-US (6) CS- (6) Neutral feedback (2)	CS+ (6) CS- (6)	P-US (4)	CS+ (2) CS- (2)
Physical/ Social	CS+ (3) CS- (3)	CS+ / P-US (6) CS- (6) Neutral feedback (2)	CS+ (6) CS- (6)	S-US (4)	CS+ (2) CS- (2)
Social/ Social	CS+ (3) CS- (3)	CS+ / S-US (6) CS- (6) Neutral feedback (2)	CS+ (6) CS- (6)	S-US (4)	CS+ (2) CS- (2)
Social/ Physical	CS+ (3) CS- (3)	CS+ / S-US (6) CS- (6) Neutral feedback (2)	CS+ (6) CS- (6)	P-US (4)	CS+ (2) CS- (2)

Results

For results of Acquisition, Extinction, and Reinstatement only significant findings are

reported. Tables including means, standard deviations, and all significant and non-significant statistical values are reported in Appendix 2.

Baseline Measures

Mean scores on baseline measures were as follows: Mini-SPIN: $M=8.27$, $SD=1.78$, BDI: $M=8.35$, $SD=6.77$, ASI-X: $M=70$, $SD=20.2$, MAAS: $M=56.77$, $SD=16.61$, STAI: $M=44.69$, $SD=12.30$. One-way ANOVAs revealed no significant differences between the four groups on Mini-SPIN ($F(3,52)=2.13$, $p=0.109$), BDI ($F(3,52)=1.83$, $p=0.153$), ASI-X ($F(3,52)=0.70$, $p=0.556$), MAAS ($F(3,52)=0.948$, $p=0.424$), or STAI ($F(3,52)=0.50$, $p=0.684$). There were also no significant differences among the groups on age ($F(3,52)=0.248$, $p=0.862$), gender ($\chi^2(3, N=57)=2.97$, $p=0.396$), or ethnicity ($\chi^2(12, N=57)=4.67$, $p=0.33$).

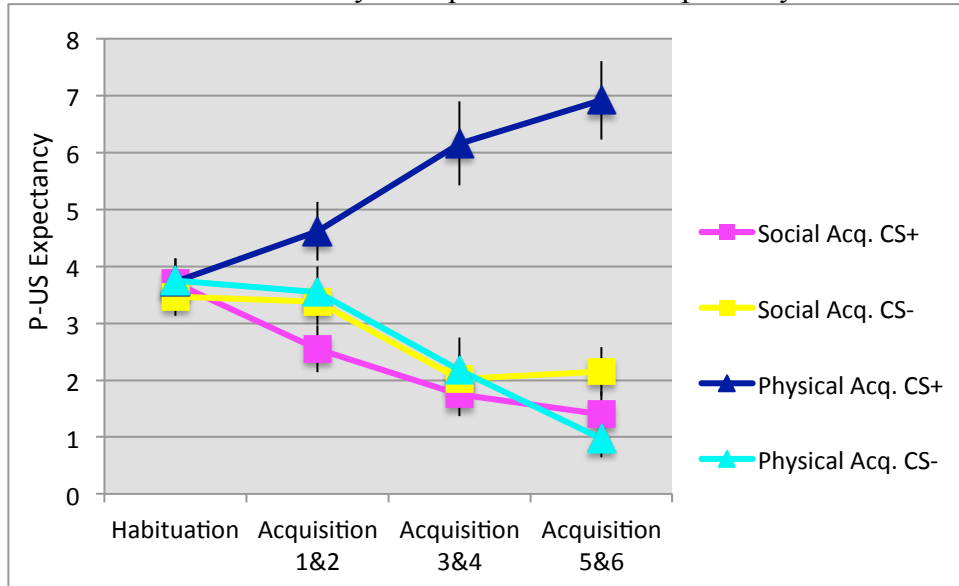
Acquisition

Values of P-US expectancy S-US expectancy, SCR, ASR, and PPG were averaged across the 3 habituation trials, across acquisition trials 1 and 2, across acquisition trials 3 and 4, and across acquisition trials 5 and 6. 2 (Reinforcement; CS+, CS-) x 2 (Acquisition US; Physical, Social) x 4 (Time; Habituation average, Acquisition trials 1 and 2, Acquisition trials 3 and 4, Acquisition trials 5 and 6) mixed-design ANOVAs were conducted for P-US expectancy S-US expectancy, SCR, ASR, and PPG. 2 (Reinforcement; CS+, CS-) x 2 (Acquisition US; Physical, Social) x 2 (Time; Post-Habituation, Post-Acquisition) mixed-design ANOVAs were conducted for Valence ratings and Arousal ratings.

P-US Expectancy: There was a significant three-way Reinforcement x Acquisition US x Time interaction ($F(3,50)=24.69$, $p=0.000$, $\eta^2=0.08$). There were significant two-way Reinforcement x Acquisition US ($F(1,50)=34.57$, $p=0.000$, $\eta^2=0.11$), Reinforcement x Time ($F(3,50)=17.32$, $p=0.000$, $\eta^2=0.05$), and Acquisition US x Time ($F(3,50)=5.48$, $p=0.001$,

$\eta^2=0.03$) interactions. Tests of simple two-way interactions found significant Reinforcement x Time interactions in the P-US Acquisition group ($F(3,27)=29.71, p=0.000, \eta^2=0.17$) and in the S-US Acquisition group ($F(3,23)=2.91, p=0.041, \eta^2=0.02$), significant Reinforcement x Acquisition interactions at Acquisition trials 1 and 2 ($F(1,50)=7.44, p=0.009, \eta^2=0.13$), Acquisition trials 3 and 4 ($F(1,50)=17.32, p=0.000, \eta^2=0.24$) and Acquisition trials 5 and 6 ($F(1,50)=17.32, p=0.000, \eta^2=0.41$), and a significant Acquisition US x Time interaction for the CS+ ($F(3,50)=18.56, p=0.000, \eta^2=0.25$). Tests of simple main effects indicated significantly higher P-US Expectancy to the CS+ in the P-US Acquisition group than to the CS+ in the S-US Acquisition group at Acquisition trials 1 and 2 ($F(1,50)=7.59, p=0.008$), Acquisition trials 3 and 4 ($F(1,50)=22.72, p=0.000$) and Acquisition trials 5 and 6 ($F(1,50)=39.68, p=0.000$). Tests of simple main effects indicated significantly higher CS+ than CS- in the P-US Acquisition group at Acquisition trials 1 and 2 ($F(1,50)=7.59, p=0.029$), Acquisition trials 3 and 4 ($F(1,50)=40.36, p=0.000$) and Acquisition trials 5 and 6 ($F(1,50)=104.72, p=0.000$). Tests of simple main effects indicated a significant effect of Time on the CS+ in the S-US Acquisition group ($F(3,48)=4.01, p=0.013$), the CS- in the S-US Acquisition group ($F(3,48)=3.67, p=0.019$), the CS+ in the P-US Acquisition group ($F(3,48)=8.83, p=0.000$), and the CS- in the P-US Acquisition group ($F(3,48)=12.85, p=0.000$).

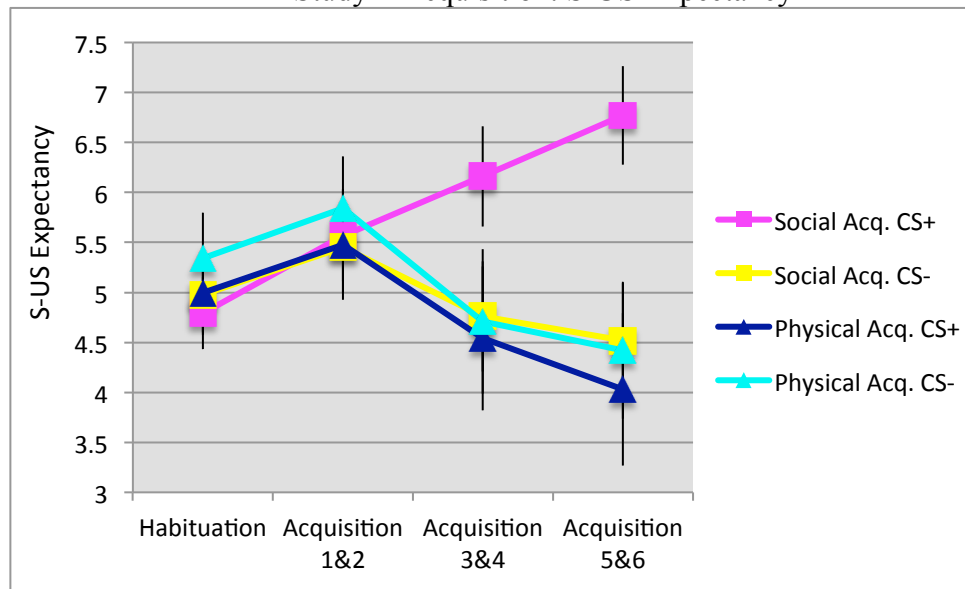
Figure 1.
Study 1 Acquisition: P-US Expectancy



S-US Expectancy: There was a significant three-way Reinforcement x Acquisition US x Time interaction ($F(3,50)=3.55, p=0.016, \eta^2=0.02$). There were significant two-way Reinforcement x Acquisition US ($F(1,50)=4.91, p=0.031, \eta^2=0.02$), Reinforcement x Time ($F(3,50)=3.66, p=0.014, \eta^2=0.02$), and Acquisition US x Time ($F(3,50)=4.29, p=0.006, \eta^2=0.03$) interactions. Tests of simple two-way interactions found significant Reinforcement x Time interactions in the S-US Acquisition group ($F(3,23)=5.49, p=0.002, \eta^2=0.06$), significant Reinforcement x Acquisition interactions at Acquisition trials 5 and 6 ($F(1,50)=6.64, p=0.013, \eta^2=0.11$), and a significant Acquisition US x Time interaction for the CS+ ($F(3,50)=8.08, p=0.000, \eta^2=0.14$). Tests of simple main effects indicated significantly higher S-US Expectancy to the CS+ in the S-US Acquisition group than in the P-US Acquisition group at Acquisition trials 5 and 6 ($F(1,50)=7.18, p=0.010$), and significantly higher CS+ than CS- in the S-US Acquisition group at Acquisition trials 3 and 4 ($F(1,50)=4.91, p=0.031$) and Acquisition trials 5 and 6 ($F(1,50)=8.98, p=0.004$). Tests of simple main effects indicated a significant effect of

Time on the CS+ in the S-US Acquisition group ($F(3,48)=3.37, p=0.026$), the CS+ in the P-US Acquisition group ($F(3,48)=1.22, p=0.036$), and the CS- in the P-US Acquisition group ($F(3,48)=2.98, p=0.041$).

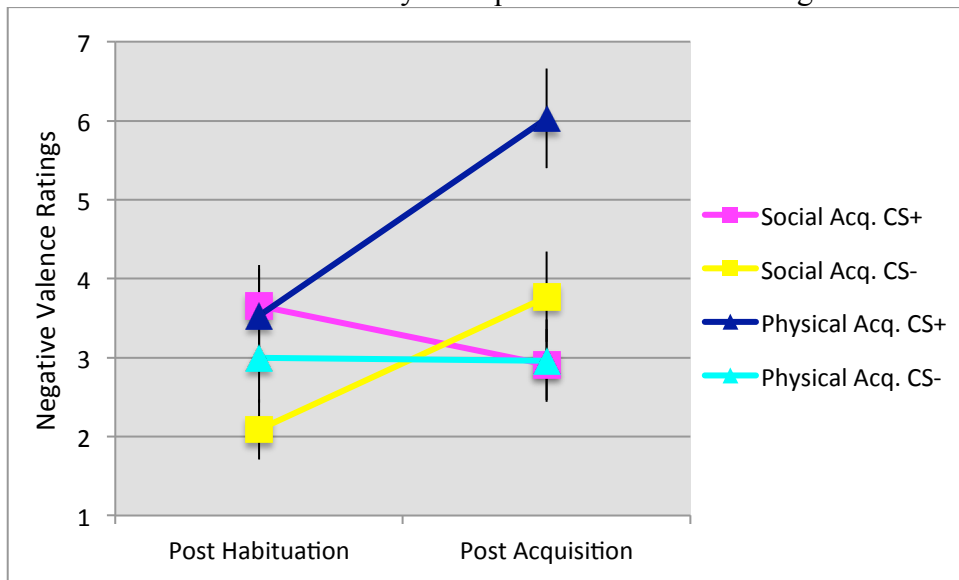
Figure 2.
Study 1 Acquisition: S-US Expectancy



Valence Ratings: There was a significant three-way Reinforcement x Acquisition US x Time interaction ($F(1,48)=13.03, p=0.00, \eta^2=0.10$) and a two-way Reinforcement x Acquisition US interaction ($F(1,48)=8.36, p=0.006, \eta^2=0.04$). Tests of simple two-way interactions found significant Reinforcement x Time interactions in the P-US Acquisition group ($F(1,27)=8.40, p=0.009, \eta^2=0.14$) and in the S-US Acquisition group ($F(1,21)=6.96, p=0.014, \eta^2=0.09$), significant Reinforcement x Acquisition interactions at Post-Acquisition ($F(1,50)=23.03, p=0.000, \eta^2=0.28$), and a significant Acquisition US x Time interaction for the CS+ ($F(1,50)=12.60, p=0.001, \eta^2=0.19$) and CS- ($F(1,50)=7.00, p=0.011, \eta^2=0.12$). Tests of simple main effects indicated significantly higher negative valence to the CS+ in the P-US Acquisition group than to CS+ in the S-US Acquisition group at Post-Acquisition ($F(1,48)=13.94, p=0.001$). Tests of simple main effects indicated significantly higher negative valence to CS- than CS+ in

the S-US Acquisition group at Post-Habituation ($F(1,48)=4.27, p=0.044$) and significantly higher CS+ than CS- in the P-US Acquisition group at Post-Acquisition ($F(1,48)=32.63, p=0.000$). Tests of simple main effects indicated a significant effect of Time on CS- in the S-US Acquisition group ($F(1,48)=1.31, p=0.015$), and on CS+ in the P-US Acquisition group ($F(1,48)=16.36, p=0.000$).

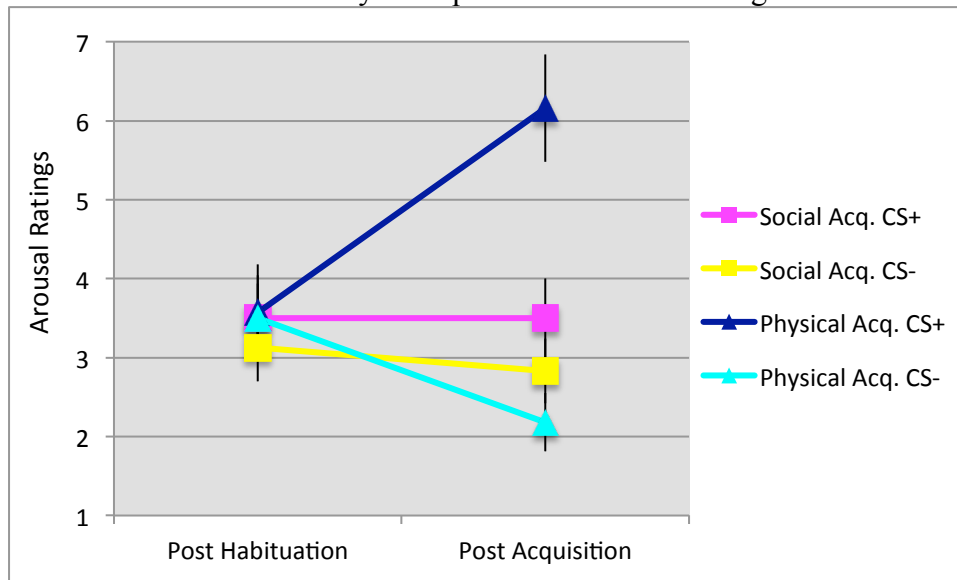
Figure 3.
Study 1 Acquisition: Valence Ratings



Arousal Ratings: There was a significant three-way Reinforcement x Acquisition US x Time interaction ($F(1,49)=9.8, p=0.03, \eta^2=0.08$), and two-way Reinforcement x Acquisition US ($F(1,49)=8.51, p=0.005, \eta^2=0.11$) and Reinforcement x Time ($F(1,49)=20.33, p=0.000, \eta^2=0.11$) interactions. Tests of simple two-way interactions found significant Reinforcement x Time interactions in the P-US Acquisition group ($F(1,27)=26.23, p=0.000, \eta^2=0.22$), a significant Reinforcement x Acquisition interaction at Post-Acquisition ($F(1,50)=15.60, p=0.000, \eta^2=0.16$), and a significant Acquisition US x Time interaction for the CS+ ($F(1,50)=9.35, p=0.004, \eta^2=0.14$). Tests of simple main effects indicated significantly higher arousal ratings to the CS+ in the P-US Acquisition group than to CS+ in the S-US Acquisition group at Post-Acquisition

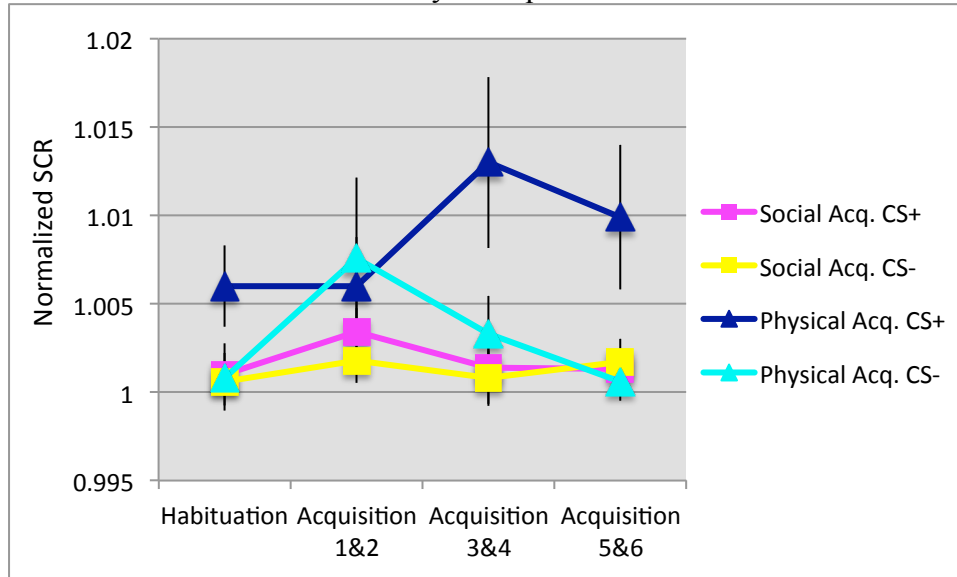
($F(1,50)=8.26, p=0.006$). Tests of simple main effects indicated significantly higher arousal ratings to the CS+ than CS- in the P-US Acquisition group at Post-Acquisition ($F(1,50)=48.89, p=0.000$). Tests of simple main effects indicated a significant effect of Time on CS+ in the P-US Acquisition group ($F(1,50)=20.26, p=0.004$) and CS- in the P-US Acquisition group ($F(1,50)=9.15, p=0.000$).

Figure 4.
Study 1 Acquisition: Arousal Ratings



SCR: There were no significant two- or three-way interactions. There was a significant main effect of Reinforcement with CS+ higher than CS- ($F(1,38)=4.91, p=0.032, \eta^2=0.02$), and a significant main effect of Acquisition US with P-US Acquisition group higher than S-US Acquisition group ($F(1,40)=5.12, p=0.029, \eta^2=0.06$). Test of simple main effects indicated significantly higher CS+ than CS- in the P-US Acquisition group at Acquisition trials 3 and 4 ($F(1,40)=9.86, p=0.003$) and Acquisition trials 5 and 6 ($F(1,40)=7.05, p=0.011$).

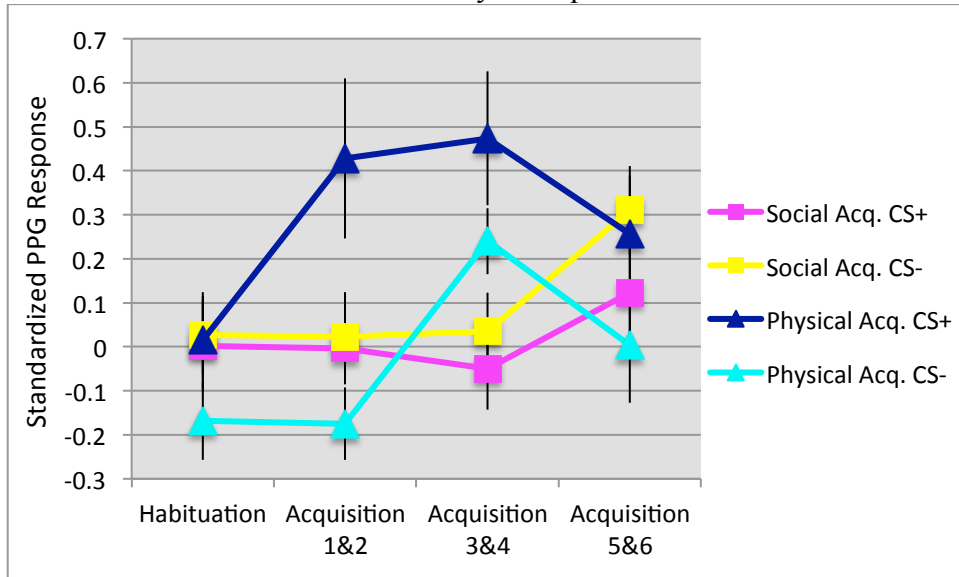
Figure 5.
Study 1 Acquisition: SCR



ASR: There were no significant two- or three-way interactions, main effects, simple two-way interaction or simple main effects.

PPG: There was no significant three-way interaction, but there was a significant two-way Reinforcement x Acquisition US interaction ($F(1,47)=14.23, p=0.000, \eta^2=0.05$). Tests of simple two-way interactions found significant Reinforcement x Time interactions in the P-US Acquisition group ($F(3,27)=4.08, p=0.009, \eta^2=0.04$) as well as significant Reinforcement x Acquisition interactions at Acquisition trials 1 and 2 ($F(1,50)=7.44, p=0.010, \eta^2=0.12$) and Acquisition trials 3 and 4 ($F(1,50)=18.19, p=0.000, \eta^2=0.23$). Tests of simple main effects indicated significantly higher PPG response to the CS+ in the P-US Acquisition group than in the S-US Acquisition group at Acquisition trials 1 and 2 ($F(1,49)=3.95, p=0.050$), Acquisition trials 3 and 4 ($F(1,49)=7.31, p=0.009$), and significantly higher PPG response to the CS- in the S-US Acquisition group than the CS- in the P-US Acquisition group at Acquisition trials 5 and 6 ($F(1,49)=37.94, p=0.007$).

Figure 6.
Study 1 Acquisition: PPG



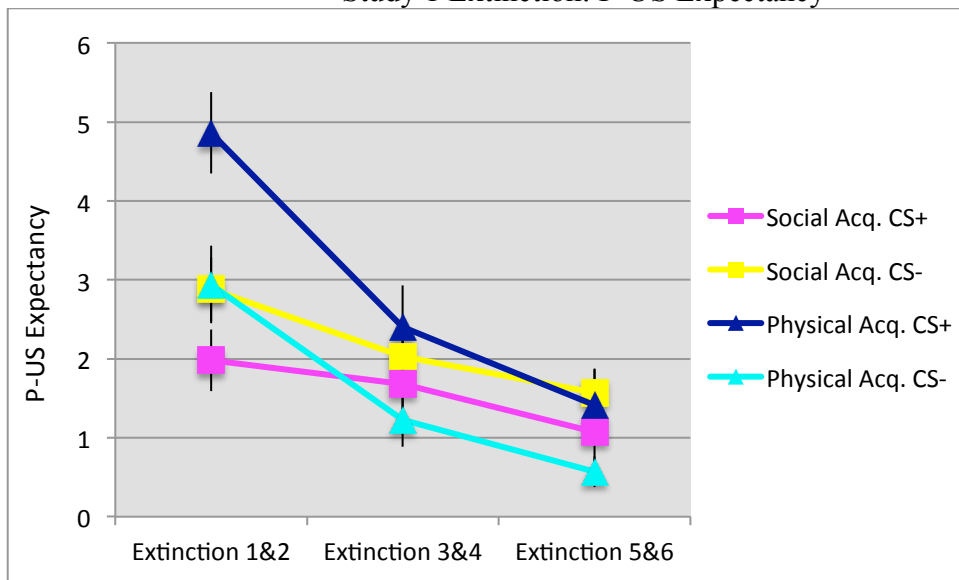
Extinction

Values of P-US expectancy, S-US expectancy, SCR, ASR, and PPG were averaged across acquisition extinction trials 1 and 2, across extinction trials 3 and 4, and across extinction trials 5 and 6. 2 (Reinforcement; CS+, CS-) x 2 (Acquisition US; Physical, Social) x 3 (Time; Extinction trials 1 and 2, Extinction trials 3 and 4, Extinction trials 5 and 6) mixed-design ANOVAs were conducted for P-US Expectancy, S-US Expectancy, SCR, ASR, and PPG. 2 (Reinforcement; CS+, CS-) x 2 (Acquisition US; Physical, Social) x 2 (Time; Post-Acquisition, Post-Extinction) mixed-design ANOVAs were conducted for Valence ratings and Arousal ratings.

P-US Expectancy: There was no significant three-way interaction, but there were significant two-way Reinforcement x Acquisition US ($F(1,46)=10.83, p=0.002, \eta^2=0.06$), and Acquisition US x Time ($F(2,45)=6.70, p=0.002, \eta^2=0.04$) interactions. There was a significant main effect of Time ($F(1,46)=30.11, p=0.000, \eta^2=0.19$). Tests of simple main effects indicated significantly higher P-US Expectancy to CS+ in the P-US Acquisition group than in the S-US Acquisition group at Extinction trials 1 and 2 ($F(1,46)=15.08, p=0.000$). Tests of simple main

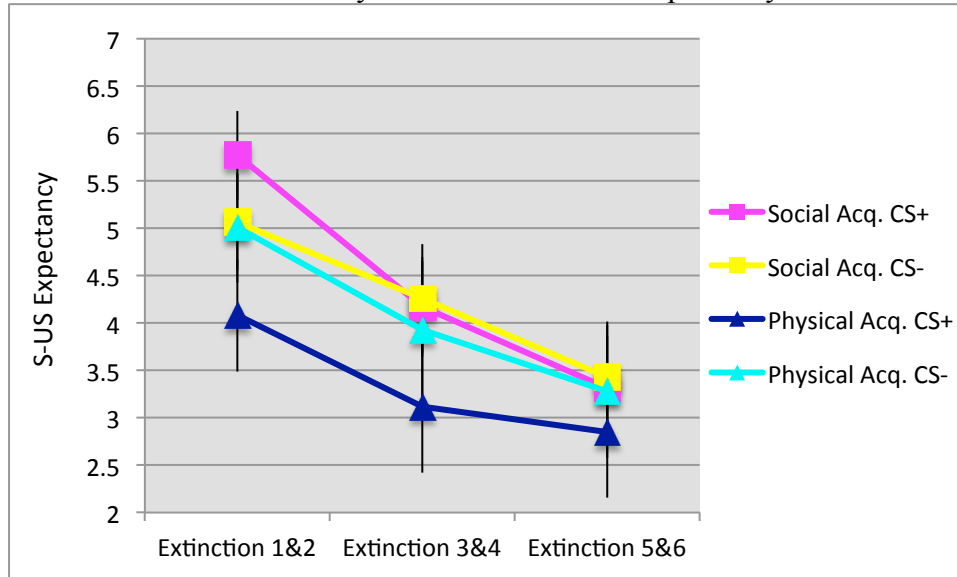
effects indicated significantly higher CS+ than CS- in the P-US Acquisition group at Extinction trials 1 and 2 ($F(1,46)=9.66, p=0.003$), Extinction trials 3 and 4 ($F(1,46)=7.67, p=0.008$) and Extinction trials 5 and 6 ($F(1,46)=4.92, p=0.032$). Tests of simple main effects indicated a significant effect of Time on the CS+ in the P-US Acquisition group ($F(2,45)=30.78, p=0.000$), the CS- in the P-US Acquisition group ($F(2,45)=11.91, p=0.000$), and the CS- in the S-US Acquisition group ($F(2,45)=3.17, p=0.050$).

Figure 7.
Study 1 Extinction: P-US Expectancy



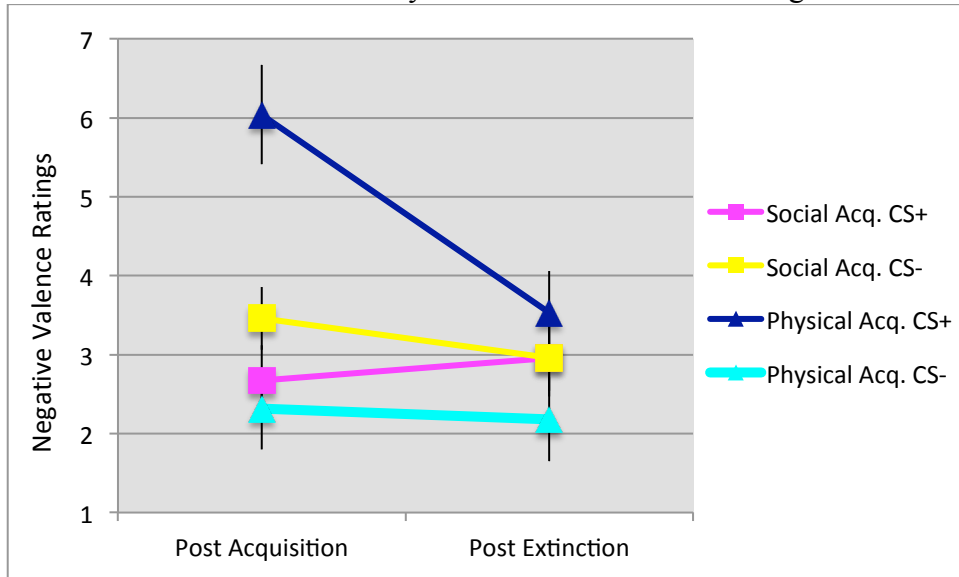
S-US Expectancy: There were no significant two- or three-way interactions. There was a significant main effect of Time ($F(2,50)=19.88, p=0.000, \eta^2=0.17$). Tests of simple main effects indicated a significant effect of Time on the CS+ in the S-US Acquisition group ($F(2,49)=8.09, p=0.001$), the CS- in the S-US Acquisition group ($F(2,45)=5.35, p=0.008$), and the CS- in the P-US Acquisition group ($F(2,45)=6.53, p=0.003$).

Figure 8.
Study 1 Extinction: S-US Expectancy



Valence Ratings: There was a significant three-way Reinforcement x Acquisition US x Time interaction ($F(1,50)=9.94, p=0.003, \eta^2=0.04$) as well as significant two-way Reinforcement x Acquisition US ($F(1,50)=16.83, p=0.000, \eta^2=0.13$) and Acquisition US x Time ($F(1,50)=7.50, p=0.009, \eta^2=0.02$) interactions. Tests of simple two-way interactions found a significant Reinforcement x Time interactions in the P-US Acquisition group ($F(1,27)=9.49, p=0.005, \eta^2=0.05$), a significant Reinforcement x Acquisition interaction at Post-Acquisition ($F(1,50)=23.03, p=0.000, \eta^2=0.28$), and a significant Acquisition US x Time interaction for the CS+ ($F(1,50)=12.59, p=0.001, \eta^2=0.18$). Tests of simple main effects indicated significantly higher negative valence to the CS+ in the P-US Acquisition group than in the S-US Acquisition group at Post-Acquisition ($F(1,48)=13.94, p=0.001$). Tests of simple main effects indicated significantly higher CS+ than CS- in the P-US Acquisition group at Post-Acquisition ($F(1,50)=33.9, p=0.000$) and Post-Extinction ($F(1,50)=6.25, p=0.016$), and indicated a significant effect of Time on the CS+ in the P-US Acquisition group ($F(1,50)=21.88, p=0.000$).

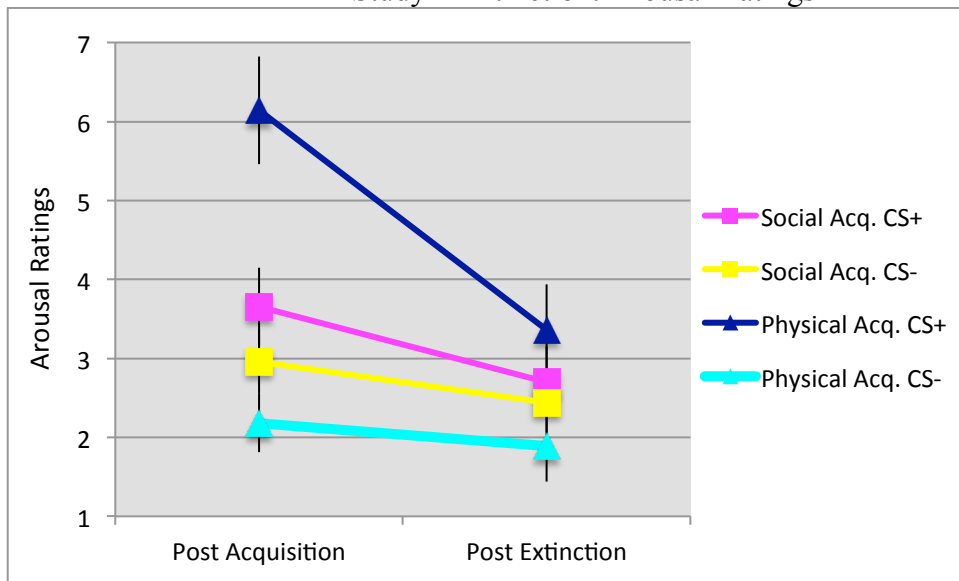
Figure 9.
Study 1 Extinction: Valence Ratings



Arousal Ratings: There was a significant three-way Reinforcement x Acquisition US x Time interaction ($F(1,50)=4.23, p=0.033, \eta^2=0.02$) as well as two-way Reinforcement x Acquisition US ($F(1,50)=15.55, p=0.000, \eta^2=0.8$) and Reinforcement x Time ($F(1,50)=4.83, p=0.003, \eta^2=0.08$) interactions. Tests of simple two-way interactions found significant Reinforcement x Time interactions in the P-US group ($F(1,27)=10.48, p=0.003, \eta^2=0.06$), a significant Reinforcement x Acquisition interactions at Post-Acquisition ($F(1,50)=15.60, p=0.000, \eta^2=0.16$) and Post-Extinction ($F(1,50)=4.05, p=0.050, \eta^2=0.06$), and a significant Acquisition US x Time interaction for the CS+ ($F(1,50)=5.27, p=0.026, \eta^2=0.07$). Tests of simple main effects indicated significantly higher Arousal to the CS+ in the P-US Acquisition group than in the S-US Acquisition group at Post-Acquisition ($F(1,49)=7.21, p=0.010$). Tests of simple main effects indicated significantly higher CS+ than CS- in the P-US Acquisition group at Post-Acquisition ($F(1,49)=47.9, p=0.000$) and Post-Extinction ($F(1,49)=13.29, p=0.001$), and

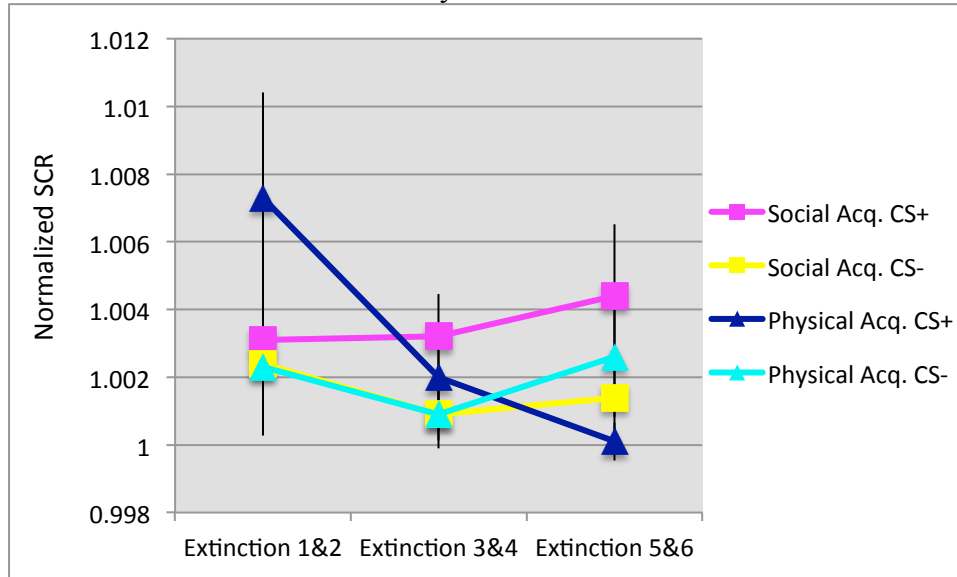
indicated a significant effect of Time on the CS+ in the P-US Acquisition group ($F(1,49)=24.90$, $p=0.000$).

Figure 10.
Study 1 Extinction: Arousal Ratings



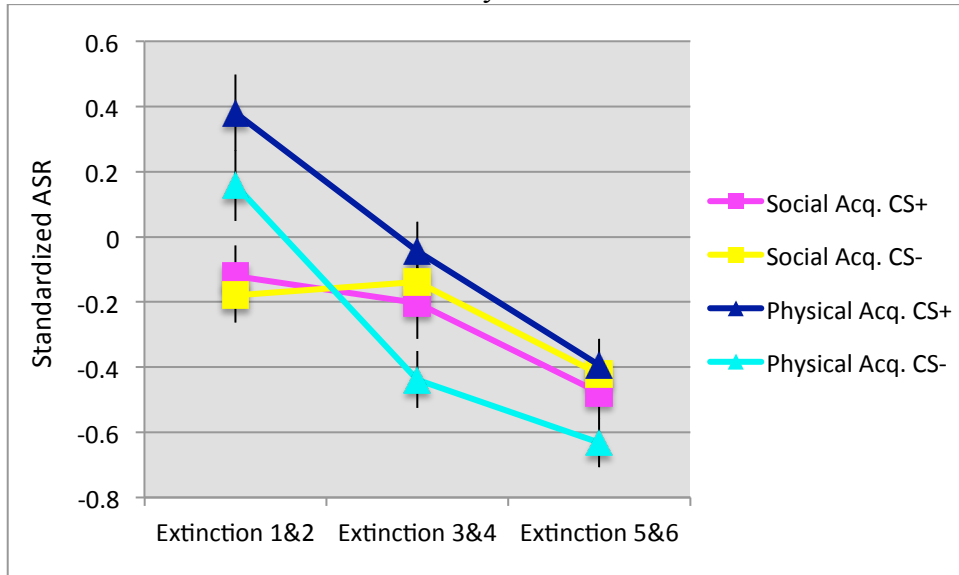
SCR: There were no significant two- or three-way interactions or main effects. Tests of simple two-way interactions found a significant Reinforcement x Time interactions in the P-US Acquisition group ($F(1,23)=4.09$, $p=0.038$). Tests of simple main effects indicated a significant effect of Time on the CS+ in the P-US Acquisition group ($F(2,23)=3.66$, $p=0.042$).

Figure 11.
Study 1 Extinction: SCR



ASR: There was no significant three-way interaction, but there were significant two-way Reinforcement x Acquisition US ($F(1,44)=12.62, p=0.001, \eta^2=0.02$) and Acquisition US x Time ($F(2,44)=5.26, p=0.007, \eta^2=0.05$) interactions. Tests of simple two-way interactions found a significant Reinforcement x Acquisition US interaction at Extinction trials 3 and 4 ($F(2,44)=2.81, p=0.027, \eta^2=0.10$), and a significant Acquisition US x Time interaction for the CS- ($F(2,44)=4.45, p=0.014, \eta^2=0.08$). Tests of simple main effects indicated significantly higher ASR to the CS+ in the P-US Acquisition group than in the S-US Acquisition group at Extinction trials 1 and 2 ($F(1,44)=11.81, p=0.001$), significantly higher ASR to the CS- in the P-US Acquisition group than in the S-US Acquisition group at Extinction trials 1 and 2 ($F(1,44)=4.77, p=0.034$), and significantly higher CS+ than CS- in the P-US Acquisition group at Extinction trials 3 and 4 ($F(1,44)=8.22, p=0.006$). Tests of simple main effects indicated a significant effect of Time on the CS+ in the P-US Acquisition group ($F(2,43)=13.03, p=0.000$), and the CS- in the P-US Acquisition group ($F(2,43)=16.54, p=0.000$).

Figure 12.
Study 1 Extinction: ASR



PPG: There were no significant two- or three-way interactions or main effects. There were no simple two-way interactions or simple main effects.

Reinstatement

Values of P-US expectancy, S-US expectancy, SCR, ASR, and PPG were averaged across extinction trials 5 and 6 and across post-reinstatement test trials 1 and 2. Four different mixed-design ANOVAs were conducted for P-US Expectancy, S-US Expectancy, SCR, ASR, and PPG. One model examined the effect of acquisition US on reinstatement: 2 (*Reinforcement*; CS+, CS-) x 2 (*Acquisition US*; Physical, Social) x 2 (*Time*; Extinction trials 5 and 6, Test trials 1 and 2). One model examined the effect of reinstatement US on reinstatement: 2 (*Reinforcement*; CS+, CS-) x 2 (*Reinstatement US*; Physical, Social) x 2 (*Time*; Extinction trials 5 and 6, Test trials 1 and 2). One model examined the effect of match in acquisition and reinstating US (same versus different) on reinstatement: 2 (*Reinforcement*; CS+, CS-) x 2 (*Match*; Same, Different) x 2 (*Time*; Extinction trials 5 and 6, Test trials 1 and 2). One model examined the effect of the

combination of acquisition and reinstatement USs on reinstatement: 2 (*Reinforcement*; CS+, CS-) x 4 (*Group*; Physical/Physical, Physical/Social, Social/Social, Social/Physical) x 2 (*Time*; Extinction trials 5 and 6, Test trials 1 and 2).

Four different mixed-design ANOVAs were conducted for Valence ratings and Arousal ratings. One model examined the effect of acquisition US on reinstatement: 2 (*Reinforcement*; CS+, CS-) x 2 (*Acquisition US*; Physical, Social) x 2 (*Time*; Post-Extinction, Post-Reinstatement). One model examined the effect of reinstatement US on reinstatement: 2 (*Reinforcement*; CS+, CS-) x 2 (*Reinstatement US*; Physical, Social) x 2 (*Time*; Post-Extinction, Post-Reinstatement). One model examined the effect of match in acquisition and reinstating US (same versus different) on reinstatement: 2 (*Reinforcement*; CS+, CS-) x 2 (*Match*; Same, Different) x 2 (*Time*; Post-Extinction, Post-Reinstatement). One model examined the effect of the combination of acquisition and reinstatement USs on reinstatement: 2 (*Reinforcement*; CS+, CS-) x 4 (*Group*; Physical/Physical, Physical/Social, Social/Social, Social/Physical) x 2 (*Time*; Post-Extinction, Post-Reinstatement).

P-US Expectancy

Reinforcement x Acquisition US x Time: There was no significant three-way interaction, but there was a significant two-way Reinforcement x Acquisition US interaction ($F(1,46)=5.91$, $p=0.019$, $\eta^2=0.03$). There was a significant main effect of Time ($F(1,46)=19.88$, $p=0.000$, $\eta^2=0.18$). Tests of simple main effects indicated significantly higher CS+ than CS- in the P-US Acquisition group at Extinction trials 5 and 6 ($F(1,46)=4.92$, $p=0.032$). Tests of simple main effects indicated a significant effect of Time on CS+ in the P-US Acquisition group ($F(1,46)=6.91$, $p=0.012$), CS+ in the S-US Acquisition group ($F(1,46)=5.78$, $p=0.020$), CS- in

the P-US Acquisition group ($F(1,46)=11.50, p=0.001$), and CS- in the S-US Acquisition group ($F(1,46)=6.45, p=0.015$).

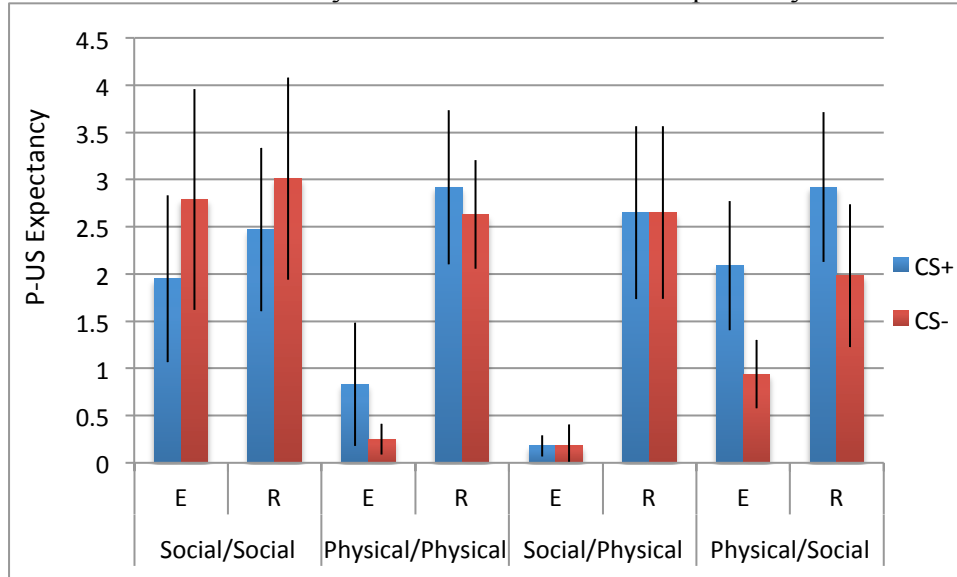
Reinforcement x Reinstatement US x Time: There was no significant three-way interaction, but there was a significant two-way Reinstatement US x Time interaction ($F(1,46)=6.93, p=0.012, \eta^2=0.06$). There was a significant main effect of Time ($F(1,46)=22.13, p=0.000, \eta^2=0.18$). Tests of simple main effects indicated a significant effect of Time on CS+ in the P-US Reinstatement group ($F(1,46)=16.11, p=0.000$), and CS- in the P-US Reinstatement group ($F(1,46)=25.13, p=0.000$).

Reinforcement x Match x Time: There were no significant two- or three-way interactions. There was a significant main effect of Time ($F(1,46)=22.13, p=0.000, \eta^2=0.18$). Tests of simple main effects indicated a significant effect of Time on the CS+ in the Same group ($F(1,46)=5.74, p=0.021$), CS+ in the Different group ($F(1,46)=7.03, p=0.011$), CS- in the Same group ($F(1,46)=7.23, p=0.010$), and CS- in the Different group ($F(1,46)=10.73, p=0.002$).

Reinforcement x Group x Time: There were no significant two- or three-way interactions, although there were marginally significant two-way Reinforcement x Group ($p=0.087$) and Group x Time ($p=0.081$) interactions. There was a significant main effect of Time ($F(1,44)=21.33, p=0.000, \eta^2=0.18$). There were no significant simple two-way interactions. Tests of simple main effects indicated significantly higher P-US Expectancy to the CS- in the Social/Social group than in other groups at Extinction trials 5 and 6 ($F(3,44)=3.89, p=0.015$), and significantly higher CS+ than CS- in the Physical/Social group at Extinction trials 5 and 6 ($F(1,44)=4.17, p=0.047$). Tests of simple main effects indicated a significant effect of Time on the CS+ in the Physical/Physical group ($F(1,44)=7.41, p=0.009$), CS+ in the Social/Physical

group ($F(1,44)=8.17, p=0.006$), CS- in the Physical/Physical group ($F(1,44)=12.42, p=0.001$), and CS- in the Social/Physical group ($F(1,44)=12.05, p=0.001$).

Figure 13.
Study 1 Reinstatement: P-US Expectancy



S-US Expectancy

Reinforcement x Acquisition US x Time: There was no significant three-way interaction, and no significant two-way interactions, although there was a marginally significant Acquisition US x Time interaction ($p=0.069$). There was a significant main effect of Time ($F(1,50)=6.69, p=0.013, \eta^2=0.06$). Tests of simple main effects indicated significantly higher S-US Expectancy to the CS+ in the S-US Acquisition group than in the P-US Acquisition group at Test trials 1 and 2 ($F(1,50)=4.06, p=0.049$), and indicated a significant effect of Time on the CS+ in the S-US Acquisition group ($F(1,50)=10.88, p=0.002$).

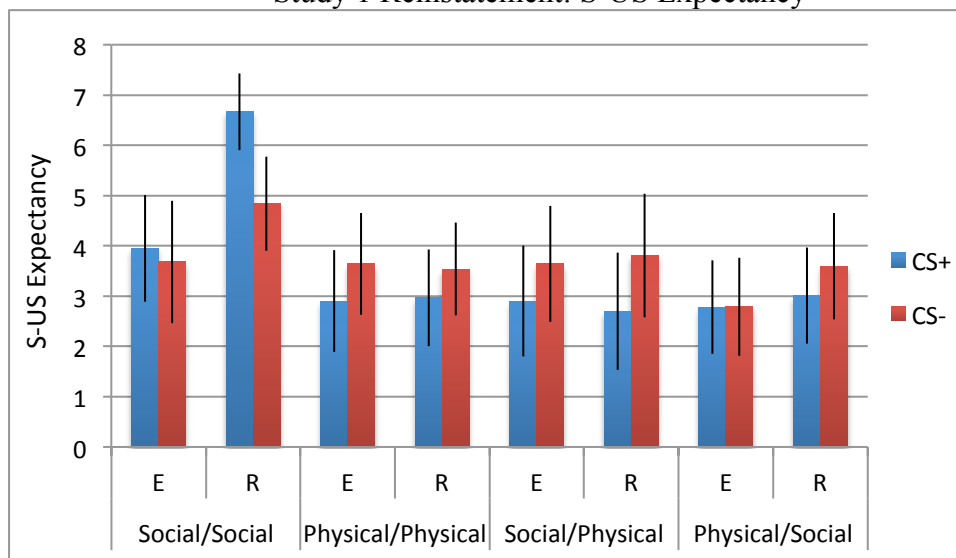
Reinforcement x Reinstatement US x Time: There were no significant two- or three-way interactions, although there was a marginally significant Reinforcement x Reinstatement US ($F(1,50)=3.31, p=0.075$) interaction. There was a significant main effect of Time ($F(1,50)=6.47, p=0.014, \eta^2=0.06$). Tests of simple main effects indicated a significant effect of Time on CS+ in

the S-US Reinstatement group ($F(1,50)=7.62, p=0.008$), and CS- in the S-US Reinstatement group ($F(1,46)=4.58, p=0.037$).

Reinforcement x Match x Time: There were no significant two- or three-way interactions. There was a significant main effect of Time ($F(1,50)=6.47, p=0.014, \eta^2=0.06$). Tests of simple main effects indicated a significant effect of Time on the CS+ in the Same group ($F(1,50)=5.79, p=0.020$).

Reinforcement x Group x Time: There were no significant two- or three-way interactions. There was a significant main effect of Time ($F(1,48)=7.04, p=0.011, \eta^2=0.06$). Tests of simple two-way interactions found a significant Reinforcement x Time interaction in the Social/Social group ($F(1,11)=4.62, p=0.050, \eta^2=0.03$), a significant Reinforcement x Group interaction at Test trials 1 and 2 ($F(3,48)=2.91, p=0.044, \eta^2=0.15$), and a significant Group x Time interaction for CS+ ($F(3,48)=3.00, p=0.040, \eta^2=0.14$). Tests of simple main effects indicated significantly higher S-US Expectancy to the CS+ in the Social/Social group than in the other groups at Test trials 1 and 2 ($F(3,48)=3.16, p=0.033$), significantly higher CS+ than CS- in the Social/Social group at Test trials 1 and 2 ($F(1,48)=6.78, p=0.012$), and a significant effect of Time on the CS+ in the Social/Social group ($F(1,48)=14.12, p=0.000$).

Figure 14.
Study 1 Reinstatement: S-US Expectancy



Valence Ratings

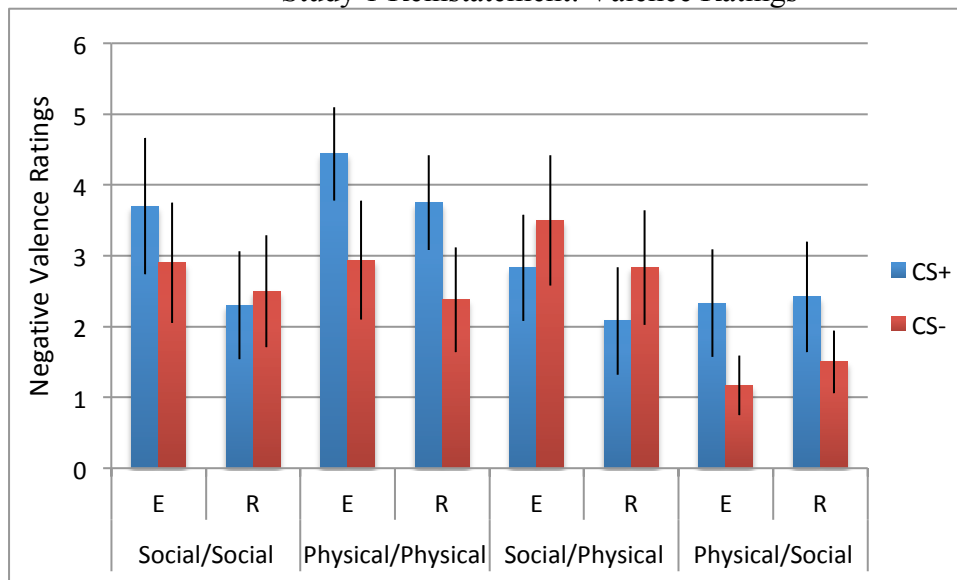
Reinforcement x Acquisition US x Time: There was no significant three-way interaction, but there was a significant two-way Reinforcement x Acquisition US interaction ($F(1,48)=7.15$, $p=0.010$, $\eta^2=0.05$). There was a significant main effect of Time ($F(1,48)=5.35$, $p=0.025$, $\eta^2=0.03$). Tests of simple main effects indicated significantly higher negative valence ratings to the CS+ than CS- in the P-US Acquisition group at Post-Extinction ($F(1,48)=6.00$, $p=0.018$) and at Post-Reinstatement ($F(1,48)=7.54$, $p=0.008$).

Reinforcement x Reinstatement US x Time: There were no significant two- or three-way interactions. There were significant main effects of Reinforcement ($F(1,48)=4.04$, $p=0.050$, $\eta^2=0.04$) and Time ($F(1,48)=4.26$, $p=0.044$, $\eta^2=0.02$). There were no significant simple two-way interactions or simple main effects.

Reinforcement x Match x Time: There were no significant two- or three-way interactions. There were significant main effects of Reinforcement ($F(1,48)=4.04$, $p=0.050$, $\eta^2=0.04$) and Time ($F(1,48)=4.26$, $p=0.044$, $\eta^2=0.02$).

Reinforcement x Group x Time: There was no significant three-way interaction, but there was a significant two-way Reinforcement x Time interaction. ($F(3,46)=2.92, p=0.044, \eta^2=0.07$) There was a significant main effect of Time ($F(1,46)=4.77, p=0.034, \eta^2=0.02$). Tests of simple main effects indicated significantly higher valence ratings to the CS+ than CS- in the Physical/Physical group at Post-Extinction ($F(1,46)=4.14, p=0.048$), and Post-Reinstatement.

Figure 15.
Study 1 Reinstatement: Valence Ratings



Arousal Ratings

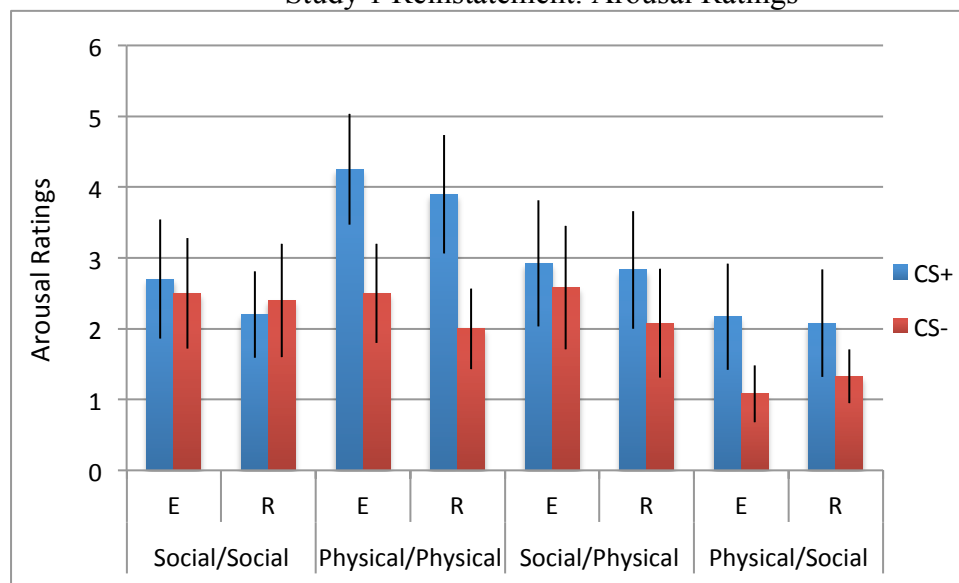
Reinforcement x Acquisition US x Time: There was no significant three-way interaction, but there was a significant two-way Reinforcement x Acquisition US interaction ($F(1,48)=4.23, p=0.045, \eta^2=0.04$). There was a significant main effect of Reinforcement ($F(1,48)=9.70, p=0.003, \eta^2=0.09$). Tests of simple main effects indicated significantly higher arousal ratings to the CS+ than CS- in the P-US Acquisition group at Post-Extinction ($F(1,48)=13.02, p=0.001$) and at Post-Reinstatement ($F(1,48)=11.35, p=0.001$).

Reinforcement x Reinstatement US x Time: There were no significant two- or three-way interactions. There were significant main effects of Reinforcement ($F(1,48)=9.74, p=0.003, \eta^2=0.09$). Tests of simple main effects indicated significantly higher arousal ratings to the CS+ than CS- in the P-US Reinstatement group at Post-Extinction ($F(1,48)=7.43, p=0.009$) and Post-Reinstatement ($F(1,48)=11.35, p=0.001$), and a significant effect of Time on the CS- in the P-US Reinstatement group ($F(1,48)=4.37, p=0.042$).

Reinforcement x Match x Time: There were no significant two- or three-way interactions. There were significant main effects of Reinforcement ($F(1,48)=9.74, p=0.003, \eta^2=0.10$).

Reinforcement x Group x Time: There were no significant two- or three-way interactions, but there was a significant main effect of Reinforcement ($F(1,46)=8.58, p=0.005, \eta^2=0.08$). Tests of simple main effects indicated significantly higher arousal ratings to the CS+ than CS- in the Physical/Physical group at Post-Extinction ($F(1,46)=10.33, p=0.002$) and Post-Reinstatement ($F(1,46)=12.17, p=0.001$).

Figure 16.
Study 1 Reinstatement: Arousal Ratings



SCR

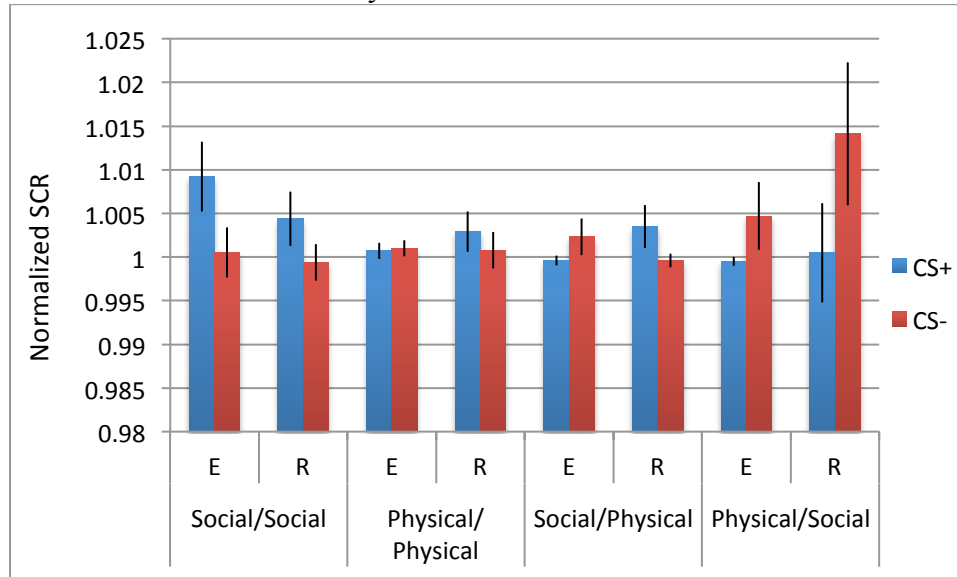
Reinforcement x Acquisition US x Time: There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects.

Reinforcement x Reinstatement US x Time: There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects.

Reinforcement x Match x Time: There was a significant two-way Reinforcement by Match interaction ($F(1,43)=6.47, p=0.050, \eta^2=0.04$). There were no significant simple two-way interactions or simple main effects.

Reinforcement x Group x Time: There were no significant three-way interactions, but there was a significant two-way Reinforcement x Group ($F(3,41)=2.89, p=0.047, \eta^2=0.03$) interaction. There were no significant main effects. There were no significant simple two-way interactions, although there was a marginally significant Reinforcement by Time interaction in the P-US Reinstatement group ($F(1,22)=4.10, p=0.055, \eta^2=0.02$). Tests of simple main effects indicated significantly higher SCR to CS- than CS+ in the Physical/Social group at Test trials 1 and 2 ($F(1,41)=5.57, p=0.023$).

Figure 17.
Study 1 Reinstatement: SCR



ASR

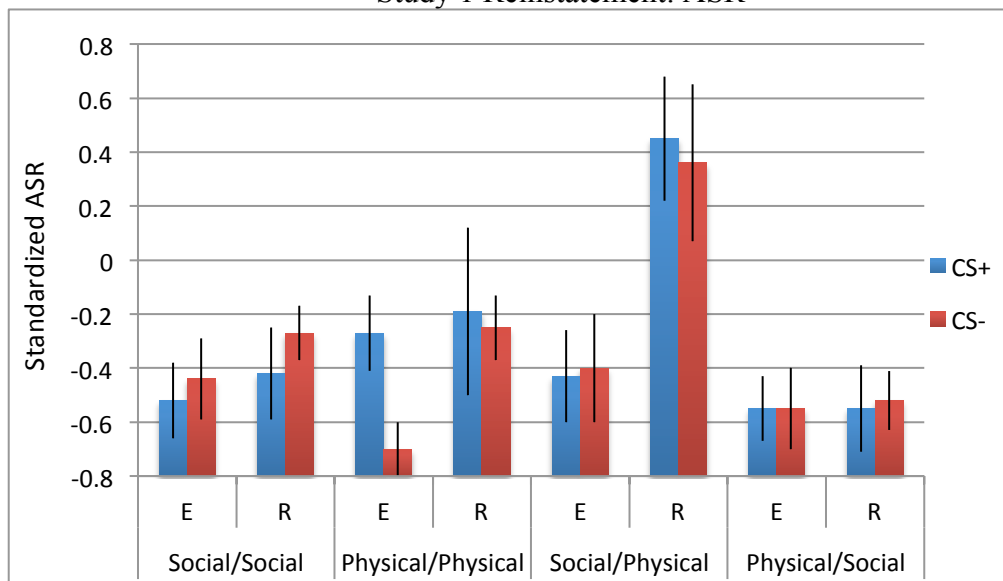
Reinforcement x Acquisition US x Time: There were no significant two- or three-way interactions. There were significant main effects of Time ($F(1,44)=10.32, p=0.002, \eta^2=0.08$). There were no significant simple two-way interactions or simple main effects.

Reinforcement x Reinstatement US x Time: There was no significant three-way interactions, but there was a significant two-way Reinstatement US x Time interaction ($F(1,44)=5.47, p=0.024, \eta^2=0.04$). Tests of simple main effects indicated significantly higher ASR to CS+ in the P-US Reinstatement group than in the S-US Reinstatement group at Test trials 1 and 2 ($F(1,44)=6.94, p=0.012$), and significantly higher ASR to CS- in the P-US Reinstatement group than CS- in the S-US Reinstatement group at Test trials 1 and 2 ($F(1,44)=4.76, p=0.035$). Tests of simple main effects also indicated a significant effect of Time on the CS+ in the P-US Reinstatement group ($F(1,44)=4.86, p=0.033$), and a significant effect of Time on the CS- in the P-US Reinstatement group ($F(1,44)=17.29, p=0.000$).

Reinforcement x Match x Time: There were no significant two- or three-way interactions. There were significant main effects of Time ($F(1,44)=10.32, p=0.002, \eta^2=0.08$). There were no significant simple two-way interactions or simple main effects.

Reinforcement x Group x Time: There were no significant three-way interactions, but there was a significant two-way Group x Time interaction ($F(1,42)=3.80, p=0.017, \eta^2=0.08$). There were significant main effects of Group ($F(3,42)=5.71, p=0.002, \eta^2=0.15$) and Time ($F(1,42)=13.16, p=0.001, \eta^2=0.09$). There were no significant simple two-way interactions. Tests of simple main effects indicated significantly higher ASR to CS+ in the Social/Physical group than in other groups ($F(3,42)=4.14, p=0.012$), significantly higher ASR to CS- in the Social/Physical group than in other groups ($F(3,42)=4.43, p=0.009$), and significantly higher CS+ than CS- in the Physical/Physical group at Extinction trials 5 and 6 ($F(3,42)=4.43, p=0.24$). Tests of simple main effects also indicated a significant effect of Time on the CS+ in the Social/Physical group ($F(1,42)=10.02, p=0.003$), CS- in the Social Physical group ($F(1,42)=12.07, p=0.016$), and CS- in the Physical/Physical Group ($F(1,42)=6.33, p=0.001$).

Figure 18.
Study 1 Reinstatement: ASR



PPG

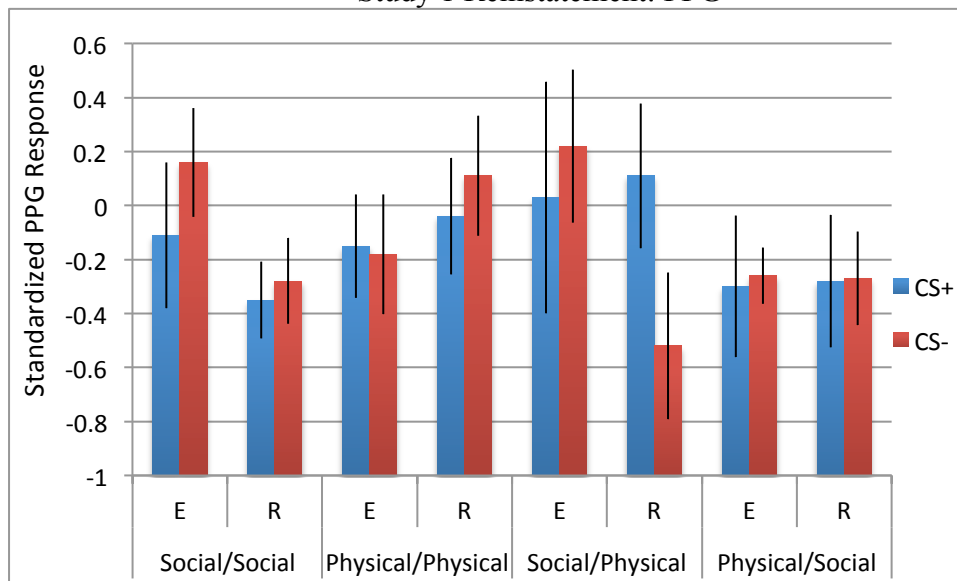
Reinforcement x Acquisition US x Time: There were no significant two- or three-way interactions, main effects, or simple two-way interactions. Tests of simple main effects indicated a significant effect of Time on the CS- in the S-US Acquisition group ($F(1,49)=7.22, p=0.010$).

Reinforcement x Reinstatement US x Time: There were no significant two- or three-way interactions, main effects, simple two-way interactions, or simple main effects.

Reinforcement x Match x Time: There were no significant two- or three-way interactions, main effects, simple two-way interactions, or simple main effects.

Reinforcement x Group x Time: There were no significant two- or three-way interactions, main effects, or simple two-way interactions. Tests of simple main effects indicated a significant effect of Time on the CS- in the Social/Physical group ($F(1,47)=5.45, p=0.024$), and a marginally significant effect of higher PPG response to CS+ than CS- in the Social/Physical group at Test trials 1 and 2 ($p=0.065$).

Figure 19.
Study 1 Reinstatement: PPG



Discussion

Three hypotheses were tested in study 1. First, it was hypothesized that both a physically painful muscle stimulation US (P-US) and socially painful negative feedback as a US (S-US) would elicit increased conditional responding to the CS+ relative to the CS- following acquisition trials. Second, it was hypothesized that the presentation of both P-US and S-US during reinstatement would produce subsequently greater conditional responding than was observed following extinction. Third, it was hypothesized that standard reinstatement with the same US as was used during acquisition would produce greater return of conditional responding (i.e. differential increase in CS+ relative to CS-) than cross-US reinstatement with a different US from acquisition. Therefore, it was expected that greater reinstatement would be observed in the Physical/Physical and Social/Social groups than in the Physical/Social and Social/Physical groups. The results are supportive of hypothesis 1, and partially supportive of hypotheses 2 and 3.

Acquisition

As hypothesized, P-US acquisition participants demonstrated significant differential conditioning during acquisition to the CS+ relative to CS- as measured by P-US expectancy ratings, valence ratings, arousal ratings, SCR and PPG. S-US acquisition participants demonstrated significant differential conditioning during acquisition to the CS+ relative to CS- as measured by S-US expectancy ratings. Neither acquisition US group demonstrated acquisition as measured by ASR; in both groups ASR findings appeared most consistent with habituation rather than excitatory conditioning. Thus, both US types elicited significant differential conditioning through acquisition trials, although P-US elicited acquisition across a broader range of measures than S-US.

Reinstatement: Physical and social pain

Before examining the effect of physical and social pain on reinstatement it was necessary to demonstrate that both acquisition and extinction had occurred. For the P-US acquisition group, both acquisition learning and subsequent extinction were demonstrated on P-US expectancy ratings, valence ratings, arousal ratings, and SCR, while acquisition but not extinction were demonstrated on PPG. For the P-US acquisition group, acquisition was not demonstrated for ASR, but there was a significant decrease in both ASR to CS+ and CS- across extinction trials, demonstrating evidence of habituation, and there was no evidence of acquisition or extinction on S-US expectancy. For the S-US acquisition group, both acquisition and subsequent extinction were found on S-US expectancy ratings, but not for any other measures.

P-US reinstatement participants subsequently demonstrated a significant overall increase in responding, but not in differential responding to the CS+ and CS-, as measured by P-US expectancy and ASR. P-US reinstatement participants demonstrated a significant decrease in arousal ratings to CS- following reinstatement but no change for CS+. In the P-US reinstatement group there was no evidence of reinstatement as measured by S-US expectancy, valence ratings, SCR, or PPG. The findings on the effect of P-US on post-reinstatement responding are somewhat mixed, but overall they are most indicative of P-US having a non-differential reinstatement/sensitizing effect, whereby responding to all stimuli subsequently increases, rather than a differential reinstating effect whereby conditional responding increases to the CS+ relative to the CS-.

S-US reinstatement participants demonstrated a significant overall increase in responding following reinstatement trials, but not in differential responding to CS+ and CS-, as measured by S-US expectancy. Similar to the findings of P-US reinstatement, the findings on the effect of S-

US on post-reinstatement responding are most indicative of a non-differential/sensitizing effect, whereby responding to both CS+ and CS- subsequently increases, rather than a differential reinstating effect whereby conditional responding increases to the CS+ relative to the CS-.

Standard versus cross-US reinstatement

Participants who received the same US during reinstatement as during acquisition subsequently demonstrated a significant elevation in S-US expectancy to the CS+, while cross-US participants showed a greater increase in SCR to the CS- following reinstatement. There were no differences between the Same US and Different US groups following reinstatement in P-US expectancy, valence or arousal ratings, EMG, or PPG. While there was only a small amount of evidence suggesting stronger standard reinstatement than cross-US reinstatement, there were notable findings regarding differences between the four combinations of acquisition and reinstatement US (Physical/Physical, Physical/Social, Social/Social, Social/Physical). In the Social/Social group, following reinstatement there was a significant increase in S-US expectancy to the CS+ relative to CS-, providing strong support for the occurrence of differential reinstatement. In the Social/Physical group a significant decrease in PPG to CS- and marginally significant differential between CS+ and CS-following reinstatement provided potential evidence for differential reinstatement. However, given that S-US acquisition participants did not demonstrate acquisition learning on PPG, this finding might best be interpreted as sensitization resulting from the P-US reinstatement. Non-differential reinstatement/sensitization occurring as the result of the P-US would be consistent with findings from P-US expectancy (increased CS+ and CS- responding in both the Social/Physical and Physical/Physical groups), and from ASR (increased CS+ and CS- in the Social/Physical group and increased CS- in the Physical/Physical

group). Finally, the only notable finding in the Physical/Social group was a significant increase in SCR to CS- relative to CS+ following reinstatement.

These findings provide partial support for the hypothesis that standard reinstatement with the same US as during acquisition would produce a differential return of conditional responding, as observed when the S-US was presented in both acquisition and extinction. However, these results did not demonstrate differential reinstatement resulting from P-US presentation. Rather, unpaired P-US presentations following extinction produced subsequent sensitization (non-differential reinstatement) regardless of which US had been presented during acquisition.

STUDY 2

Introduction

Clinical research has demonstrated that level of arousal can act as a retrieval cue for fear, and change in arousal state from the state experienced during fear extinction can produce a return of fear (Mystkowski et al., 2003). Extant findings on the role of arousal on fear reinstatement are mixed. From research on drug-use and drug-relapse there is strong evidence that the experience of a discrete stressor following extinction of drug-seeking behavior will reinstate drug-seeking behavior, a process mediated by elevated neural activity of CRF and noradrenaline. (Stewart, 2000, 2008). This non-specific arousal-based account of reinstatement suggests that any unpaired US that produces a UR consisting of sufficiently high arousal and neuroendocrine response should be adequate to reinstate the CR. Alternatively, a small amount of research also supports a similar-arousal-based theory of fear reinstatement which contends that in order for arousal to reinstate fear it must be qualitatively similar to the arousal experienced during fear acquisition (Hourutunian & Riccio, 1979; Jacobs & Nadel, 1985). No human research to date has examined whether the magnitude of the UR or concordance between acquisition UR and reinstatement UR moderate the subsequent return in conditional responding.

As previously outlined, the limited research on the qualities of unpaired USs necessary to reinstate fear has either examined the relationship between reinstating US and acquisition US or between reinstating UR and acquisition UR, while little consideration has been given to the relationship between the CS and reinstating US. A substantial body of research indicates that the belongingness of CS with US significantly impacts fear learning. URs to qualitatively different aversive USs may differ significantly, and CS-US learning is stronger when the UR is biologically and evolutionarily relevant to the CS (e.g., Garcia & Koelling, 1966; Rescorla,

2008). For example, Garcia and Koelling found that rats readily learned to associate an audiovisual cue with discrete physical pain (shock) but not with nausea (induced by administration of an x-ray or toxin), and readily learned to associate a gustatory cue with nausea but not with discrete physical pain. Faster and stronger acquisition of fear occurs to fear-relevant CSs than to fear irrelevant CSs in humans and monkeys (Mineka & Öhman, 2002; Öhman & Mineka, 2001). For example, a series of experiments found that lab-reared monkeys naïve to the study stimuli learned fear through vicarious conditioning to fear-relevant stimuli (i.e. snakes, crocodiles) but not to fear-irrelevant stimuli (i.e. flowers, rabbits) (Cook & Mineka, 1989, 1990). However, there is little existing evidence regarding the role of CS belongingness in reinstatement, either between CS and acquisition US or CS and reinstating US.

The aims of Study 2 were to replicate findings from Study 1, evaluate the effect of CS belongingness on standard and cross-US reinstatement, and combined with Study 1 data examine physiological arousal experienced during reinstatement as a predictor of return of differential conditional responding. Study 2 compared S-US and P-US reinstatement for participants who had completed S-US acquisition with a socially relevant CS+. Indices of conditional responding through all experimental phases were the same as in Study 1: self-report measures (P-US expectancy, S-US expectancy, valence and arousal ratings) and physiological measures (ASR, SCR, PPG). Several hypotheses were tested in Study 2. First, Study 2 aimed to replicate findings from Study 1, so it was hypothesized that Social/Social participants would show differential reinstatement, while Social/Physical participants would show non-differential reinstatement/sensitization. Second, it was predicted that due to the inclusion of the socially relevant CS+, Study 2 Social/Social and Social/Physical participants would show stronger differential reinstatement and non-differential reinstatement/sensitization, respectively, than

Study 1 participants in the corresponding groups. Third, it was hypothesized that across both Study 1 and Study 2 greater physiological arousal experienced during the reinstatement period would predict greater return of conditional responding during post-reinstatement test trials.

Methods

Overview of design

Experimental procedures in Study 2 were identical to those in Study 1, except that Study 2 utilized different stimuli as the CS, and all participants completed S-US rather than P-US acquisition. Participants were randomly assigned to one of two experimental groups based on Reinstatement US (Physical or Social). The distribution of participants across the groups was as follows: P-US Reinstatement=13, S-US Reinstatement=10.

Participants

Twenty-three participants (17 females, 6 males), with a mean age of 22.22 ($SD=3.29$) were recruited from undergraduate introductory psychology courses and through posting recruitment flyers on a university campus. Study participants received either course credit or \$30 for 2 hours of study participation. The ethnic distribution of the sample was 8.7% Caucasian, 65.2% Asian, 13.0% Hispanic, 8.7% African American, and 4.3% who classified themselves as multiethnic or other. Participants were recruited if they met the following eligibility criteria: a) age 18 years or older; b) English speaking; c) score 6 or higher on the Mini Social Phobia Inventory (Mini-SPIN). Exclusion criteria for study participation included: a) physician recommended to stay away from stressful situations; b) serious respiratory, cardiovascular, pulmonary, or neurological condition, c) hearing impaired, d) pregnant.

Measures

All measures in Study 2 were identical to those used in Study 1.

Stimuli

Conditional Stimuli: In Study 2, a cartoon picture of a yellow-colored neutral face (yellow circle, with two black dots and a vertical black line) served as CS+. A blue square with two black dots and a diagonal black line (included in order to match the face CS for complexity of visual components) served as the CS-. Pictures of conditional stimuli are included in the Appendix 1.

Unconditional stimuli: Study 2 used the same two US as in Study 1: P-US (simultaneous presentation of a picture of a spark and muscle stimulation), and S-US (simultaneous presentation of a picture of an angry face and a personally relevant auditory insult).

Procedures

Study 2 procedures were virtually identical to Study 1 procedures, other than a few minor differences. Both groups in Study 2 completed S-US acquisition, and subsequently either S-US or P-US reinstatement, so Study 2 was comprised of a Social/Social (S-US Reinstatement) and Social/Physical (P-US Reinstatement) group. Study 2 used similar, but slightly more complex CSs than Study 1. Study 2 used a socially relevant CS+ (yellow circle with two black dots and a vertical black line), while the CS- was a blue square with two black dots and a diagonal black line. All other procedures during Study 2 were identical to Study 1.

Table 2.
Study 2 Conditioning Procedures Flow Chart

Group	Habituation	Acquisition	Extinction	Reinstatement	Test Phase
S-US Reinstatement	Socially relevant CS+ (3) CS- (3)	CS+ / S-US (6) CS- (6) Neutral feedback (2)	CS+ (6) CS- (6)	S-US (4)	CS+ (2) CS- (2)
P-US Reinstatement	Socially relevant CS+ (3) CS- (3)	CS+ / S-US (6) CS- (6) Neutral feedback (2)	CS+ (6) CS- (6)	P-US (4)	CS+ (2) CS- (2)

Results

For results of Acquisition, Extinction, and Reinstatement only significant findings are reported. Means, and statistical values for all non-significant findings are reported in Appendix 3.

Baseline measures

Mean scores on baseline measures were as follows: Mini-SPIN: $M=8.78$, $SD=1.83$; BDI: $M=6.43$, $SD=5.75$; ASI-X: $M=67.91$, $SD=18.31$; MAAS: $M=57.00$, $SD=14.02$; STAI: $M=46.09$, $SD=11.02$. Independent samples t-tests revealed no significant differences between the two groups on Mini-SPIN ($t(21)=1.11$, $p=0.278$), BDI ($t(21)=1.88$, $p=0.074$), ASI-X ($t(21)=0.454$, $p=0.655$), MAAS ($t(21)=0.561$, $p=0.581$), or STAI ($t(77)=1.71$, $p=0.103$). There were also no significant differences among the groups on age ($t(21)=0.196$, $p=0.847$), gender ($\chi^2(1, N=23)=2.28$, $p=0.123$), or ethnicity ($\chi^2(4, N=23)=7.81$, $p=0.099$).

Stimulus ratings

Data from Study 1 and Study 2 were combined for analyses of stimulus ratings. Independent samples t-tests revealed significantly higher P-US distress ($t(77)=1.69$, $p=0.005$) in Study 1 ($M=3.75$, $SD=1.83$) than Study 2 ($M=2.91$, $SD=2.35$). Independent samples t-tests revealed no significant differences between Study 1 and Study 2 on self-selected muscle stimulation level ($t(76)=0.014$, $p=0.853$), S-US distress ($t(77)=0.343$, $p=0.528$), S-US believability ($t(77)=0.992$, $p=0.717$), or acoustic startle probe distress ($t(71)=0.233$, $p=0.913$).

Independent samples t-tests revealed significantly higher P-US distress ($t(77)=2.95$, $p=0.001$) in the P-US Acquisition group ($M=4.34$, $SD=1.56$) than S-US Acquisition group ($M=3.02$, $SD=2.12$), but revealed no significant differences between groups on self-selected

muscle stimulation level ($t(76)=0.780, p=0.524$), S-US distress ($t(77)=1.82, p=0.433$), S-US believability ($t(77)=1.18, p=0.284$), or acoustic startle probe distress ($t(71)=0.132, p=0.715$).

Paired samples t-tests indicated significantly higher startle probe distress than P-US distress ($t(72)=3.17, p=0.000$), and significantly higher startle probe distress than S-US distress ($t(72)=4.98, p=0.000$), but no significant difference between P-US and S-US distress ($t(78)=1.17, p=0.247$). Across both studies, average stimulus ratings were: muscle stimulation level: $M=39.95, SD=16.62$; P-US distress: $M=3.51, SD=2.02$; S-US distress: $M=3.17, SD=2.10$; S-US believability: $M=2.35, SD=1.54$; startle probe distress: $M=4.82, SD=1.84$.

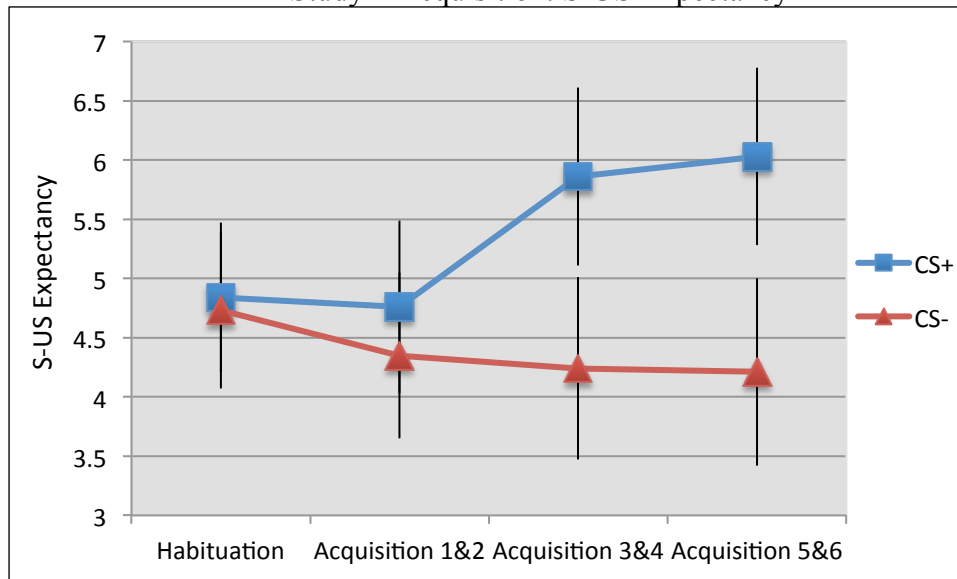
Acquisition

Values of P-US expectancy S-US expectancy, SCR, ASR, and PPG were averaged across the 3 habituation trials, across acquisition trials 1 and 2, across acquisition trials 3 and 4, and across acquisition trials 5 and 6. 2 (*Reinforcement*; CS+, CS-) x 4 (*Time*; Habituation average, Acquisition trials 1 and 2, Acquisition trials 3 and 4, Acquisition trials 5 and 6) mixed-design ANOVAs were conducted for P-US expectancy S-US expectancy, SCR, ASR, and PPG. 2 (*Reinforcement*; CS+, CS-) x 2 (*Time*; Post-Habituation, Post-Acquisition) mixed-design ANOVAs were conducted for Valence ratings and Arousal ratings.

P-US Expectancy: There were no significant two-way interactions, main effects, or simple main effects.

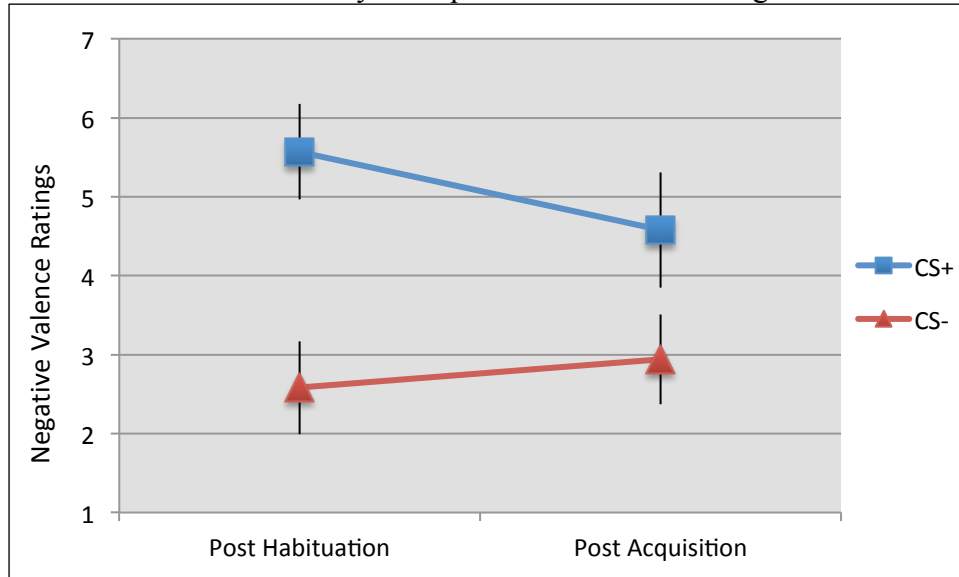
S-US Expectancy: There was a significant two-way Reinforcement x Time interaction ($F(3,17)=7.26, p=0.038, \eta^2=0.04$), and a significant main effect of Reinforcement ($F(1,19)=5.57, p=0.029, \eta^2=0.07$). Tests of simple main effects indicated significantly higher CS+ than CS- at Acquisition trials 3 and 4 ($F(1,19)=5.77, p=0.027$) and Acquisition trials 5 and 6 ($F(1,19)=4.44, p=0.049$).

Figure 20.
Study 2 Acquisition: S-US Expectancy



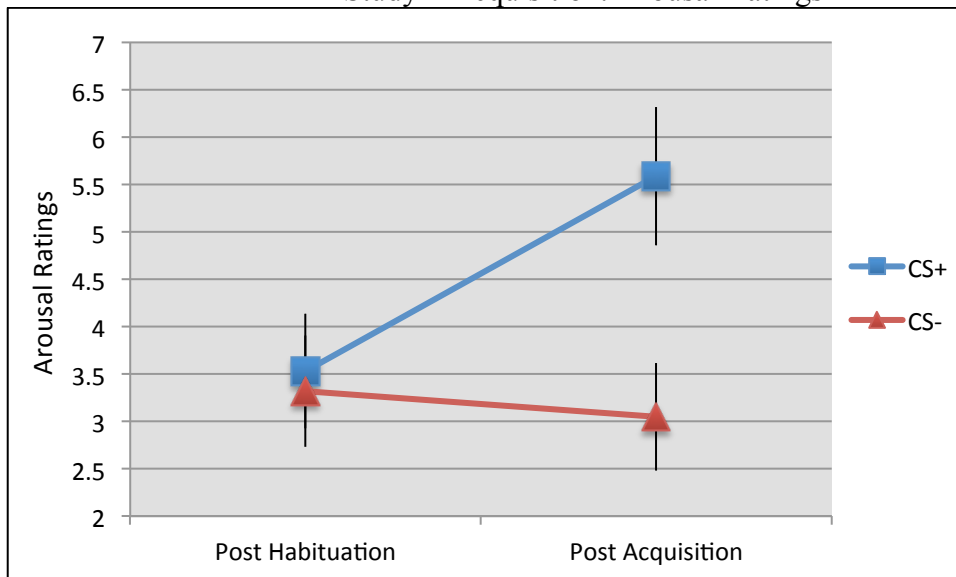
Valence Ratings: There was no significant two-way interaction, but there was a significant main effect of Reinforcement ($F(1,19)=16.15, p=0.001, \eta^2=0.29$). Tests of simple main effects indicated significantly higher CS+ than CS- at Post-Habituation ($F(1,19)=13.62, p=0.002$) and Post-Acquisition ($F(1,19)=8.40, p=0.010$).

Figure 21.
Study 2 Acquisition: Valence Ratings



Arousal Ratings: There was a significant two-way Reinforcement x Time interaction ($F(1,19)=13.28, p=0.002, \eta^2=0.09$), and a significant main effect of Reinforcement ($F(1,19)=6.93, p=0.017, \eta^2=0.13$). Tests of simple main effects indicated significantly higher CS+ than CS- at Post-Acquisition ($F(1,19)=11.57, p=0.003$), and found a significant effect of Time on CS+ ($F(1,19)=8.95, p=0.008$).

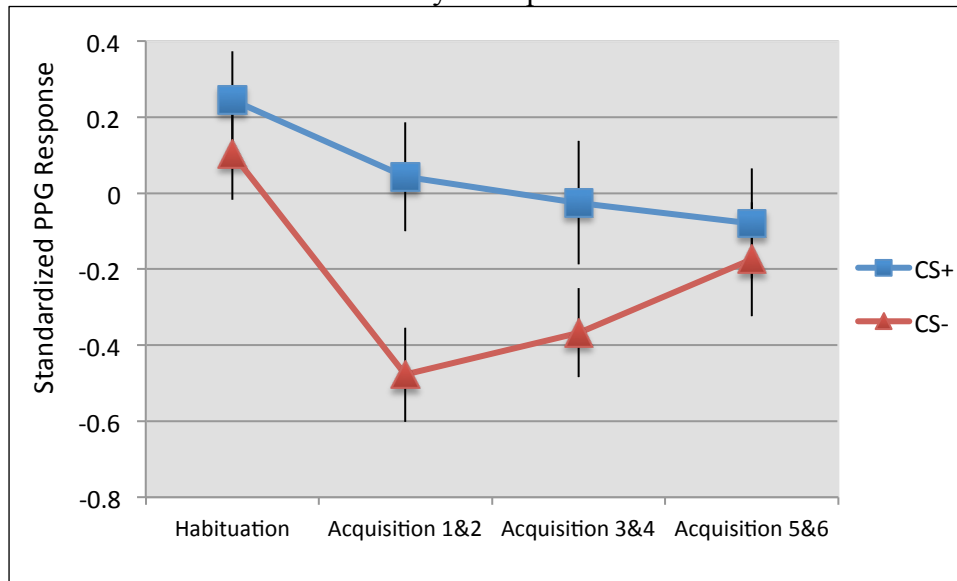
Figure 22.
Study 2 Acquisition: Arousal Ratings



SCR: There was no significant two-way interaction, main effects, or simple main effects.

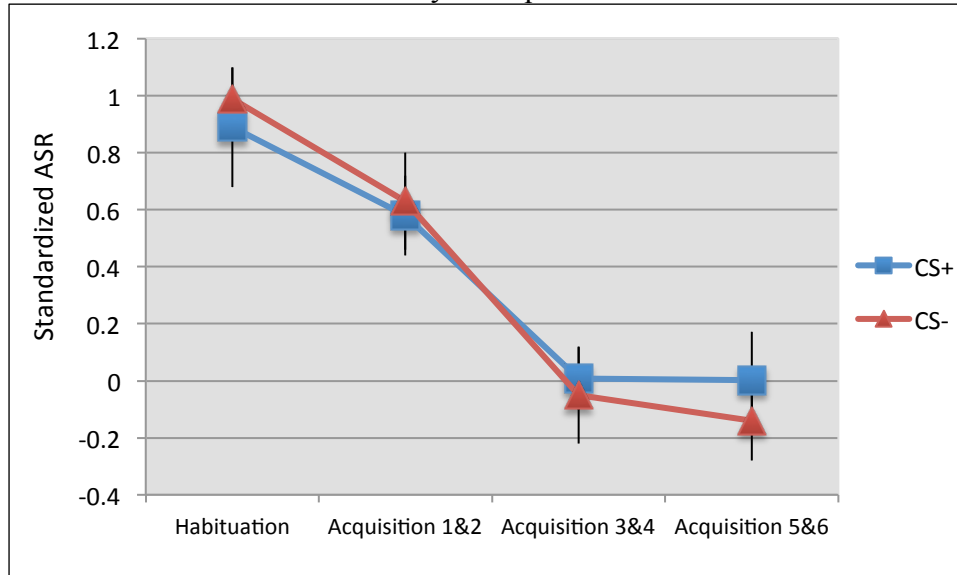
PPG: There was no significant two-way interaction, but there were significant main effects of Reinforcement ($F(1,19)=5.28, p=0.034, \eta^2=0.06$), and Time ($F(1,19)=3.84, p=0.015, \eta^2=0.07$). Tests of simple main effects indicated significantly higher CS+ than CS- at Acquisition trials 1 and 2 ($F(1,19)=5.33, p=0.033$), and found a significant effect of Time on CS- ($F(1,19)=4.58, p=0.017$).

Figure 23.
Study 2 Acquisition: PPG



ASR: There was no significant two-way interaction, but there was a significant main effect of Time ($F(3,18)=6.64, p=0.006$). Tests of simple main effects indicated a significant effect of Time on CS+ ($F(3,18)=6.64, p=0.006$) and CS- ($F(3,18)=15.30, p=0.000$).

Figure 24.
Study 1 Acquisition: ASR

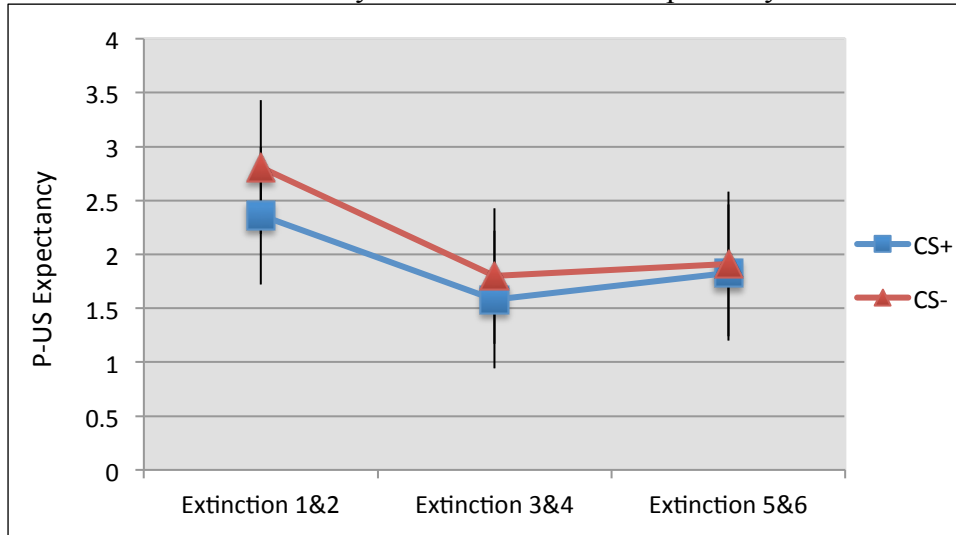


Extinction

2 (Reinforcement; CS+, CS-) x 3 (Time; Extinction trials 1 and 2, Extinction trials 3 and 4, Extinction trials 5 and 6) mixed-design ANOVAs were conducted for P-US expectancy S-US expectancy, SCR, ASR, and PPG. 2 (Reinforcement; CS+, CS-) x 2 (Time; Post-Acquisition, Post-Extinction) mixed-design ANOVAs were conducted for Valence ratings and Arousal ratings.

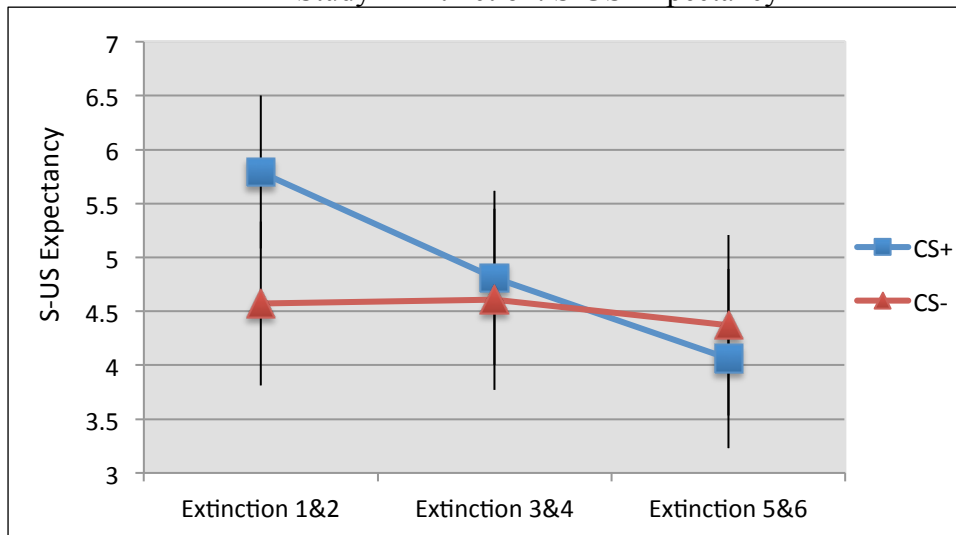
P-US Expectancy: There was no significant two-way interaction, but there was a significant main effect of Time ($F(1,20)=3.87, p=0.011, \eta^2=0.19$). Tests of simple main effects indicated significantly higher CS- than CS+ at Extinction trials 1 and 2 ($F(1,19)=5.02, p=0.040$), and found a significant effect of Time on CS- ($F(1,19)=4.32, p=0.033$).

Figure 25.
Study 2 Extinction: P-US Expectancy



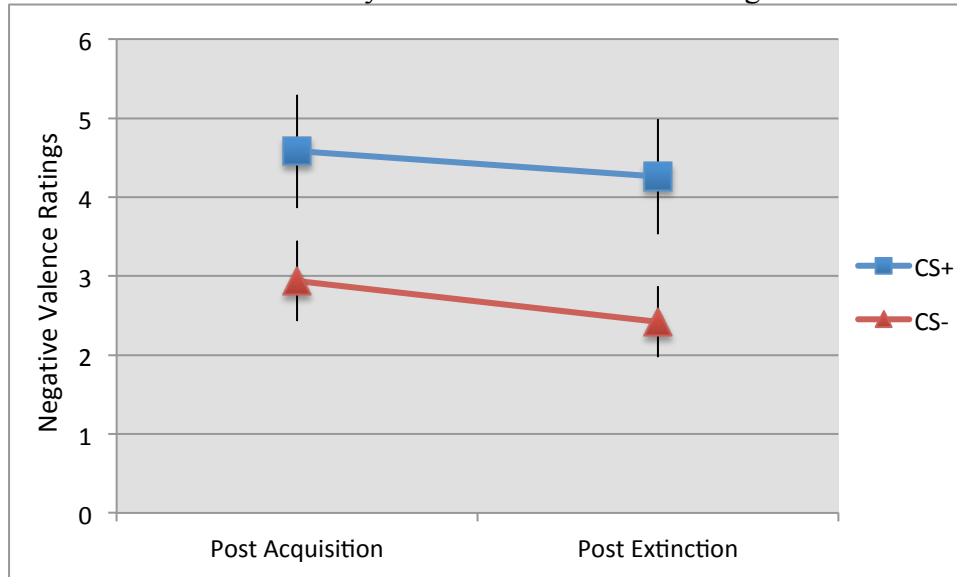
S-US Expectancy: There was a significant two-way Reinforcement x Time interaction ($F(3,18)=4.5, p=0.017, \eta^2=0.06$), and no significant main effects. Tests of simple main effects indicated significantly higher CS+ than CS- at Extinction trials 1 and 2 ($F(1,19)=4.27, p=0.050$) and a significant effect of Time on CS+ ($F(2,18)=3.91, p=0.039$).

Figure 26.
Study 2 Extinction: S-US Expectancy



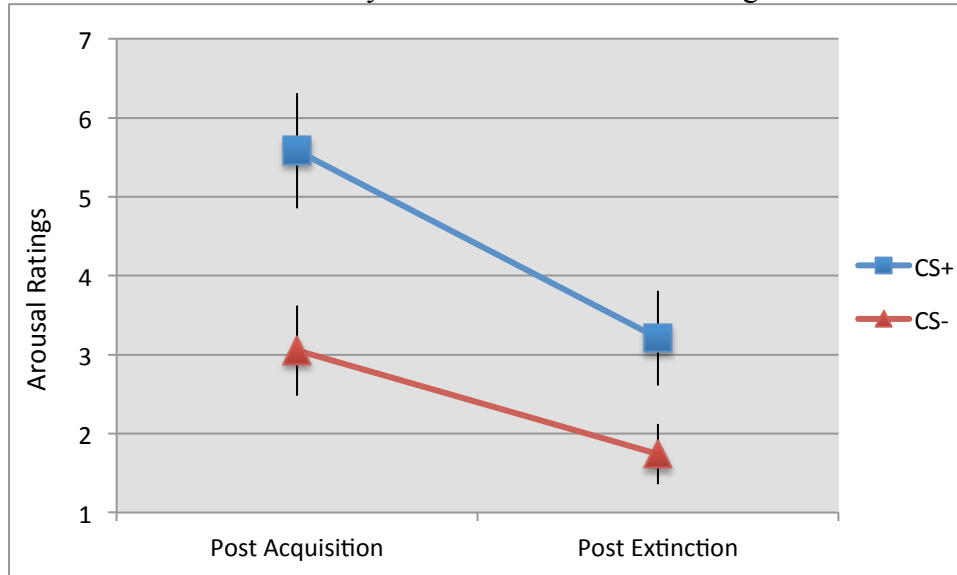
Valence Ratings: There was no significant two-way interaction, but there was a main effect for Reinforcement ($F(1,18)=8.96, p=0.008, \eta^2=0.26$). Tests of simple main effects indicated significantly higher CS+ than CS- at Post-Acquisition ($F(1,19)=8.39, p=0.050$) and Post-Extinction ($F(1,19)=6.96, p=0.010$).

Figure 27.
Study 2 Extinction: Valence Ratings



Arousal Ratings: There was no significant two-way interaction, but there were main effects of Reinforcement ($F(1,18)=14.87, p=0.001, \eta^2=0.23$) and Time ($F(1,18)=26.66, p=0.000, \eta^2=0.20$). Tests of simple main effects indicated significantly higher CS+ than CS- at Post-Acquisition ($F(1,19)=11.56, p=0.003$) and Post-Extinction ($F(1,19)=10.21, p=0.005$). Tests of simple main effects also found a significant effect of time on CS+ ($F(1,19)=16.48, p=0.001$) and CS- ($F(1,19)=12.31, p=0.003$).

Figure 28.
Study 2 Extinction: Arousal Ratings



PPG: There was no significant two-way interaction, main effects, or simple main effects.

SCR: There was no significant two-way interaction, main effects, or simple main effects.

ASR: There were no significant two-way interaction, main effects, or simple main effects.

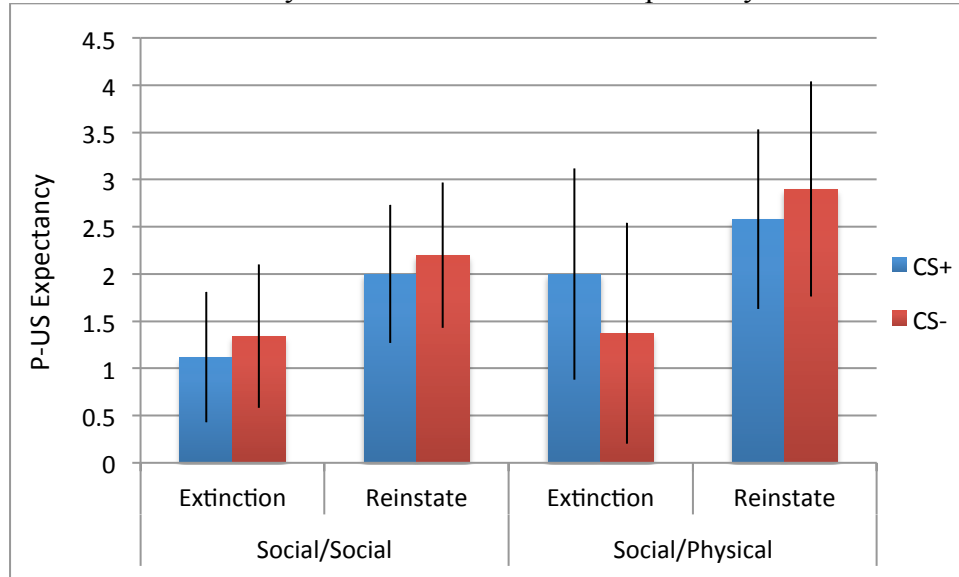
Reinstatement

Values of P-US expectancy, S-US expectancy, SCR, ASR, and PPG were averaged across extinction trials 5 and 6, and post-reinstatement test trials 1 and 2. 2 (*Reinforcement*; CS+, CS-) x 2 (*Reinstatement US*; Physical, Social) x 2 (*Time*; Extinction trials 5 and 6, Test trials 1 and 2) mixed-design ANOVAs were conducted for P-US expectancy, S-US expectancy, SCR, ASR, and PPG. 2 (*Reinforcement*; CS+, CS-) x 2 (*Reinstatement US*; Physical, Social) x 2 (*Time*; Post-Extinction, Post-Reinstatement) mixed-design ANOVAs were conducted for Valence ratings and Arousal ratings.

P-US Expectancy: There were no significant two- or three-way interactions or main effects, although there was a marginally significant main effect of Time ($p=0.056$). Tests of

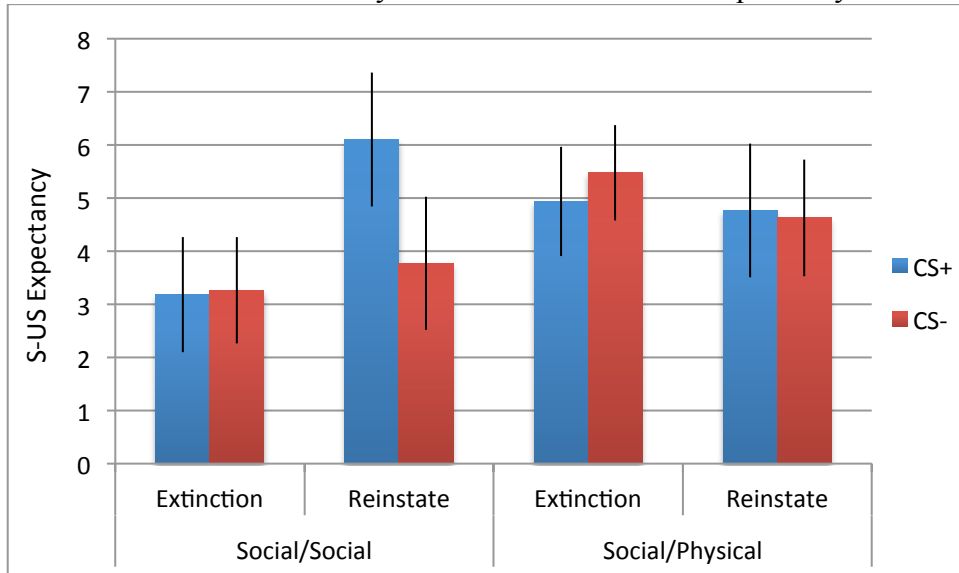
simple main effects indicated a significant effect of Time on CS- in the P-US Reinstatement group ($F(1,18)=5.42, p=0.032$).

Figure 29.
Study 2 Reinstatement: P-US Expectancy



S-US Expectancy: There was no significant three-way interaction (marginally significant, $p=0.084$), but there were significant two-way Reinforcement x Time ($F(1,18)=10.65, p=0.004, \eta^2=0.06$), Reinstatement US x Time ($F(1,18)=4.93, p=0.040, \eta^2=0.12$), and Reinforcement x Reinstatement US ($F(3,18)=4.85, p=0.041, \eta^2=0.04$) interactions. Tests of simple two-way interactions found significant Reinforcement x Time interactions in the S-US Reinstatement US group ($F(1,10)=9.26, p=0.014, \eta^2=0.08$), significant Reinforcement x Reinstatement US interactions at test trials 1 and 2 ($F(1,18)=4.83, p=0.041, \eta^2=0.17$), and Reinstatement US x Time interactions for CS+ ($F(1,18)=5.88, p=0.026, \eta^2=0.21$). Tests of simple main effects indicated significantly higher CS+ than CS- in the S-US Reinstatement group at test trials 1 and 2 ($F(1,18)=10.93, p=0.004$), and found a significant effect of Time on CS+ in the S-US Reinstatement group ($F(1,18)=10.47, p=0.005$).

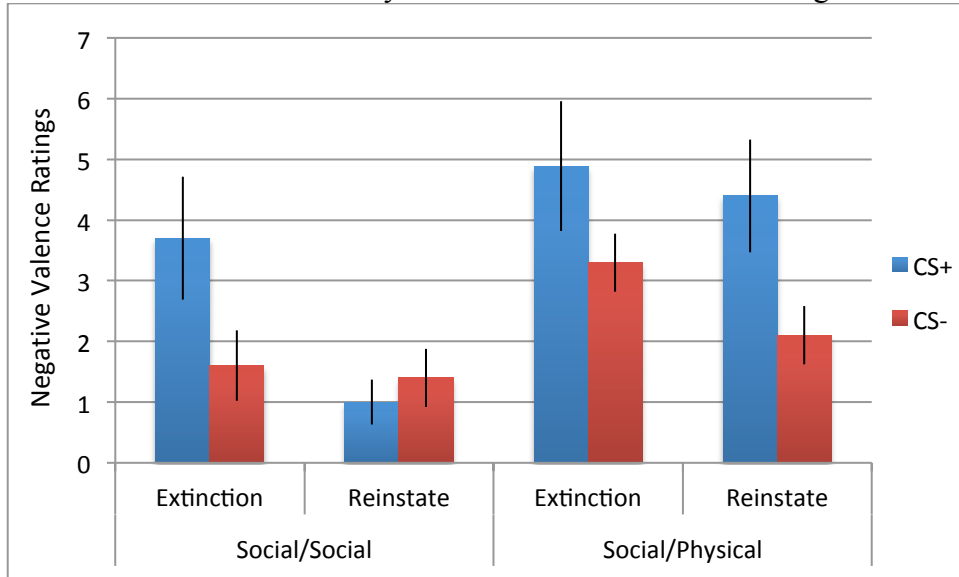
Figure 30.
Study 2 Reinstatement: S-US Expectancy



Valence Ratings: There was a significant three-way Reinforcement x Reinstatement US x Time interaction ($F(1,17)=4.28, p=0.050, \eta^2=0.05$), but no significant two-way interactions. Tests of simple two-way interactions indicated a significant Reinforcement x Time interaction in the S-US Reinstatement group ($F(1,19)=5.60, p=0.042, \eta^2=0.12$), and a significant Reinforcement by Reinstatement US interaction at Post-Reinstatement ($F(1,19)=11.06, p=0.004, \eta^2=0.23$). There were significant main effects of Reinforcement ($F(1,17)=10.61, p=0.005, \eta^2=0.16$), Reinstatement US ($F(1,17)=4.95, p=0.040, \eta^2=0.07$), and Time ($F(1,17)=9.71, p=0.006, \eta^2=0.10$). Tests of simple main effects indicated significantly higher CS- at Post-Extinction in the P-US Reinstatement than the S-US Reinstatement group ($F(1,18)=4.45, p=0.050$), significantly higher CS+ at Post-Reinstatement in the P-US Reinstatement group ($F(1,18)=12.87, p=0.002$), significantly higher CS+ than CS- in the S-US Reinstatement group at Post-Extinction ($F(1,18)=4.54, p=0.048$), and significantly higher CS+ than CS- in the P-US Reinstatement group at Post-Reinstatement ($F(1,18)=15.31, p=0.001$). Tests of simple main

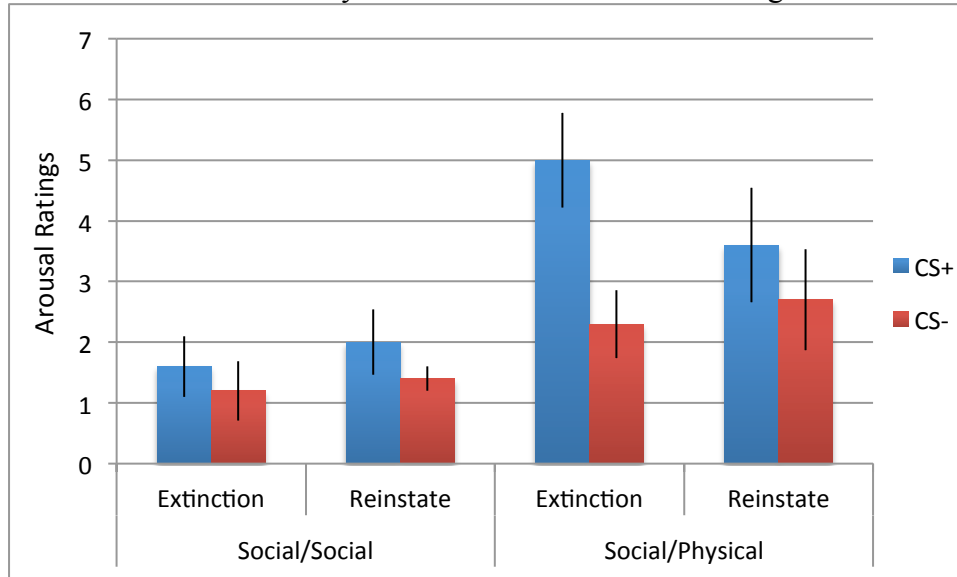
effects also found a significant effect of Time on CS+ in the S-US Reinstatement group ($F(1,18)=7.93, p=0.012$) and CS- in the P-US Reinstatement group ($F(1,18)=7.33, p=0.015$).

Figure 31.
Study 2 Reinstatement: Valence Ratings



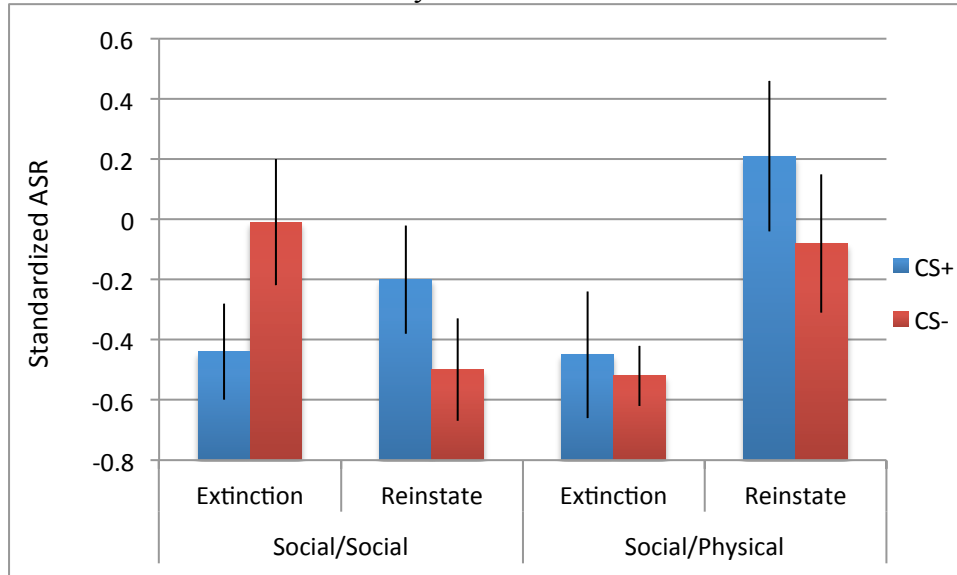
Arousal Ratings: There were no significant two- or three-way interactions, but there were significant main effects of Reinforcement ($F(1,18)=8.35, p=0.010, \eta^2=0.16$) and Reinstatement US ($F(1,18)=6.54, p=0.020, \eta^2=0.09$). Tests of simple two-way interactions indicated a significant Reinforcement x Reinstatement US interaction at Extinction trials 5 and 6 ($F(1,19)=8.55, p=0.009, \eta^2=0.20$). Tests of simple main effects indicated significantly higher CS+ than CS- at Extinction trials 5 and 6 in the P-US Reinstatement group ($F(1,18)=14.02, p=0.002$), and significantly higher CS+ than CS- in the P-US Reinstatement group at Post-Extinction ($F(1,18)=22.48, p=0.000$).

Figure 32.
Study 2 Reinstatement: Arousal Ratings



ASR: There were no significant three-way interactions, but there was a significant two-way Reinforcement x Time interaction ($F(1,15)=84.68, p=0.030, \eta^2=0.05$). Tests of simple two-way interactions indicated a significant Reinforcement x Time interaction in the S-US Reinstatement group ($F(1,9)=5.78, p=0.040, \eta^2=0.13$), and a significant Reinstatement US x Time interaction for the CS- ($F(1,15)=5.78, p=0.033, \eta^2=0.27$). Tests of simple main effects indicated a significant effect of Time on CS+ in the P-US group ($F(1,15)=4.83, p=0.044$).

Figure 33.
Study 2 Reinstatement: ASR



SCR: There were no significant two- or three-way interactions, main effects, or simple main effects.

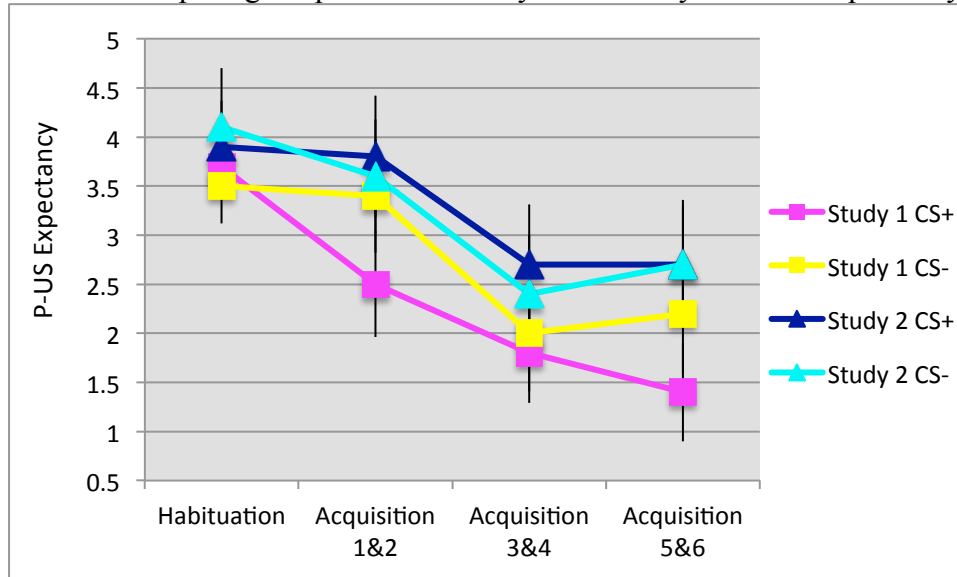
PPG: There were no significant two- or three-way interactions, main effects, or simple main effects.

Belongingness: Comparing Study 1 and Study 2

Acquisition

P-US Expectancy: There were no significant two- or three-way interactions, main effects or simple two-way interactions involving study. Tests of simple main effects indicated significantly higher CS- than CS+ in Study 1 at Acquisition trials 1 and 2 ($F(1,40)=4.14$, $p=0.049$) and Acquisition trials 5 and 6 ($F(1,40)=4.53$, $p=0.040$), but not at any time point in Study 2.

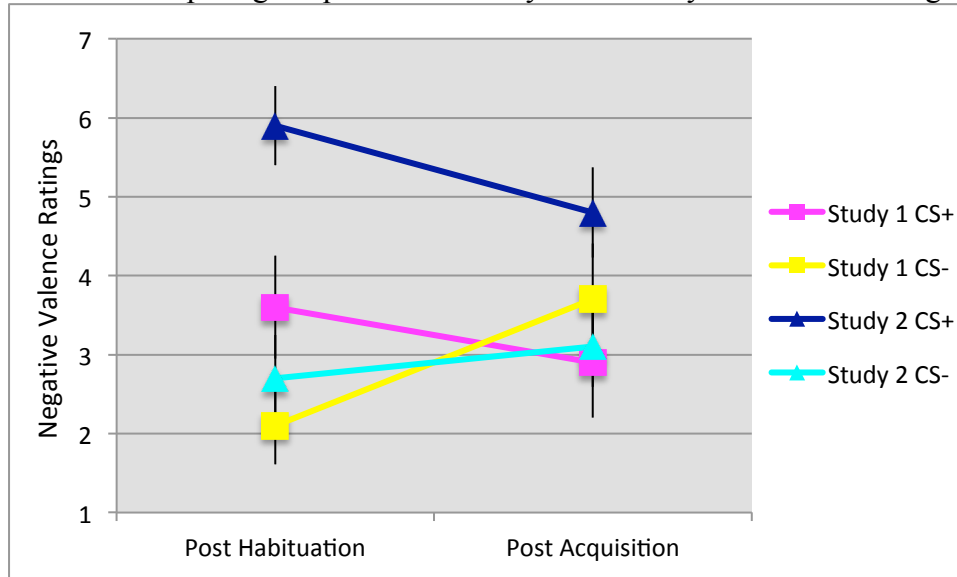
Figure 34.
Comparing Acquisition in Study 1 and Study 2: P-US Expectancy



S-US Expectancy: There were no significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

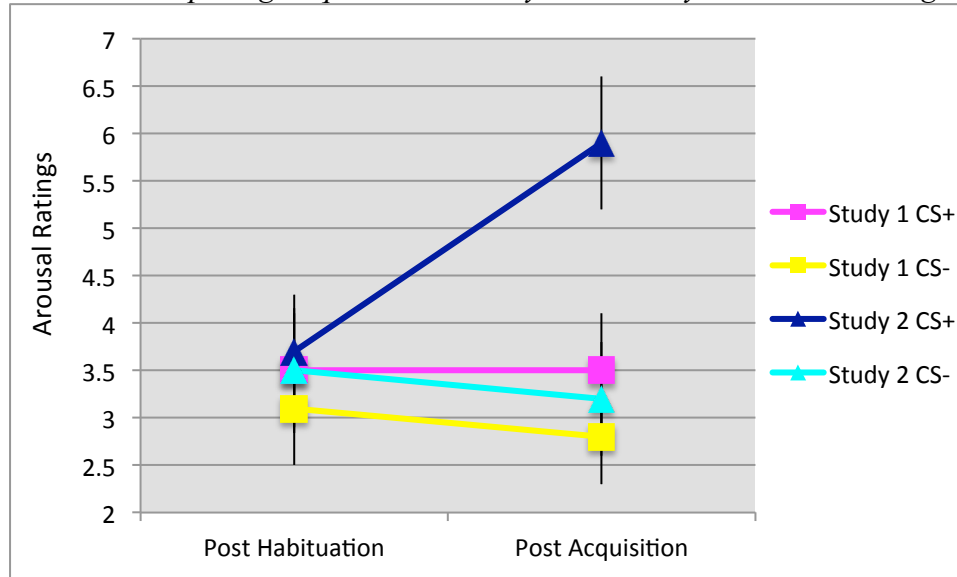
Valence Ratings: There was no significant three-way interaction, but there was a significant two-way Reinforcement x Study interaction ($F(1,38)=8.94, p=0.005, \eta^2=0.08$), and a marginally significant Time x Study interaction ($p=0.053$). Tests of simple two-way interactions indicated a significant Reinforcement x Time interaction in Study 1 ($F(1,21)=8.40, p=0.009, \eta^2=0.14$). Tests of simple main effects indicated significantly higher CS+ in Study 2 than Study 1 at Post-Habitation ($F(1,38)=5.01, p=0.031$) and Post-Acquisition ($F(1,38)=5.12, p=0.030$), significantly higher CS+ than CS- at Post-Habitation in Study 1 ($F(1,38)=4.76, p=0.035$) and in Study 2 ($F(1,38)=16.33, p=0.000$), and at Post-Acquisition in Study 2 ($F(1,38)=9.30, p=0.004$). Tests of simple main effects indicated significant effect of Time on CS- in Study 1 ($F(1,38)=13.06, p=0.001$).

Figure 35.
Comparing Acquisition in Study 1 and Study 2: Valence Ratings



Arousal Ratings: There was a significant three-way Reinforcement x Time x Study interaction ($F(1,40)=7.35, p=0.010, \eta^2=0.02$), but no significant two-way interactions. Tests of simple two-way interactions indicated a significant Reinforcement x Time interaction in Study 2 ($F(1,19)=13.28, p=0.002, \eta^2=0.09$). Tests of simple main effects indicated significantly higher CS+ at Post-Acquisition in Study 2 than in Study 1 ($F(1,40)=6.71, p=0.013$), significantly higher CS+ than CS- in Study 2 at Post-Acquisition ($F(1,40)=19.74, p=0.000$), and a significant effect of Time on CS+ in Study 2 ($F(1,40)=11.64, p=0.001$).

Figure 36.
Comparing Acquisition in Study 1 and Study 2: Arousal Ratings

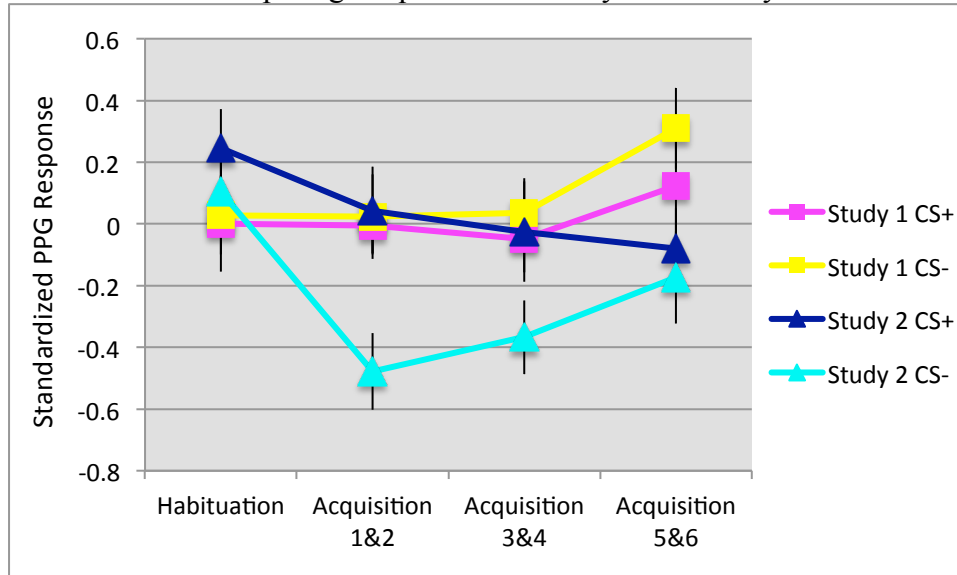


SCR: There were no significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

ASR: There were no significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

PPG: There were no significant two- or three-way interactions, although there were marginally significant Reinforcement x Study ($p=0.067$) and Time x Study ($p=0.065$) interactions. There was a main effect of Study ($F(1,40)=4.39, p=0.043, \eta^2=0.11$). Tests of simple main effects indicated significantly higher CS- in Study 1 than Study 2 at Acquisition trials 1 and 2 ($F(1,40)=7.03, p=0.011$) and at Acquisition trials 3 and 4 ($F(1,40)=6.09, p=0.018$), and significantly higher CS+ than CS- in Study 2 at Acquisition trials 1 and 2 ($F(1,40)=7.50, p=0.009$). Tests of simple main effects found a significant effect of Time on CS- in Study 2 ($F(3,38)=5.26, p=0.004$).

Figure 37.
Comparing Acquisition in Study 1 and Study 2: PPG



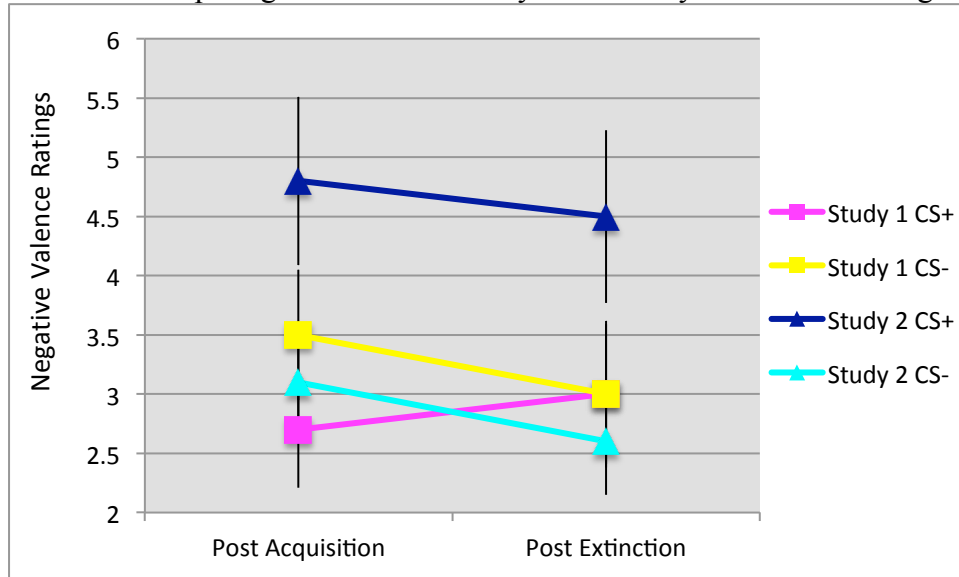
Extinction

P-US Expectancy: There were no significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

S-US Expectancy: There were no significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

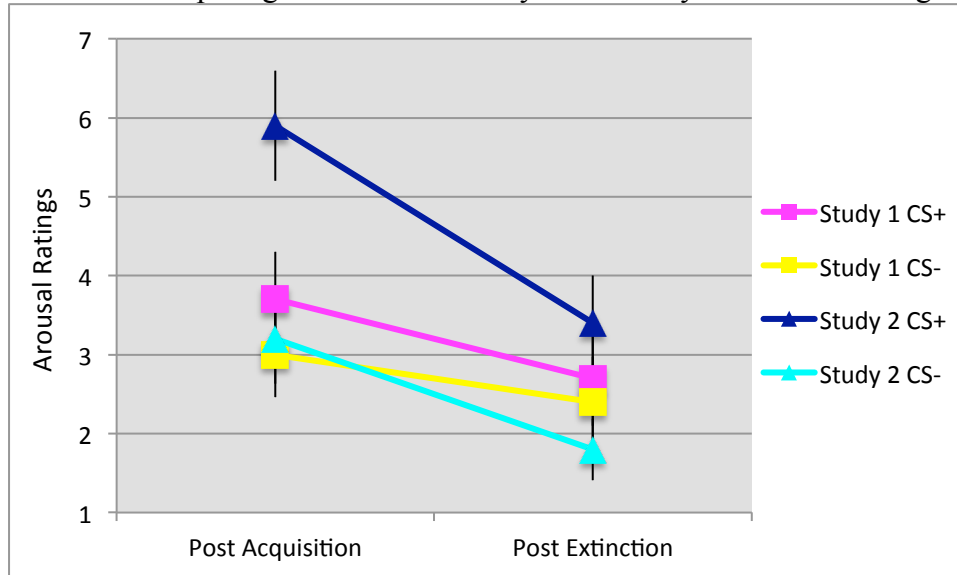
Valence Ratings: There was no significant three-way interaction, but there was a significant two-way Reinforcement x Study interaction ($F(1,40)=10.68, p=0.002, \eta^2=0.05$). There were no significant simple two-way interactions, but tests of simple main effects indicated significantly higher CS+ in Study 2 than Study 1 at Post-Extinction ($F(1,40)=9.73, p=0.013$), and significantly higher CS+ than CS- at Post-Acquisition ($F(1,40)=6.70, p=0.003$) and Post-Extinction ($F(1,40)=8.81, p=0.005$).

Figure 38.
Comparing Extinction in Study 1 and Study 2: Valence Ratings



Arousal Ratings: There was no significant three-way interaction, but there were significant two-way Reinforcement x Study ($F(1,39)=6.80, p=0.013, \eta^2=0.06$) and significant two-way Time x Study ($F(1,39)=7.80, p=0.008, \eta^2=0.03$) interactions. There were no significant simple two-way interactions, but tests of simple main effects indicated significantly higher CS+ in Study 2 than Study 1 at Post-Acquisition ($F(1,39)=5.85, p=0.020$), and significantly higher CS+ than CS- at Post-Acquisition ($F(1,39)=19.28, p=0.000$) and Post-Extinction ($F(1,39)=9.71, p=0.003$). Tests of simple main effects also indicated a significant effect of Time on CS+ in Study 1 ($F(1,39)=5.15, p=0.029$) and Study 2 ($F(1,39)=27.51, p=0.000$), and CS- in Study 2 ($F(1,39)=12.09, p=0.001$).

Figure 39.
Comparing Extinction in Study 1 and Study 2: Arousal Ratings



SCR: There were no significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

ASR: There were no significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

PPG: There were no significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

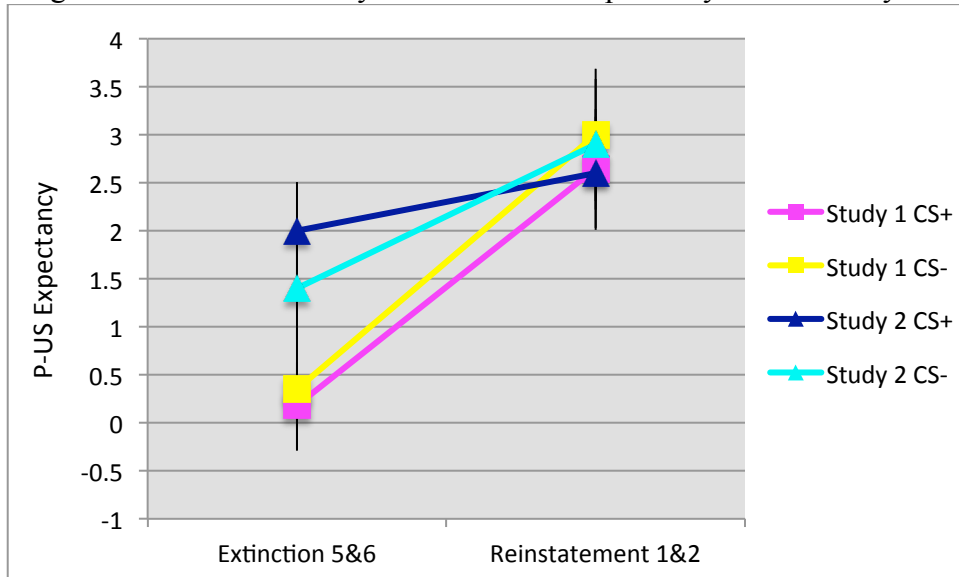
Reinstatement

P-US Expectancy

Social/Physical Reinstatement: There were no significant two- or three-way interactions, main effects, or simple two-way effects. Tests of simple main effects indicated significantly higher CS+ in Study 2 than Study 1 at Extinction trials 5 and 6 ($F(1,19)=5.98, p=0.024$), higher CS+ than CS- in Study 2 at Extinction trials 5 and 6 ($F(1,19)=4.62, p=0.044$), and found a significant effect of Time on CS+ ($F(1,19)=6.70, p=0.016$) and CS- ($F(1,19)=8.47, p=0.099$) in Study 1.

Figure 40.

Comparing Reinstatement in Study 1 and 2: P-US Expectancy in Social/Physical Reinstatement



Social/Social Reinstatement: There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

S-US Expectancy

Social/Physical Reinstatement: There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

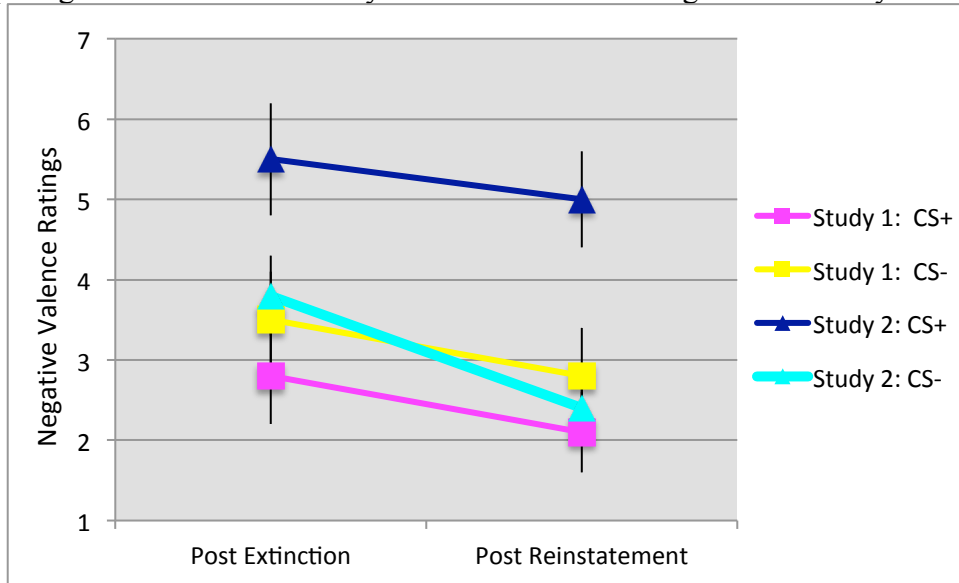
Social/Social Reinstatement: There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

Valence Ratings

Social/Physical Reinstatement: There were no significant three-way interactions, but there was a significant Reinforcement x Study interaction ($F(1,18)=12.45, p=0.002, \eta^2=0.19$). There were no simple two-way interactions, but tests of simple main effects indicated

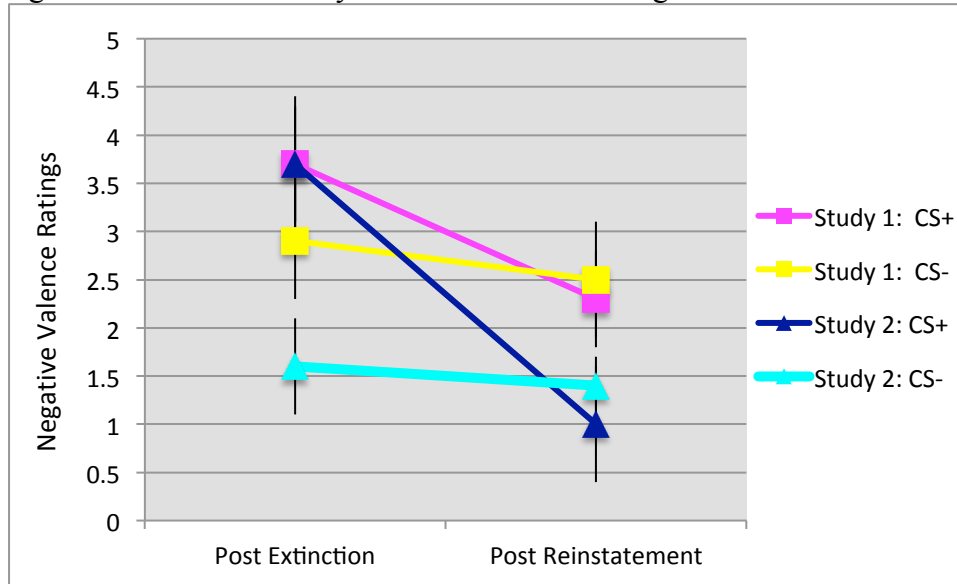
significantly higher CS+ in Study 2 than Study 1 at Post-Reinstatement ($F(1,18)=6.29, p=0.022$), and a significant effect of Time on CS- in Study 2, ($F(1,18)=7.45, p=0.014$).

Figure 41.
Comparing Reinstatement in Study 1 and 2: Valence Ratings in Social/Physical Reinstatement



Social/Social Reinstatement: There were no significant two- or three-way interactions, main effects, or simple two-way interactions involving Study. Tests of simple main effects indicated significantly higher CS+ than CS- at Post-Extinction in Study 2 ($F(1,18)=4.83, p=0.041$), and a significant effect of Time on CS+ in Study 2 ($F(1,18)=9.08, p=0.007$).

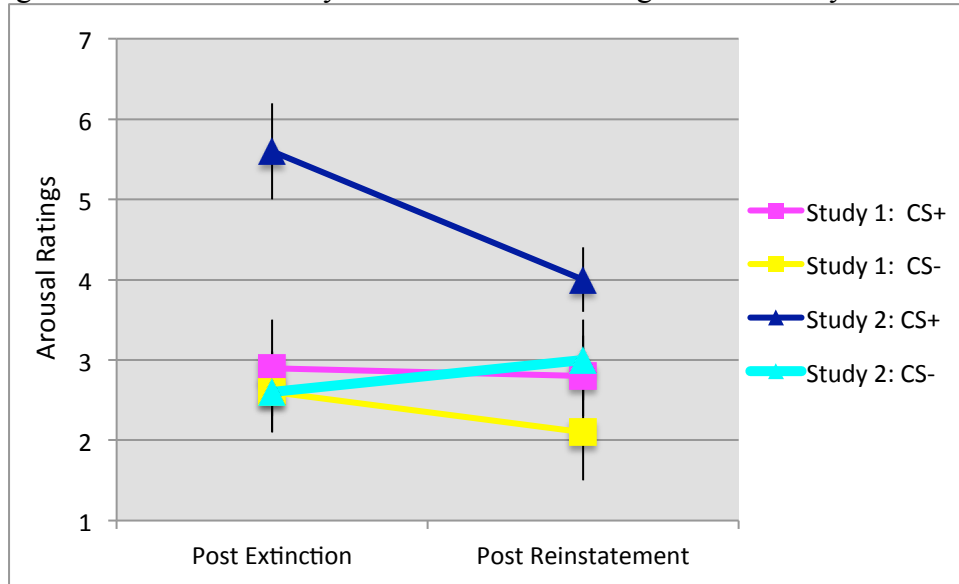
Figure 42.
Comparing Reinstatement in Study 1 and 2: Valence Ratings in Social/Social Reinstatement



Arousal Ratings

Social/Physical Reinstatement: There was a significant three-way Reinforcement x Time x Study interaction ($F(1,18)=6.74, p=0.018, \eta^2=0.04$), but no significant two-way interactions. Tests of simple main effects indicated significantly higher CS+ in Study 2 than Study 1 at Post-Extinction ($F(1,18)=4.77, p=0.042$), significantly higher CS+ than CS- in Study 1 at Post-Extinction ($F(1,18)=12.63, p=0.002$), and found a significant effect of Time on CS+ in Study 2 ($F(1,18)=8.49, p=0.002$).

Figure 43.
Comparing Reinstatement in Study 1 and 2: Arousal Ratings in Social/Physical Reinstatement



Social/Social Reinstatement: There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

SCR

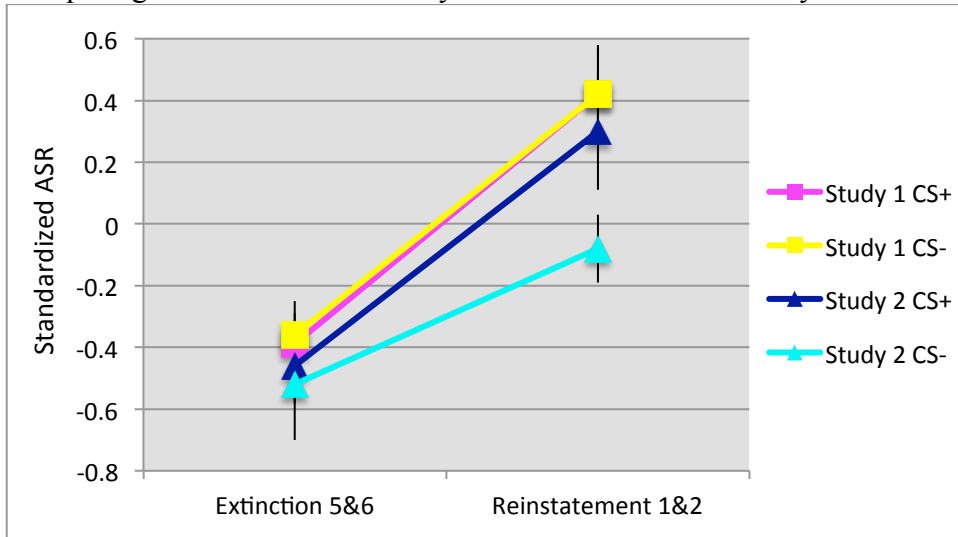
Social/Physical Reinstatement: There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

Social/Social Reinstatement: There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

ASR

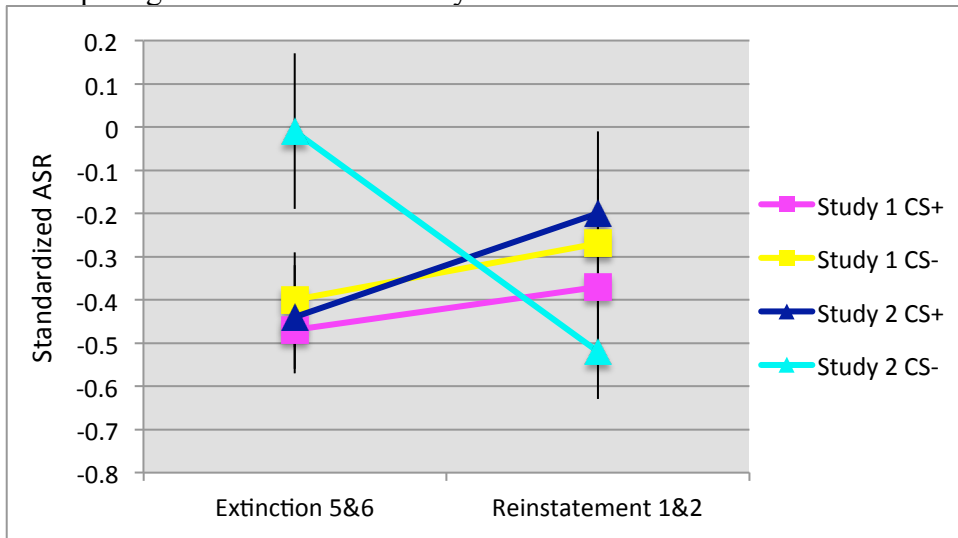
Social/Physical Reinstatement: There were no significant two- or three-way interactions, main effects or simple two-way interactions involving Study. Tests of simple main effects indicated a significant effect of Time on CS+ ($F(1,15)=5.47, p=0.012$) and CS- Study 1 ($F(1,15)=8.54, p=0.011$) and CS+ in Study 2 ($F(1,15)=5.02, p=0.041$).

Figure 44.
Comparing Reinstatement in Study 1 and 2: ASR in Social/Physical Reinstatement



Social/Social Reinstatement: There were no significant two- or three-way interactions, main effects or simple two-way interactions involving Study. Tests of simple main effects indicated a significant effect of Time on CS- in Study 2 ($F(1,21)=5.47, p=0.029$).

Figure 45.
Comparing Reinstatement in Study 1 and 2: ASR in Social/Social Reinstatement



PPG

Social/Physical Reinstatement: There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

Social/Social Reinstatement: There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study.

Predictors of change in responding from end of extinction to post-reinstatement

Data from Study 1 and Study 2 were collapsed for multiple linear regression analyses conducted examining predictors of change in responding between the end of extinction and post-reinstatement test trials. Outcome measures included change in CS+, change in CS-, and change in the differential between CS+ and CS- for the following outcome measures: S-US expectancy, P-US expectancy, Valence ratings, Arousal ratings, SCR, ASR, and PPG.

Several variables were calculated to be included as predictors in the regression analyses. *SCR percent change*, a measure of total change from SCR during acquisition USs to SCR during reinstatement USs, was calculated as: $(\text{average SCR to US during reinstatement} - \text{average SCR to US during acquisition}) / (\text{average SCR to US during reinstatement} + \text{average SCR to US during acquisition})$. Higher SCR percent change indicates less concordance between SCR from acquisition to reinstatement. *PPG percent change*, a measure of total change from PPG during acquisition USs to PPG during reinstatement USs, was calculated as: $(\text{average PPG to US during reinstatement} - \text{average PPG to US during acquisition}) / (\text{average PPG to US during reinstatement} + \text{average PPG to US during acquisition})$. Higher PPG percent change indicates less concordance between SCR from acquisition to reinstatement.

Demographics and baseline measures that were entered into the first level of the linear regression model included: age, gender, ethnicity, BDI, ASI, and Mini-SPIN. Predictors entered

into the model in a stepwise approach included: self-selected muscle stimulation level, average SCR response during reinstatement, SCR percent change, average PPG response during reinstatement, and PPG percent change.

P-US expectancy

Average SCR response during reinstatement significantly predicted variability in differential CS change in P-US from extinction to reinstatement, above and beyond the variability accounted for by demographic and baseline variables ($F(1,38)=5.23$, $p=0.028$, $R^2=0.106$, $\beta^*_i=-0.370$). No predictors significantly predicted CS+ change from extinction to reinstatement or CS- change in P-US expectancy from extinction to reinstatement above and beyond the variability accounted for by demographic and baseline variables.

S-US expectancy

PPG percent change significantly predicted variability in CS+ change in S-US expectancy from extinction to reinstatement, above and beyond the variability accounted for by demographic and baseline variables ($F(1,35)=4.36$, $p=0.043$, $R^2=0.105$, $\beta^*_i=-0.344$). No predictors significantly predicted CS- change in S-US expectancy from extinction to reinstatement or differential CS change from extinction to reinstatement above and beyond the variability accounted for by demographic and baseline variables.

Valence

Self-selected muscle stimulation level significantly predicted variability in CS+ change in valence ratings from extinction to reinstatement, above and beyond the variability accounted for by demographic and baseline variables ($F(1,38)=12.19$, $p=0.001$, $R^2=0.219$, $\beta^*_i=0.496$). Self-selected muscle stimulation level ($F(1,38)=6.58$, $p=0.014$, $R^2=0.131$, $\beta^*_i=0.383$) significantly predicted variability in differential CS change from extinction to reinstatement, above and

beyond the variability accounted for by demographic and baseline variables. No predictors significantly predicted CS- change in valence ratings from extinction to reinstatement above and beyond the variability accounted for by demographic and baseline variables.

Arousal

No predictors significantly predicted variability in CS+ change, CS- change, or differential CS change in arousal ratings from extinction to reinstatement, above and beyond the variability accounted for by demographic and baseline variables.

SCR

Average SCR response during reinstatement significantly predicted variability in CS+ change in SCR from extinction to reinstatement, above and beyond the variability accounted for by demographic and baseline variables ($F(1,41)=5.01$, $p=0.044$, $R^2=0.078$, $\beta_i^*=0.317$). Average SCR response during reinstatement significantly predicted variability in CS- change in SCR from extinction to reinstatement, above and beyond the variability accounted for by demographic and baseline variables ($F(1,41)=4.44$, $p=0.041$, $R^2=0.084$, $\beta_i^*=0.330$). No predictors significantly predicted differential CS change from extinction to reinstatement above and beyond the variability accounted for by demographic and baseline variables.

ASR

No predictors significantly predicted variability in CS+ change, CS- change, or differential CS change in ASR from extinction to reinstatement, above and beyond the variability accounted for by demographic and baseline variables.

PPG

Average PPG response during reinstatement significantly predicted variability in CS- change in PPG from extinction to reinstatement, above and beyond the variability accounted for

by demographic and baseline variables ($F(1,41)=5.27, p=0.027, R^2=0.079, \beta^*_i=0.287$). No predictors significantly predicted CS+ change or differential CS change in PPG from extinction to reinstatement above and beyond the variability accounted for by demographic and baseline variables.

Discussion:

Three hypotheses were tested in study 2. First, Study 2 aimed to replicate findings from Study 1 of observed differential reinstatement in Social/Social participants and non-differential reinstatement in Social/Physical participants. Second, it was hypothesized that the socially relevant CS+ included in Study would elicit stronger acquisition and reinstatement of conditional responding than was observed in Study 1 Social/Social and Social/Physical groups. Third, it was hypothesized that examining data combined from Study 1 and Study 2 would indicate that greater physiological arousal experienced during the reinstatement period would elicit greater return of conditional responding.

Stimulus Ratings

The only significant difference between Study 1 and Study 2 stimulus ratings was higher P-US distress in Study 1, which can likely be attributed to half of Study 1 participants completing P-US acquisition versus none completing P-US acquisition in Study 2. This interpretation is consistent with the P-US acquisition participants across having significantly higher P-US distress than S-US acquisition participants.

It is notable that the novel S-US utilized in this methodology was rated as equivalently distressing as the P-US. This finding was somewhat surprising given that muscle stimulation has been firmly established as a US aversive enough to support associative learning while only a small number of studies have successfully utilized social pain in associative learning procedures

(Lissek et al., 2008). The equivalence in P-US and S-US distress ratings is particularly surprising in light of low believability ratings of social feedback, with an average of 2.35, on a “1=not believable” to “7=very believable” likert scale. Indeed, if future methodological adjustments are able to enhance S-US believability then social pain might be utilized as a particularly effective US in associative learning research. It should be noted that participants in this research were selected based on moderate social anxiety symptoms (6 or more on the Mini-SPIN) as were participants in previous research utilizing S-USs in associative learning procedures (Lissek et al., 2008), and were thus likely highly sensitive to social pain and rejection. Therefore, it should not be expected that the effectiveness of the S-US methodology utilized in this study would generalize to a non-socially anxious population.

It is interesting that the acoustic startle probe, a brief blast of white noise, was rated as significantly more aversive than either US in these studies. While auditory stimuli have been utilized as aversive US in conditioning research, acoustic startle probes have also been widely used in conditioning research (Grillon, 2002) without being observed to interfere or compete with excitatory learning driven by a different primary US. Research using potentiated startle as a measure of fear conditioning requires methodological care (as was done in this research) to ensure equal contingency between startle probes and other study stimuli (i.e. CS+, CS-, US, ITI) so that startle probes themselves do not drive associative learning. High distress to the acoustic startle probes might be particularly relevant to the current research given that both loud noises and the P-US would be expected to elicit physical threat responses consistent with acute fear, while the S-US would be expected to elicit social threat responses serving to avert attack or rejection from dominant conspecifics (Öhman, 1986).

Replicating Differential Reinstatement and Non-Differential Reinstatement/Sensitization

Before examining the effect of physical and social pain on reinstatement it was necessary to demonstrate that both acquisition and extinction had occurred. Evidence of both acquisition and extinction were found for S-US expectancy and arousal ratings. For PPG there was moderate support for acquisition as evidenced by increased differential between CS+ and CS-, although overall responding to CS+ did not increase. There was no evidence of an extinction curve in PPG, but by the end of extinction there was no significant difference between CS+ and CS-. There was no evidence of acquisition or extinction as measured by P-US expectancy (as would be expected in S-US acquisition), valence ratings, SCR, or ASR.

Participants in the Social reinstatement group demonstrated significant reinstatement of differential responding to CS+ and CS- as measured by S-US expectancy. The Social reinstatement group also demonstrated differential responding to CS+ and CS- in ASR, although this result might be best interpreted as showing sensitization given that no acquisition had been observed in ASR. No reinstatement was observed in the Social reinstatement group for arousal or valence ratings, PPG, or SCR.

Following the reinstatement period participants in the Physical reinstatement group demonstrated a significant CS+ increase in ASR, and a significant CS- decrease in negative valence such that there was a significant difference between CS, although each of these results might be best interpreted as showing sensitization given that no acquisition had been observed in ASR or valence. The Physical reinstatement group also showed a significant CS- increase in P-US expectancy, again consistent with non-differential reinstatement/sensitization. No differential or non-differential reinstatement was observed in the Social/Physical group for arousal ratings, SCR, or PPG. Overall, these results are consistent with the Study 1 results, replicating the

finding of differential reinstatement for Social/Social participants and non-differential reinstatement Social/Physical participants.

Belongingness: Effect of socially relevant CS+ on conditional responding

Acquisition: Compared with participants presented with a socially irrelevant CS+ paired with S-US during acquisition (Study 1), participants who had a socially relevant CS+ paired with S-US during acquisition (Study 2) had higher negative valence to CS+ after habituation, higher negative valence and arousal to CS + after acquisition, and significantly greater differential in valence and arousal to CS+ relative to CS- after acquisition. Participants who received the S-US and socially relevant CS+ showed acquisition of conditional responding in PPG, while participants receiving the socially irrelevant CS+ did not. Participants who received the S-US and socially irrelevant CS+ showed elevated P-US expectancy to the CS- relative to the CS+. CS+ belongingness did not significantly influence S-US expectancy during acquisition, which is notable given that S-US expectancy was the strongest and most reliable measure of social pain acquisition learning across both studies. Overall, these results provide strong support that social relevance of the CS+ enhanced acquisition learning to S-US across a number of measures.

Extinction: Compared with participants who received the S-US and socially irrelevant CS+ during acquisition, participants who received the S-US and socially relevant CS+ reported greater negative valence and arousal to CS+ relative to CS- before and after extinction. These results indicate that participants who received the S-US and socially relevant CS+ did not extinguish conditional responding in terms of valence and arousal. It is possible that this finding is the result of particularly strong acquisition learning in valence and arousal observed by Study 2 participants, and also possible that six extinction trials for each CS were insufficient to achieve optimal extinction.

Reinstatement

P-US Reinstatement: The effect of the socially relevant CS+ on reinstatement in Social/Physical participants was somewhat mixed. Study 2 Social/Physical reinstatement participants receiving the socially relevant CS+ showed higher P-US expectancy to CS+ following extinction, but did not demonstrate a significant increase in both CS+ and CS- following reinstatement as did Study 1 participants receiving the socially irrelevant CS+. Social/Physical reinstatement participants receiving the socially relevant CS+ reported higher negative valence and arousal to CS+ both before and after reinstatement, and showed a decrease in negative valence to CS- following reinstatement. Social/Physical participants receiving the socially relevant CS+ showed a differential increase in ASR following reinstatement (significant increase in CS+ but not CS-), while those who received the socially irrelevant CS+ showed a significant increase in ASR for both CSs. This finding could be taken to suggest that the socially relevant CS+ promoted reinstatement of differential responding while the socially irrelevant CS+ supported non-specific sensitization. However, given that ASR acquisition was not observed in either study with either US, these results are most likely indicative of non-differential reinstatement/sensitization in the Social/Physical group rather than differential reinstatement.

S-US Reinstatement: The effect of the socially relevant CS+ on reinstatement in the Social/Social group was somewhat mixed. Social/Social participants receiving the socially relevant CS+ showed a significant decrease in negative valence to the CS+ following reinstatement, while participants receiving the socially irrelevant CS+ did not. Social/Social participants who received the socially relevant CS+ also showed a significant decrease in ASR to the CS- following reinstatement, while participants receiving the socially irrelevant CS+ did not.

Physiological Predictors of Reinstatement

After controlling for baseline measures and demographics, greater PPG percent change from acquisition to reinstatement predicted significantly greater decrease in CS+ for S-US expectancy, while greater PPG response during reinstatement predicted greater CS- increase in PPG following reinstatement. After controlling for baseline measures and demographics, higher SCR response during the reinstatement period predicted significantly greater CS+ and CS- increase in SCR following reinstatement. Additionally, higher SCR response during the reinstatement period predicted significantly greater decrease in CS differential for P-US expectancy. After controlling for baseline measures and demographics, higher self-selected levels of muscle stimulation predicted significantly greater increase in CS+ and in CS differential for valence ratings. These findings provide some preliminary evidence that arousal experienced during reinstatement influences post-reinstatement responding, although the results are somewhat underwhelming given the relatively few significantly predictors of 4 self-report and 3 physiological outcome measures. These findings do not support the similar-arousal theory of reinstatement that concordance between UR-arousal experienced during acquisition and reinstatement may predict greater differential reinstatement, but rather indicate that overall higher arousal experienced during reinstatement predicted a greater non-differential reinstatement.

Conclusions

These two studies utilized novel methodology to conduct standard and cross-US reinstatement with a socially painful US (S-US) and physically painful US (P-US), in order to examine the role of associative and arousal-based factors in the reinstatement of conditioned fear. One important finding from this research was the effectiveness of the S-US in supporting

both acquisition and reinstatement. This methodology built upon previous studies of socially painful USs in conditioning (simultaneous presentation of angry face plus auditory insult; Lissek et al., 2008) and added a personally-relevant social pain manipulation designed to strengthen the aversive quality of the S-US (Eisenberger, 2012). Additionally, the S-US (picture of angry face, auditory negative feedback) and the P-US (picture of electrical spark, muscle stimulation) were matched as discrete, compound USs each with a visual component.

As a means of examining associative factors involved in reinstatement of fear, standard reinstatement US (Social/Social and Physical/Physical groups) was compared with cross-US reinstatement (Social/Physical and Physical/Social groups). Interestingly, no overall differences were found between standard and cross-US reinstatement, suggesting that US similarity alone does not drive reinstatement. However, across both studies the Social/Social group was the only group to show consistent differential reinstatement, while the Physical/Physical and Social/Physical groups showed non-differential reinstatement in post-reinstatement test trials. The Physical/Social group did not demonstrate any change in responding following the reinstatement manipulation. There is reason to believe that methodological factors (discussed later) may have contributed to non-differential reinstatement rather than differential-reinstatement in the P-US reinstatement group, particularly for Physical/Physical participants, as there is substantial extant human research demonstrating differential reinstatement in shock/shock experimental designs (LaBar & Phelps, 2005, Dirikx et al., 2004; Dirikx et al., 2007; Hermans et al., 2005; Sokol & Lovibond, 2012). The Physical/Social group showed no evidence of cross-US reinstatement, while the Social/Physical group showed non-differential reinstatement. Thus, there is that some evidence that qualitatively distinct aversive USs can elicit cross-US non-differential reinstatement. There was no evidence for cross-US differential

reinstatement, as there was for standard reinstatement in the Social/Social group. Sokol and Lovibond (2012) found evidence for cross-US differential reinstatement, but the USs in their study (shock, loud noise) were qualitatively similar in eliciting physical threat CRs.

Does physiological arousal predict reinstatement?

The findings from these studies provide extremely preliminary evidence that after controlling for demographics and baseline measures, greater physiological arousal (PPG, ASR) during reinstatement predicts non-differential post-reinstatement return of differential responding to both CS+ and CS-. Alternatively, there was no evidence that concordance in physiological arousal experienced during reinstatement and acquisition was predictive of greater differential post-reinstatement responding. These results thus provide tentative support for the non-specific arousal-based account of reinstatement adapted from the drug-relapse literature (Stewart, 2000, 2008), suggesting that higher arousal during reinstatement predicts non-differential increases in responding to both CS+ and CS- (i.e., sensitization).

It is worth noting that the observation of concordance predicting differential reinstatement specifically involved concordance in blushing response (PPG) predicting differential reinstatement in expectancy of social pain. A possible implication is that experiencing arousal unique to social pain during reinstatement may be particularly predictive of differential return of social fear. However, this conclusion is questionable given that in Study 1 physical pain elicited acquisition of differential PPG responding, suggesting that to some extent PPG captures general arousal rather than a unique social-threat response.

Differential social pain reinstatement and non-differential physical pain reinstatement?

An unexpected finding from this research was that Social/Social participants demonstrated differential reinstatement of conditional responding, while P-US reinstatement

participants showed non-differential reinstatement (i.e., increased responding to both CS+ and CS-) regardless of their acquisition US. Most previous human research has demonstrated that unpaired shocks presented following extinction produce differential reinstatement (Dirikx et al., 2004; Dirikx et al., 2007; Hermans et al., 2005; Sokol & Lovibond, 2012), although several studies have also found non-differential reinstatement to the CS+ and CS- (Dirikx et al., 2009; Kull et al., 2012). In the current research it was interesting that negative social feedback elicited standard differential reinstatement across both studies (with Social/Social participants), while in both studies muscle stimulation elicited non-differential reinstatement.

Several theories of reinstatement have been proposed with the potential to address non-differential reinstatement, all based on the effects of context conditioning (for more thorough review see Dirikx et al., 2009). One account suggests that reinstatement results from summation between excitatory context conditioning elicited by reinstating USs being paired with residual excitatory value of the CS+ following extinction (Bouton & Bolles, 1979). According to the theory, if contextual fear is strong enough following reinstatement it might potentiate non-differential increase in responding to both CS. Two other accounts contend that both CS+ and CS- can form associations with the conditioning context, and after reinstating USs become associated with the context non-differential responding to both CS may occur as the result of higher order conditioning/associative chaining (Westbrook et al., 2002; Schmajuk, Larrauri, & LaBar, 2007). These theories all offer useful consideration for understanding reinstatement, but as they do not pertain to the effects of qualitatively distinct USs they are not optimally suited for explaining the current findings.

One explanation of sorts for these findings is that P-US reinstatement generated such high arousal that all subsequent responding was potentiated, while the lower level of arousal

generated by S-US reinstatement was sufficient to recall S-US acquisition learning but not to potentiate overall responding. This interpretation would be consistent with the previously discussed findings of physiological predictors of reinstatement; higher arousal during reinstatement predicted non-differential increase in responding, while concordance in social threat related arousal (blushing) experienced during reinstatement and acquisition predicted greater differential responding. However, this interpretation is more descriptive than explanatory, and fails to address *why* arousal generated by social and physical pain seemed to have different effects on reinstatement.

The AESOP model of associative learning may be a useful lens for viewing the findings of non-differential P-US reinstatement but differential S-US reinstatement. Following the acquisition and extinction phases, the CS+ would be expected to have formed excitatory as well as inhibitory connections with the acquisition US. More specifically, S-US acquisition participants should have formed inhibitory CS-US connections (during extinction) that compete with previously formed excitatory sensory associations of social feedback and excitatory emotional associations of social threat/pain. Similarly, P-US acquisition participants should have formed inhibitory CS-US connections (during extinction) that compete with previously formed excitatory sensory associations of shock and excitatory emotional associations of physical threat/pain. Another stimulus that should be considered in these findings is the acoustic startle probe (brief blasts of white noise), which unexpectedly was rated by participants as the most aversive stimulus in the study. Loud noises may be used to support fear conditioning as a US which signals physical threat, and as startle probes were presented throughout all study phases except reinstatement during CS+, CS-, and ITI with no contingency for prediction it is likely that they elicited contextual fear conditioning (e.g., Fanselow, 1980; Rescorla & Wagner, 1972;

Vansteenwegen et al., 2008). Thus, by the end of extinction we would expect that the context should have formed an excitatory association with the startle probes, such that the context would recall the startle probe's emotional response (i.e., threat of physical pain) and sensory elements.

According to AESOP, unpaired US presentations during reinstatement should have had two types of associative effects. First, the unpaired reinstating US should have elicited excitatory conditioning to the context. So, following S-US reinstatement it would be expected that the context would recall the S-US emotional response (threat of social pain), while following P-US reinstatement the context would recall the P-US response (threat of physical pain). Second, in the standard reinstatement (Physical/Physical, Social/Social) the reinstating US should have acted as a reminder of the CS+. Other than noting that USs can generate responses while CSs typically cannot, AESOP treats CSs and USs as similar stimuli with regards to associative learning processes. Given that excitatory learning occurred between CS+ and US during acquisition, the sensory and emotional nodes of a standard reinstating US should selectively recall previously learned excitatory associations with the CS+. This recalling of excitatory CS+ associations should not occur with a qualitatively different US in cross-US reinstatement (e.g., in Physical/Social participants, the sensory and emotional elements of the reinstating S-US should not recall an excitatory association between P-US and CS+).

In sum, by the post-reinstatement test phase, standard reinstatement participants could be expected to have three distinct associative relationships influencing conditional responding: context conditionings from startle probe, context conditioning from reinstating US, and cued conditioning to CS+. Physical/Physical participants would be influenced by contextual fear of physical threat (from startle probes), contextual fear of physical threat (from P-US), and fear of physical threat from CS+ (from P-US). Social/Social participants would be influenced by

contextual fear of physical threat (from startle probes), contextual fear of social threat (from S-US), and fear of social threat from CS+ (from S-US). Alternatively, in the post-reinstatement phase cross-US reinstatement participants could be expected to have only two distinct associative relationships influencing responding: context conditionings from startle probe and context conditioning from reinstating US. At post-reinstatement, Physical/Social participants would be influenced by contextual fear of physical threat (from startle probes) and contextual fear of social threat (from S-US). At post-reinstatement, Social/Physical participants would be influenced contextual fear of physical threat (from startle probes) and contextual fear of physical threat (from P-US).

These predictions of associative differences between study groups offer a potential explanation for why non-differential reinstatement was observed for P-US reinstatement participants while differential reinstatement was observed in Social/Social participants. Though sensitization has traditionally been thought of as a non-associative process (e.g., Davis, 1989), more recent research suggests that sensitization of startle response following unpaired shocks is mediated by contextually conditioned fear (Richardson, 2000). The startle probe and P-US each elicit physical threat responses, so it would be expected that the emotional elements of both stimuli which enter into excitatory associations with other stimuli should be very similar. By post-reinstatement, P-US reinstatement participants would have acquired contextual fear of physical threat in association with both startle probes and P-US, and since the emotional elements of each association are fear of physical threat they could be expected to have an additive effect in contributing to greater contextual fear of physical threat (i.e, summation). By post-reinstatement, S-US reinstatement participants would have acquired contextual fear of physical threat in association with startle probes and contextual fear of social threat in

association with S-US, but since the emotional elements of each association are different there should be no summation or additive effect in contributing to greater contextual fear of physical threat.

Additionally, in both standard reinstatement groups reinstating US presentations should have recalled the excitatory CS-US association from acquisition, which presumably would have contributed to differential reinstatement of responding to the CS+ but not CS-. Differential reinstatement was indeed observed in Social/Social participants, but in the Physical/Physical group, which showed non-differential reinstatement. It may be that particularly high levels of contextual fear of physical threat potentiated overall responding beyond the point of observable CS differentiation. This interpretation is consistent with findings that anxious individuals tend to show more non-differential return of fear, possibly because of elevated contextual fear (for review, see Vervliet, Craske, & Hermans, 2013). This same effect of contextually potentiated responding would explain non-differential reinstatement in the Social/Physical group as well, although this group would not have been expected to show CS differentiation because the excitatory CS-US association was not primed during cross-US reinstatement. The fact that Social/Physical participants showed sensitization following reinstatement on P-US expectancy (not S-US expectancy), a measure on which they did not demonstrate acquisition, provides evidence that Social/Physical reinstatement was driven by fear of physical threat, not fear of social threat learning during acquisition. This finding is consistent with Sokol and Lovibond's (2012) suggestion that cross-US reinstatement involves the development of new fears, not the reactivation of old ones. Physical/Social participants did not show any reinstatement of responding.

In addition to reconciling the divergent findings between P-US and S-US within this research, this explanation is also generally consistent with other human experimental reinstatement studies. Of the published studies demonstrating shock-elicited differential reinstatement in humans (LaBar & Phelps, 2005, Dirikx et al., 2004; Dirikx et al., 2007; Hermans et al., 2005; Sokol & Lovibond, 2012), none included startle probes as part of their procedures, so none would have had the startle-plus-shock additive effect on contextual fear of physical threat. It should be noted that two human experimental reinstatement studies which found non-differential reinstatement included shocks but not startle probes, which is contrary to the explanation provided here (Dirikx et al., 2009; Kull et al., 2012). The one study that utilized acoustic startle probes in demonstrating reinstatement of potentiated startle in humans did not use a shock as US, but rather a blast of air to the throat as the US (Norrholm et al., 2006).

Future research should further explore whether post-extinction unpaired shocks in conjunction with non-contingent acoustic startle probes elicit non-differential reinstatement, while post-extinction unpaired shocks in the absence of startle probes elicit differential reinstatement. Pursuing this line of research may help clarify the associative factors contributing to reinstatement, both differential and non-differential. For example, would reinstating shocks have the same effect in conjunction with non-contingent acoustic startle probes as in conjunction with paired, contingent acoustic startle probes?

Belongingness

S-US acquisition was demonstrated in both studies, but participants in Study 2 who received the socially relevant CS+ (a neutral face comprised of a yellow circle with two black dots and one black line) showed significantly stronger acquisition of conditional responding as measured by self-report and physiological measures. This demonstration of enhanced excitatory

learning as the result of CS-US belongingness is in line with an extensive body of research indicating that CS-US learning is stronger when the UR is biologically and evolutionarily relevant to the CS (e.g., Garcia & Koelling, 1966; Rescorla, 2008). This is also consistent with the AESOP model, as elements in CS and US that “go together” should engender stronger associations. A minor but interesting finding regarding the socially relevant CS+ was that it was rated with more negative valence than the non-socially relevant CS+ following habituation, though it was not higher on other measures after baseline. This group difference before the onset of conditioning suggests that participants selected for moderate to high social anxiety experience stronger negative affect to even seemingly neutral social stimuli. Participants who received the socially relevant CS+ showed diminished extinction of self-reported negative valence and arousal relative to participants receiving the non-socially relevant CS+. As a general methodological consideration it is possible that in this study 6 extinction trials for each CS were insufficient to achieve optimal extinction. However, the diminished extinction observed to the socially relevant CS+ suggests that particularly robust acquisition learning was the result of CS-US belongingness.

The findings regarding the effect of CS belongingness on reinstatement were mixed, but there is some indication that CS+ belongingness may have promoted greater CS differential following both standard and cross-US reinstatement. For Social/Physical participants, those receiving the socially relevant CS+ showed greater decrease in responding to CS- following reinstatement on several measures, and showed greater differential increase to CS+ on ASR. Given the previously discussed effect of sensitization elicited by P-US reinstatement, it is especially notable that CS belongingness may have promoted differential CS responding following P-US reinstatement. The effect of CS+ belongingness on reinstatement for

Social/Social participants was less clear or easily understood (*why do Social/Social participants receiving the socially relevant CS+ show decreased negative valence to CS+ following reinstatement?*), but participants who received the socially relevant CS+ did show a greater decrease in ASR to the CS- following reinstatement. Together, these findings offer tentative evidence that CS+ belongingness promotes differential return of conditional responding in both standard and cross-US reinstatement, and that this effect is not simply the result of stronger acquisition learning.

Limitations

Several potential limitations of this research should be noted. First, the S-US may not have been sufficiently aversive to fully explore the effects of social pain on reinstatement. S-US acquisition participants only demonstrated acquisition of differential conditional responding on one physiological measure (PPG, in participants who received the socially relevant CS+), and this differential responding was driven by a decrease in CS- rather than increased responding to CS+. The only observed S-US reinstatement of physiological responding was SCR in Social/Social participants who received the socially relevant CS+. Thus, without the addition of a socially relevant CS+ the S-US may not have been a strong enough stimulus to elicit observable physiological change as the result of associative learning. Indeed, believability of the social feedback manipulation was rated as fairly low, as was overall distress to the S-US. However, there is some reason to believe that the S-US was at least moderately effective as a US given that the S-US was rated as equivalently distressing as the P-US. Threat of social pain may simply elicit a different physiological response pattern with less significant or discrete sympathetic nervous system activation than threat of physical pain (Ohman, 1985). Given the potential for temporal and qualitative aspects of the UR to drive associative processes (Wagner & Brandon,

1989) it may be that different CS-US timing parameters than those used in this research would be better for excitatory learning driven by social pain. If future research can further enhance S-US believability, perhaps through the use of in-person confederates giving the negative feedback, then social pain may be increasingly utilized in advancing understanding of differences between qualitatively distinct aversive USs or improving experimental analogues for social anxiety disorder (Lissek et al., 2008). Future research should also clarify whether S-USs can support associative learning in a non-socially anxious sample.

Another potential limitation of this research was that certain conditioning parameters may not have been ideal for observing reinstatement and other return of fear phenomena. Increasing the number of extinction trials used in this research (6 for each CS, 12 total) likely would have produced greater extinction of conditional responding, which might have allowed for clearer observation of post-reinstatement change in responding. Having a longer period of time between the end of extinction and the reinstatement phase would have made it possible to examine both reinstatement and spontaneous recovery of fear. Additionally, having a longer period of time between reinstatement and the post-reinstatement test phase would have allowed for better differentiation between changes in responding driven by residual arousal from the UR to the reinstating US (which should decrease with sufficient time) versus changes driven by associative processes (which should not significantly degrade over time). It was surprising that acoustic startle probes in this research were rated as significantly more distressing than either shock or social pain, particularly given that past research using identical parameters for startle probe and muscle stimulation level did not find startle to be as highly aversively (e.g., Glenn, Minor, Vervliet, & Craske, 2014). Future conditioning fear conditioning research should be mindful of

the potential for acoustic startle probes (intended to function as a measurement tool) to act as an aversive US driving fear of physical threat.

A third limitation in this research was that the effect of belongingness between CS and reinstating US was examined in only a limited number of acquisition/reinstatement US combinations due to concerns over sample size with additional groups. In future research the effect of CS-US belongingness on reinstatement should be examined more comprehensively through all combinations of CS relevance, acquisition US, and reinstating US. For example, what is the effect of a socially relevant CS+ on Physical/Social reinstatement, the effect of a physical-pain relevant CS+ on reinstatement, or the effect of belongingness in both CS+ and CS- on reinstatement?

Clinical Implications

This research provides the first evidence of fear reinstatement elicited by social pain, as all previous demonstration of human fear reinstatement used physical threat related USs such as shock, loud noise, and a blast of air to the neck (Norrholm et al., 2006). Social pain is a central concept in understanding the development and maintenance over time of social anxiety disorder (SAD), so demonstrating that experimentally induced social pain functions similarly to physical pain in both acquisition and reinstatement is important for validating laboratory models of SAD (Lissek et al., 2008). One interesting finding from this research was that while social pain reinstated previously extinguished fear of social threat, it did not reinstate previously extinguished fear of physical threat. Several possible reasons for this finding have already been discussed, including the S-US not being aversive enough, or the arousal following social pain not being strong enough. Whatever the reason, a tentative suggestion of this finding is that mild to moderately socially painful experiences may not reinstate fears of physical danger. For example,

following successful treatment of posttraumatic stress disorder (PTSD) we might not expect the experience of having a serious argument with a friend to reinstate symptoms of PTSD, while we would expect reinstatement following a physical assault or automobile accident. However, this research does not address the effect of severe social pain, such as being broken-up with by a significant other, which might function differently from mild to moderate social pain in its capacity to engender cross-US reinstatement of fear.

Unforeseen methodological issues involving the acoustic startle probes may have muddled the findings regarding cross-US reinstatement with qualitatively distinct USs. However, it is notable that Social/Physical participants (social pain during acquisition, physical pain during reinstatement) showed strong cross-US non-differential reinstatement (increased responding to both CSs), potentially as the result of additive context conditioning effects from startle probes and shocks. This unexpected finding is consistent with research showing the important role of contextual learning in reinstatement (Bouton et al., 2006). Additionally, this finding may suggest a useful model for understanding the interactions between chronic and acute stressors on reinstatement of fear.

Clinical studies and case examples suggest that the occurrence of stressful events following treatment for anxiety is associated with a greater return of fear (Rachman, 1979; Steketee, 1993; Wade et al., 1993), and experimental research has demonstrated differential fear reinstatement in humans following acute stressors (Dirikx et al., 2004; Dirikx et al., 2007; Hermans et al., 2005; Neumann, 2008; Sokol & Lovibond, 2012). Chronic stress has been observed and demonstrated to contribute to reinstatement of drug craving (Stewart, 2008; Sinha, 2008), but there is little research regarding the effect of chronic stressors on human fear reinstatement. One interpretation of the non-differential reinstatement findings from the current

research is that chronic stressors (startle probes presented during all study phases) produced elevated levels of chronic stress and anxiety (CR to contextual fear of startle probes) which exacerbated the response to an acute stressor (a series of unpredicted shocks) and subsequently produced a generalized increase in fearful responding (sensitization to both CS+ and CS-). It should be noted that this apparent additive effect of aversive stimuli leading to sensitization only occurred for stimuli both eliciting a similar emotional response (fear of physical threat); sensitization was not observed for social pain reinstatement participants, so it appears startle probes were not additive to the socially painful feedback.

Clinically, these results suggests that individuals under chronic stress are not only at elevated risk of an acute stressor triggering the return of a previously extinguished fear, but potentially at risk for this return of fear to be generalized beyond the original feared stimulus. A similar pattern of fear generalization and poor differential learning is seen in individuals with anxiety disorders, such as patients with PTSD who generalize fear across stimuli and are sensitized by stress (Grillon & Morgan, 1999). The overall findings from this research do not suggest that reinstatement requires chronic stress to occur, but rather that a discrete reinstating stressor experienced in the context of chronic stress may elicit a stronger and more generalized return of fear. This may be particularly true when the acute stressor involves physical threat. For example, a patient who completes successful treatment for social anxiety, then experiences a series of medical complications pertaining to physical health, and several months later survives a serious automobile accident might be expected to show renewed and more generalized social anxiety as well as new fear of driving.

The factors that contribute to reinstatement of fear, and more broadly to the return of fear, represent significant limitations to the effectiveness of exposure therapy for anxiety disorders.

These factors adversely affect many patients who experience a substantial reduction in fear during treatment but experience a return of fear following treatment (Craske & Mystkowski, 2006; Craske & Rachman, 1987; Rachman, 1989; Rose & McGlynn, 1997). This research suggests that a number of factors may increase the likelihood that an unexpected unpleasant event reinstates an extinguished fear, including qualitative similarity between the event and the originally fear, the amount of arousal experienced during the event, the relatedness of feared conditional stimuli to the original and reinstating events, and chronic stress or anxiety surrounding the acute event. A broad range of research is currently underway on therapeutic interventions that may increase the long-term effectiveness of treatments for anxiety disorders by mitigating factors contributing to reinstatement and return of fear (for review, see Vervliet, Craske, & Hermans, 2013).

APPENDIX 1: STUDY STIMULI

Participant Rating Form

Below is a list of words that may describe the person in the interview that you have just watched. Please place an "X" next to the words that you feel best describe the person. Please choose up to 10 words. Your ratings will be kept completely confidential.

- ADEQUATE
- ANNOYING
- ARROGANT
- ATTRACTIVE
- AWKWARD
- BORING
- COMPASSIONATE
- CONFUSING
- DECENT
- DUMB
- ENERGETIC
- ENGAGING
- ENTERTAINING
- FINE
- FORGETTABLE
- FUNNY
- INADEQUATE
- INFORMED
- INTELLIGENT
- INTERESTING
- KIND
- LIKEABLE
- MEAN
- OFF-PUTTING
- OKAY
- OPINIONATED
- PERSONABLE
- RESERVED
- RUDE
- SELFISH
- SENSITIVE
- SERIOUS
- SHALLOW
- SWEET
- TALKATIVE
- THOUGHTFUL
- UNIQUE
- UNLIKEABLE
- WEIRD
- WITTY

Social pain manipulation “Example Neutral Face” shown prior to conditioning



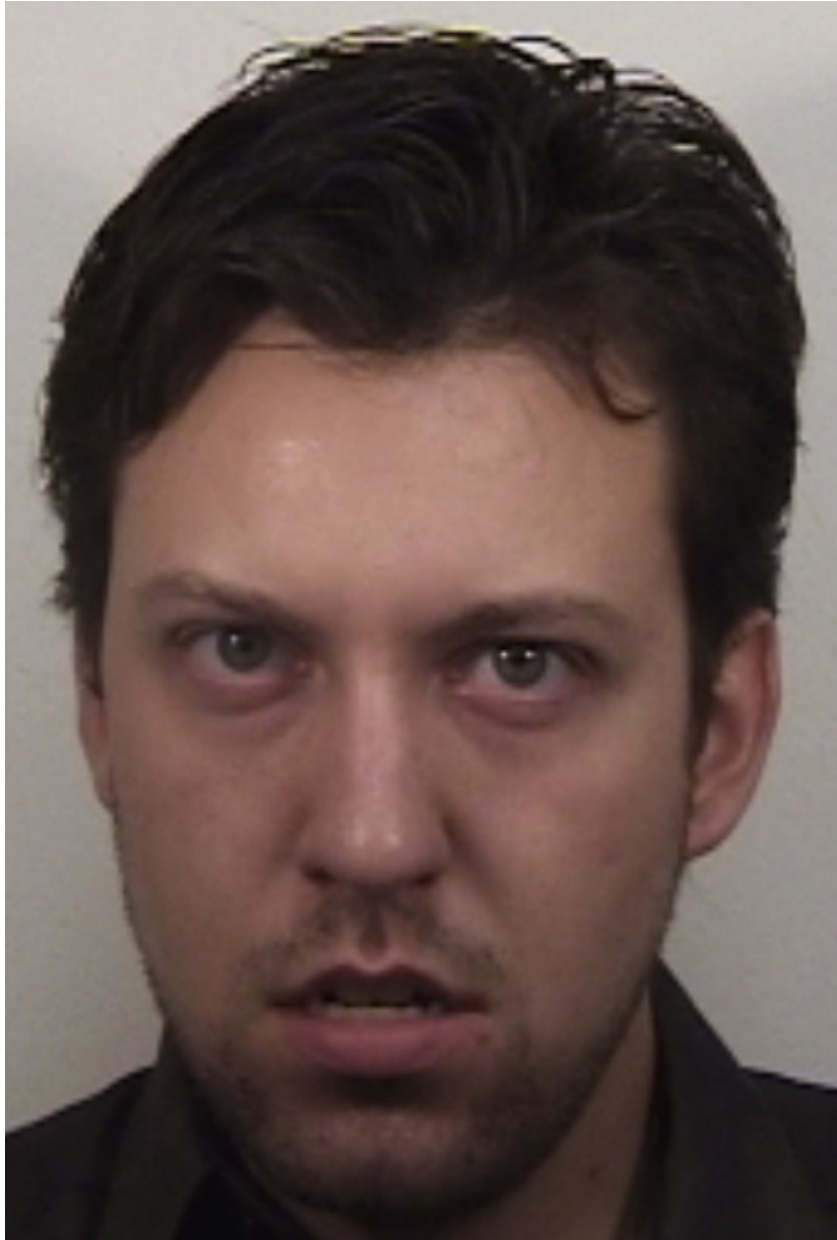
Social pain manipulation “Reviewer Face” shown prior to conditioning



“Neutral Face” shown during conditioning



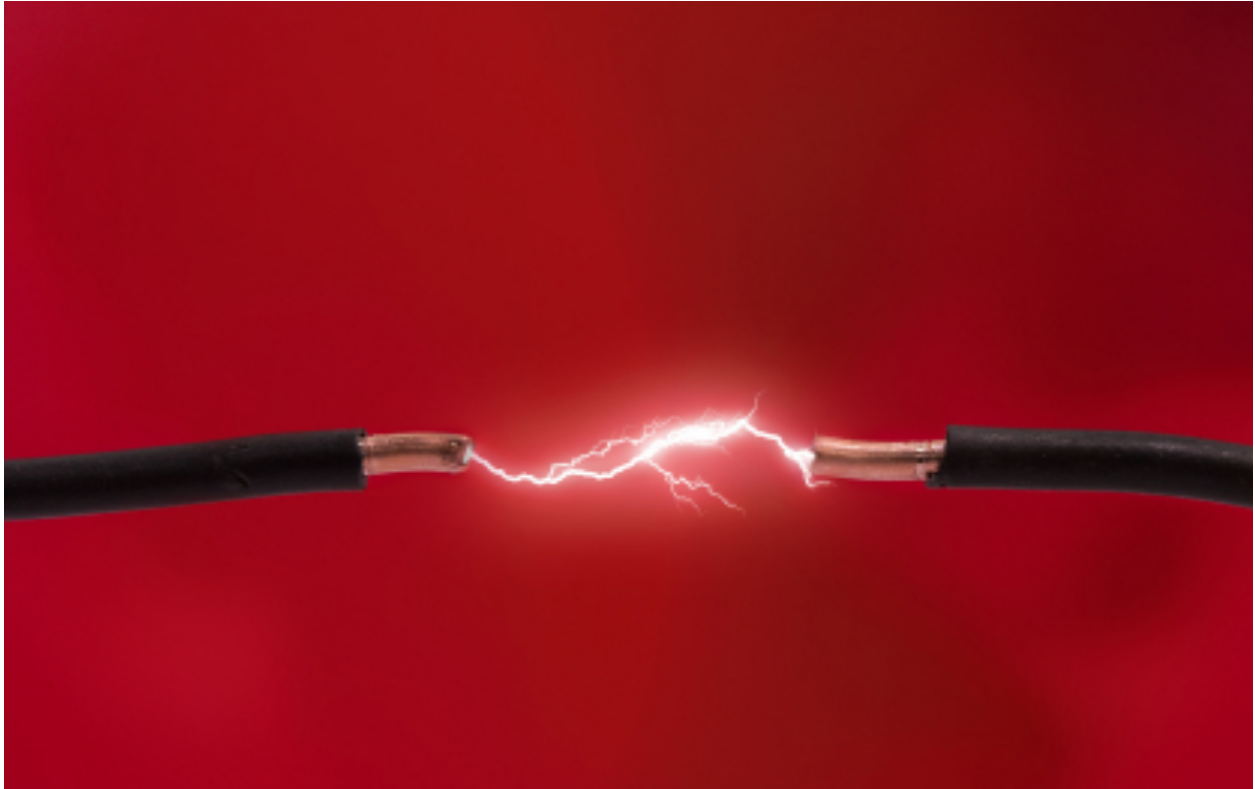
Social pain US angry picture



Social Pain US Auditory Negative Feedback:

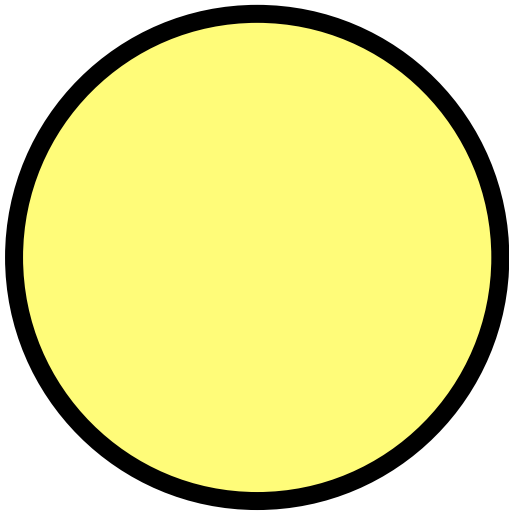
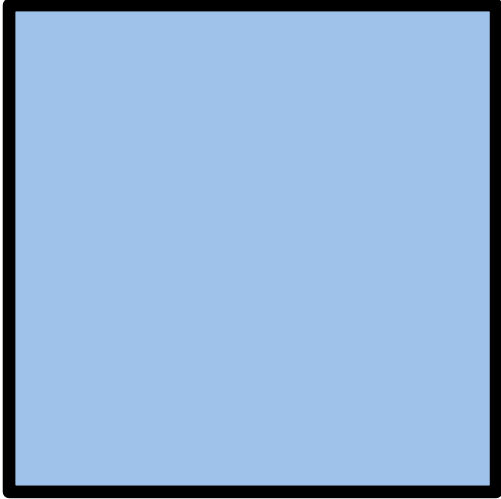
- *“Unlikeable”*
- *“Annoying”*
- *“Dumb”*
- *“Forgettable”*
- *“Inadequate”*
- *“Rude”*
- *“Selfish”*
- *“Shallow”*
- *“Off-putting”*
- *“Unlikeable”*

Physical Pain US (Simultaneous muscle stimulation plus picture of spark)

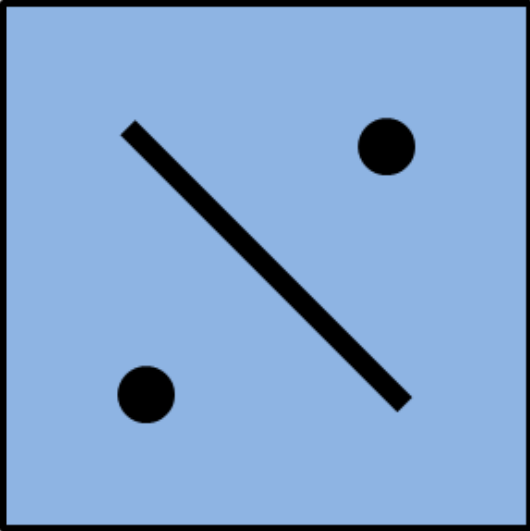


Picture presented simultaneously with shock delivery

Study 1 CS: Geometric shapes



Study 2: Geometric shape CS-



Study 2: Socially Relevant CS+



APPENDIX 2: STUDY 1 STATISTICAL OUTPUT

Table A1.

Study 1 Acquisition Means (SD): P-US Expectancy

Time	Social Acq. CS+	Social Acq. CS-	Physical Acq. CS+	Physical Acq. CS-
<i>Habituation</i>	3.71 (1.61)	3.47 (1.87)	3.73 (2.10)	3.75 (2.05)
<i>Acquisition 1&2</i>	2.55 (2.64)	3.38 (2.84)	4.62 (2.74)	3.55 (2.34)
<i>Acquisition 3&4</i>	1.75 (2.48)	2.02 (2.91)	6.16 (3.89)	2.18 (3.00)
<i>Acquisition 5&6</i>	1.4 (2.43)	2.15 (2.99)	6.92 (3.65)	0.97 (1.71)

Table A2.

Study 1 Acquisition Means (SD): S-US Expectancy

Time	Social Acq. CS+	Social Acq. CS-	Physical Acq. CS+	Physical Acq. CS-
<i>Habituation</i>	4.79 (2)	4.98 (2.11)	5 (2.24)	5.34 (2.42)
<i>Acquisition 1&2</i>	5.57 (2.86)	5.45 (2.89)	5.48 (2.94)	5.84 (2.76)
<i>Acquisition 3&4</i>	6.16 (3.35)	4.76 (3.82)	4.54 (3.81)	4.71 (3.82)
<i>Acquisition 5&6</i>	6.77 (3.18)	4.52 (4.09)	4.03 (4.03)	4.42 (3.61)

Table A3.

Study 1 Extinction Means (SD): P-US Expectancy

Time	Social Acq. CS+	Social Acq. CS-	Physical Acq. CS+	Physical Acq. CS-
<i>Extinction 1&2</i>	1.98 (2.46)	2.88 (2.70)	4.86 (2.64)	2.94 (2.67)
<i>Extinction 3&4</i>	1.68 (2.80)	2.03 (3.08)	2.40 (2.87)	1.23 (1.83)
<i>Extinction 5&6</i>	1.07 (2.37)	1.57 (3.00)	1.41 (2.02)	0.57 (1.00)

Table A4.
Study 1 Extinction Means (SD): S-US Expectancy

Time	Social Acq. CS+	Social Acq. CS-	Physical Acq. CS+	Physical Acq. CS-
<i>Extinction 1&2</i>	5.77 (2.46)	5.06 (2.70)	4.08 (2.64)	5.01 (2.67)
<i>Extinction 3&4</i>	4.17 (2.80)	4.26 (3.08)	3.12 (2.87)	3.93 (1.83)
<i>Extinction 5&6</i>	3.31 (2.37)	3.43 (3.00)	2.85 (2.03)	3.28 (1.00)

Table A5.
Study 1 Reinstatement Means (SD): P-US Expectancy

Time	S/S CS+	S/S CS-	P/P CS+	P/P CS-	S/P CS+	S/P CS-	P/S CS+	P/S CS-
<i>Extinction 5&6</i>	1.95 (3.15)	2.79 (3.89)	0.83 (1.54)	0.25 (0.61)	0.18 (0.40)	0.18 (0.75)	2.09 (2.37)	0.94 (1.25)
<i>Reinstatement 1&2</i>	2.47 (3.05)	3.01 (3.78)	2.92 (3.05)	2.63 (2.41)	2.65 (3.32)	2.65 (3.27)	2.92 (2.74)	1.98 (2.61)

Table A6.
Study 1 Reinstatement Means (SD): S-US Expectancy

Time	S/S CS+	S/S CS-	P/P CS+	P/P CS-	S/P CS+	S/P CS-	P/S CS+	P/S CS-
<i>Extinction 5&6</i>	3.95 (3.68)	3.68 (4.22)	2.9 (4.06)	3.64 (4.05)	2.9 (3.82)	3.64 (3.98)	2.78 (3.23)	2.79 (3.37)
<i>Reinstatement 1&2</i>	6.67 (2.65)	4.84 (3.26)	2.97 (3.86)	3.54 (3.67)	2.7 (4.04)	3.81 (4.27)	3.01 (3.31)	3.6 (3.68)

Table A7.
Study 1 Acquisition: P-US Expectancy

Test	<i>P</i>	DF	F	η^2
Reinforcement x Acquisition US x Time interaction	0.000	3, 50	24.69	0.08
Reinforcement x Acquisition US interaction	0.000	1, 50	34.57	0.011
Acquisition US x Time interaction	0.001	3, 50	5.48	0.03
Reinforcement x Trial interactions in the P-US Acquisition group	0.000	3, 27	29.71	0.17
Reinforcement x Trial interactions in the S-US Acquisition group	0.041	3, 23	2.91	0.02
Reinforcement x Acquisition interactions at Acquisition trials 1 and 2	0.009	1, 50	7.44	0.13
Reinforcement x Acquisition interactions at Acquisition trials 3 and 4	0.000	1, 50	17.32	0.24
Reinforcement x Acquisition interactions at Acquisition trials 5 and 6	0.000	1, 50	7.35	0.41
Acquisition US x Time interaction for the CS+	0.000	3, 50	18.56	0.25
Higher P-US Expectancy to the CS+ in the P-US Acquisition group than to the CS+ in the S-US Acquisition group at Acquisition trials 1 and 2	0.008	1, 50	7.59	
Higher P-US Expectancy to the CS+ in the P-US Acquisition group than to the CS+ in the S-US Acquisition group at Acquisition trials 3 and 4	0.000	1, 50	22.72	
Higher P-US Expectancy to the CS+ in the P-US Acquisition group than to the CS+ in the S-US Acquisition group at Acquisition trials 5 and 6	0.000	1, 50	39.68	
Higher CS+ than CS- in the P-US Acquisition group at Acquisition trials 1 and 2	0.029	1, 50	7.59	
Higher CS+ than CS- in the P-US Acquisition group at Acquisition trials 3 and 4	0.000	1, 50	40.36	
Higher CS+ than CS- in the P-US Acquisition group at Acquisition trials 5 and 6	0.000	1, 50	104.72	
Time on the CS+ in the S-US Acquisition group	0.013	3, 48	4.01	
Time on the CS- in the S-US Acquisition group	0.019	3, 48	3.67	
Time on the CS+ in the P-US Acquisition group	0.000	3, 48	8.83	
Time on the CS- in the P-US Acquisition group	0.000	3, 48	12.85	

Table A8.
Study 1 Acquisition: S-US Expectancy

Test	<i>P</i>	DF	F	η^2
Reinforcement x Acquisition US x Time interaction	0.016	3, 50	3.55	0.02
Reinforcement x Acquisition US interaction	0.031	1, 50	4.91	0.02
Reinforcement x Trial interaction	0.014	3, 50	3.66	0.02
Acquisition US x Time interaction	0.006	3, 50	4.29	0.03
Reinforcement x Trial interactions in the Social Acquisition US group	0.002	3, 23	5.49	0.06
Reinforcement x Acquisition interactions at Acquisition trials 5 and 6	0.013	1, 50	6.64	0.11
Acquisition US x Time interaction for the CS+ Higher S-US Expectancy to the CS+ in the S-US Acquisition group than in the P-US Acquisition group at Acquisition trials 5 and 6	0.000	3, 50	8.08	0.14
Higher CS+ than CS- in the S-US Acquisition group at Acquisition trials 3 and 4	0.010	1, 50	7.18	
Higher CS+ than CS- in the S-US Acquisition group at Acquisition trials 5 and 6	0.031	1, 50	4.91	
Higher CS+ than CS- in the S-US Acquisition group at Acquisition trials 5 and 6	0.004	1, 50	8.98	
Time on the CS+ in the S-US Acquisition group	0.026	3, 48	3.37	
Time on the CS+ in the P-US Acquisition group	0.036	3, 48	1.22	
Time on the CS- in the P-US Acquisition group	0.041	3, 48	2.98	

Table A9.
Study 1 Acquisition: Valence Ratings

Test	P	DF	F	η^2
Reinforcement x Acquisition US x Time interaction	0.00	1, 48	13.03	0.10
Reinforcement x Acquisition US interaction	0.006	1, 48	8.36	0.04
Reinforcement x Trial interactions in the Physical Acquisition US group	0.009	1, 27	8.40	0.14
Reinforcement x Trial interaction in the Social Acquisition US group	0.014	1, 21	6.96	0.09
Reinforcement x Acquisition interactions at Post Acquisition	0.000	1, 50	23.03	0.28
Acquisition US x Time interaction for the CS+	0.001	1, 50	12.60	0.19
Acquisition US x Time interaction for the CS-	0.011	1, 50	7.00	0.12
Higher negative Valence to the CS+ in the P-US Acquisition group than to CS+ in the S-US Acquisition group at Post Acquisition	0.001	1, 48	13.94	
Higher CS- than CS+ in the S-US Acquisition group at Post Habituation	0.044	1, 48	4.27	
Higher CS+ than CS- in the P-US Acquisition group at Post Acquisition	0.000	1, 48	32.63	
Time on the CS- in the S-US Acquisition group	0.015	1, 48	1.31	
Time on the CS+ in the P-US Acquisition group	0.000	1, 48	16.36	

Table A10.
Study 1 Acquisition: Arousal Ratings

Test	P	DF	F	η^2
Reinforcement x Acquisition US x Time interaction	0.03	1, 49	9.8	0.08
Reinforcement x Acquisition US interaction	0.005	1, 49	8.51	0.11
Reinforcement x Time	0.000	1, 49	20.33	0.11
Reinforcement x Trial interactions in the Physical Acquisition US group	0.000	1, 27	26.23	0.22
Reinforcement x Acquisition interaction at Post Acquisition	0.000	1, 50	15.60	0.16
Acquisition US x Time interaction for the CS+	0.004	1, 50	9.35	0.14
Higher Arousal to the CS+ in the P-US Acquisition group than to CS+ in the S-US Acquisition group at Post Acquisition	0.006	1, 50	8.26	
Higher CS+ than CS- in the P-US Acquisition group at Post Acquisition	0.000	1, 50	48.89	
Time on CS+ in the P-US Acquisition group	0.004	1, 50	20.26	
Time on CS- in the P-US Acquisition group	0.000	1, 50	9.15	

Table A11.
Study 1 Acquisition: SCR

Test	<i>P</i>	DF	F	η^2
Reinforcement	0.032	1, 38	4.91	0.02
Acquisition US	0.029	1, 40	5.12	0.06
Higher CS+ than CS- in the P-US Acquisition group at Acquisition trials 3 and 4	0.003	1, 40	9.86	
Higher CS+ than CS- in the P-US Acquisition group at Acquisition trials 5 and 6	0.011	1, 40	7.05	

Table A12.
Study 1 Acquisition: ASR

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, simple two-way interaction or simple main effects				

Table A13.
Study 1 Acquisition: PPG

Test	<i>P</i>	DF	F	η^2
No significant three-way interaction				
Reinforcement x Acquisition US interaction	0.000	1, 47	14.23	0.05
Reinforcement x Time interactions in the Physical Acquisition US group	0.009	3, 27	4.08	0.04
Reinforcement x Acquisition interactions at Acquisition trials 1 and 2	0.010	1, 50	7.44	0.12
Reinforcement x Acquisition interactions at Acquisition trials 3 and 4	0.000	1, 50	18.19	0.23
Higher PPG response to the CS+ in the P-US Acquisition group than the CS+ in the S-US Acquisition group at Acquisition trials 1 and 2	0.050	1, 49	3.95	
Higher PPG response to the CS+ in the P-US Acquisition group than the CS+ in the S-US Acquisition group at Acquisition trials 3 and 4	0.009	1, 49	7.31	
Higher PPG response to the CS- in the S-US Acquisition group than the CS- in the P-US Acquisition group at Acquisition trials 5 and 6	0.007	1, 49	37.94	

Table A14.
Study 1 Extinction: P-US Expectancy

Test	<i>P</i>	DF	F	η^2
No significant three-way interaction				
Reinforcement x Acquisition US	0.002	1, 46	10.83	0.06
Acquisition US x Time	0.002	2, 45	6.70	0.04
Time	0.000	1, 46	30.11	0.19
Higher P-US Expectancy to CS+ in the P-US Acquisition group than to CS+ in the S-US Acquisition group at Extinction trials 1 and 2	0.000	1, 46	15.08	
Higher CS+ than CS- in the P-US Acquisition group at Extinction trials 1 and 2	0.003	1, 46	9.66	
Higher CS+ than CS- in the P-US Acquisition group at Extinction trials 3 and 4	0.008	1, 46	7.67	
Higher CS+ than CS- in the P-US Acquisition group at Extinction trials 5 and 6	0.032	1, 46	4.92	
Time on the CS+ in the P-US Acquisition group	0.000	2, 45	30.78	
Time on the CS- in the P-US Acquisition group	0.000	2, 45	11.91	
Time on the CS- in the S-US Acquisition group	0.050	2, 45	3.17	

Table A15.
Study 1 Extinction: S-US Expectancy

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions				
Time	0.000	2, 50	19.88	0.17
Time on the CS+ in the S-US Acquisition group	0.001	2, 49	8.09	
Time on the CS- in the S-US Acquisition group	0.008	2, 45	5.35	
Time on the CS- in the P-US Acquisition group	0.003	2, 45	6.53	

Table A16.
Study 1 Extinction: Valence Ratings

Test	P	DF	F	η^2
Reinforcement x Acquisition US x Time interaction	0.003	1, 50	9.94	0.04
Reinforcement x Acquisition US interaction	0.000	1, 50	16.83	0.13
Acquisition US x Time interaction	0.009	1, 50	7.50	0.02
Reinforcement x Trial interactions in the P-US Acquisition group	0.005	1, 27	9.49	0.05
Reinforcement x Acquisition interactions at Post Acquisition	0.000	1, 50	23.03	0.28
Acquisition US x Time interaction for the CS+	0.001	1, 50	12.59	0.18
Higher Valence to the CS+ in the P-US Acquisition group than to CS+ in the S-US Acquisition group at Post Acquisition	0.001	1, 48	13.94	
Higher CS+ than CS- in the P-US Acquisition group at Post Acquisition	0.000	1, 50	33.9	
Higher CS+ than CS- in the P-US Acquisition group at Post Extinction	0.016	1, 50	6.25	
Time on the CS+ in the P-US Acquisition group	0.000	1, 50	21.88	

Table A17.
Study 1 Extinction: Arousal Ratings

Test	P	DF	F	η^2
Reinforcement x Acquisition US x Time interaction	0.033	1, 50	4.23	0.02
Reinforcement x Acquisition US interaction	0.000	1, 50	15.55	0.08
Reinforcement x Time interaction	0.003	1, 50	4.83	0.08
Reinforcement x Trial interactions in the P-US group	0.003	1, 27	10.48	0.06
Reinforcement x Acquisition interactions at Post Acquisition	0.000	1, 50	15.60	0.16
Reinforcement x Acquisition interactions at Post Extinction	0.050	1, 50	4.05	0.06
Acquisition US x Time interaction for the CS+	0.026	1, 50	5.27	0.07
Higher Arousal to the CS+ in the P-US Acquisition group than to CS+ in the S-US Acquisition group at Post Acquisition	0.010	1, 49	7.21	
Higher CS+ than CS- in the P-US Acquisition group at Post Acquisition	0.000	1, 49	47.9	
Higher CS+ than CS- in the P-US Acquisition group at Post Extinction	0.001	1, 49	13.29	
Time on the CS+ in the P-US Acquisition group	0.000	1, 49	24.90	

Table A18.
Study 1 Extinction: SCR

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions or main effects				
Reinforcement x Trial interactions in the P-US Acquisition group	0.038	1, 23	4.09	
Time on the CS+ in the P-US Acquisition group	0.042	2, 23	3.66	

Table A19.
Study 1 Extinction: ASR

Test	<i>P</i>	DF	F	η^2
No significant three-way interaction				
Reinforcement x Acquisition US	0.001	1, 44	12.62	0.02
Acquisition US x Time	0.007	2, 44	5.26	0.05
Reinforcement x Acquisition interaction at Extinction trials 3 and 4	0.027	2, 44	2.81	0.10
Acquisition US x Time interaction for the CS-	0.014	2, 44	4.45	0.08
Higher ASR to the CS+ in the P-US Acquisition group than the CS+ in the S-US Acquisition group at Extinction trials 1 and 2	0.001	1, 44	11.81	
Higher ASR to the CS- in the P-US Acquisition group than the CS- in the S-US Acquisition group at Extinction trials 1 and 2	0.034	1, 44	4.77	
Higher CS+ than CS- in the P-US Acquisition group at Extinction trials 3 and 4	0.006	1, 44	8.22	
Time on the CS+ in the P-US Acquisition group	0.000	2, 43	13.03	
Time on the CS- in the P-US Acquisition group	0.000	2, 43	16.54	

Table A20.
Study 1 Extinction: PPG

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects				

Table A21.

Study 1 Reinstatement (Reinforcement x Reinstatement US x Time): P-US Expectancy

Test	P	DF	F	η^2
No significant three-way interaction				
Reinstatement US x Time interaction	0.012	1, 46	6.93	0.06
Time	0.000	1, 46	22.13	0.18
Time on CS+ in the P-US Reinstatement group	0.000	1, 46	16.11	
Time on CS- in the P-US Reinstatement group	0.000	1, 46	25.13	

Table A22.

Study 1 Reinstatement (Reinforcement x Group x Time): P-US Expectancy

Test	P	DF	F	η^2
No significant two- or three-way interactions				
Reinforcement x Group	0.087	3, 44	2.34	0.03
Group x Time	0.081	3, 44	2.39	0.06
Time	0.000	1, 44	21.33	0.18
No significant simple two-way interactions				
Higher P-US Expectancy to the CS- in the Social/Social group than to CS- in other groups at Extinction trials 5 and 6	0.015	3, 44	3.89	
Higher CS+ than CS- in the Physical/Social group at Extinction trials 5 and 6	0.047	1, 44	4.17	
Time on the CS+ in the Physical/Physical group	0.009	1, 44	7.41	
Time on the CS+ in the Social/Physical group	0.006	1, 44	8.17	
Time on the CS- in the Physical/Physical group	0.001	1, 44	12.42	
Time on the CS- in the Social/Physical group	0.001	1, 44	12.05	

Table A23.

Study 1 Reinstatement (Reinforcement x Acquisition US x Time): S-US Expectancy

Test	P	DF	F	η^2
No significant two- or three-way interactions				
Acquisition US interaction x Time	0.069	1, 50	3.46	
Time	0.013	1, 50	6.69	0.06
Higher S-US Expectancy to the CS+ in the S-US Acquisition group than to CS+ in the P-US Acquisition group at Post-reinstatement trials 1 and 2	0.049	1, 50	4.06	
Time on the CS+ in the S-US Acquisition group	0.002	1, 50	10.88	

Table A24.

Study 1 Reinstatement (Reinforcement x Reinstatement US x Time): S-US Expectancy

Test	P	DF	F	η^2
No significant two- or three-way interactions				
Reinforcement x Reinstatement US interaction	0.075	1, 50	3.31	
Time	0.014	1, 50	6.47	0.06
Time on CS+ in the Social Pain Reinstatement group	0.008	1, 50	7.62	
Time on CS- in the Social Pain Reinstatement group	0.037	1, 46	4.58	

Table A25.

Study 1 Reinstatement (Reinforcement x Group x Time): S-US Expectancy

Test	P	DF	F	η^2
No significant two- or three-way interactions				
Time	0.011	1, 48	7.04	0.06
Reinforcement x Time interaction in the Social/Social group	0.050	1, 11	4.62	0.03
Reinforcement x Group interaction at Post-reinstatement trials 1 and 2	0.044	3, 48	2.91	0.15
Group x Time interaction for CS+	0.040	3, 48	3.00	0.14
Higher S-US Expectancy to the CS+ in the Social/Social group than to CS+ in the other groups at Post-reinstatement trials 1 and 2	0.033	3, 48	3.16	
Higher CS+ than CS- in the Social/Social group at Post-reinstatement trials 1 and 2	0.012	1, 48	6.78	
Time on the CS+ in the Social/Social group	0.000	1, 48	14.12	

Table A26.

Study 1 Reinstatement (Reinforcement x Acquisition US x Time): Valence Ratings

Test	P	DF	F	η^2
No significant three-way interaction				
Reinforcement x Acquisition US interaction	0.010	1, 48	7.15	0.05
Time	0.025	1, 48	5.35	0.03
Higher Valence ratings to the CS+ than CS- in the P-US Acquisition group at Extinction trials 5 and 6	0.018	1, 48	6.00	
Higher Valence ratings to the CS+ than CS- in the P-US Acquisition group at Post Post-reinstatement trials 1 and 2	0.008	1, 48	7.54	

Table A27.

Study 1 Reinstatement (Reinforcement x Reinstatement US x Time): Valence Ratings

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions				
Reinforcement	0.050	1, 48	4.04	0.04
Time	0.044	1, 48	4.26	0.02
No significant simple two-way interactions				
No simple main effects				

Table A28.

Study 1 Reinstatement (Reinforcement x Group x Time): Valence Ratings

Test	<i>P</i>	DF	F	η^2
No significant three-way interaction				
Reinforcement x Time interaction	0.044	3, 46	2.92	0.07
Time	0.034	1, 46	4.77	0.02
Higher valence ratings to the CS+ than CS- in the Physical/Physical group at Extinction trials 5 and 6	0.048	1, 46	4.14	

Table A29.

Study 1 Reinstatement (Reinforcement x Acquisition US x Time): Arousal Ratings

Test	<i>P</i>	DF	F	η^2
No significant three-way interaction				
Reinforcement x Acquisition US interaction	0.045	1, 48	4.23	0.04
Reinforcement	0.003	1, 48	9.70	0.09
Higher arousal ratings to the CS+ than CS- in the P-US Acquisition group at Extinction trials 5 and 6	0.001	1, 48	13.02	
Higher arousal ratings to the CS+ than CS- in the P-US Acquisition group at Post Post-reinstatement trials 1 and 2	0.001	1, 48	11.35	

Table A30.

Study 1 Reinstatement (Reinforcement x Reinstatement US x Time): Arousal Ratings

Test	P	DF	F	η^2
No significant two- or three-way interactions				
Reinforcement	0.003	1, 48	9.74	0.10
Higher valence ratings to the CS+ than CS- in the Physical Pain Reinstatement group at Extinction trials 5 and 6	0.009	1, 48	7.43	
Higher valence ratings to the CS+ than CS- in the Physical Pain Reinstatement group at Post-reinstatement trials 1 and 2	0.001	1, 48	11.35	
Time on the CS- in the Physical Pain Reinstatement group	0.042	1, 48	4.37	

Table A31.

Study 1 Reinstatement (Ratings Reinforcement x Group x Time): Arousal

Test	P	DF	F	η^2
No significant two- or three-way interactions				
Reinforcement	0.005	1, 46	8.58	0.08
Higher arousal ratings to the CS+ than CS- in the Physical/Physical group at Extinction trials 5 and 6	0.002	1, 46	10.33	
Higher arousal ratings to the CS+ than CS- in the Physical/Physical group at Post-reinstatement trials 1 and 2	0.001	1, 46	12.17	

Table A32.

Study 1 Reinstatement (Reinforcement x Acquisition US x Time): SCR

Test	P	DF	F	η^2
No significant two- or three-way interactions main effects, simple two-way interactions or simple main effects				

Table A33.

Study 1 Reinstatement (Reinforcement x Reinstatement US x Time): SCR

Test	P	DF	F	η^2
No significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects				

Table A34.

Study 1 Reinstatement (Reinforcement x Group x Time): SCR

Test	<i>P</i>	DF	F	η^2
No significant three-way interactions				
Reinforcement x Group	0.047	3, 41	2.89	0.03
No significant main effects				
No significant simple two-way interactions				
Reinforcement by Time interaction in the Physical Pain Reinstatement group	0.055	1, 22	4.10	0.02
Higher SCR to CS- than CS+ in the Physical/Social group at Post-reinstatement trials 1 and 2	0.023	1, 41	5.57	

Table A35.

Study 1 Reinstatement (Reinforcement x Acquisition US x Time): ASR

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions				
Time	0.002	1, 44	10.32	0.08
No significant simple two-way interactions or simple main effects				

Table A36.

Study 1 Reinstatement (Reinforcement x Reinstatement US x Time): ASR

Test	<i>P</i>	DF	F	η^2
No significant three-way interactions				
Reinstatement US x Time interaction	0.024	1, 44	5.47	0.04
Higher ASR to CS+ in the Physical Pain Reinstatement group than CS+ in the Social Pain Reinstatement group at Post-reinstatement trials 1 and 2	0.012	1, 44	6.94	
Higher ASR to CS- in the Physical Pain Reinstatement group than CS- in the Social Pain Reinstatement group at Post-reinstatement trials 1 and 2	0.035	1, 44	4.76	
Time on the CS+ in the Physical Pain Reinstatement group	0.033	1, 44	4.86	
Time on the CS- in the Physical Pain Reinstatement group	0.000	1, 44	17.29	

Table A37.

Study 1 Reinstatement (Reinforcement x Group x Time): ASR

Test	<i>P</i>	DF	F	η^2
No significant three-way interactions				
Group x Time interaction	0.017	1, 42	3.80	0.08
Group	0.002	3, 42	5.71	0.15
Time	0.001	1, 42	13.16	0.09
No significant simple two-way interactions				
Higher ASR to CS+ in the Social/Physical group than CS+ in the other groups	0.012	3, 42	4.14	
Higher ASR to CS- in the Social/Physical group than CS- in the other groups	0.009	3, 42	4.43	
Higher CS+ than CS- in the Physical/Physical group at Extinction trials 5 and 6	0.24	3, 42	4.43	
Time on the CS+ in the Social/Physical group	0.003	1, 42	10.02	
Time on the CS- in the Social Physical group	0.016	1, 42	12.07	
Time on the CS- in the Physical/Physical Group	0.001	1, 42	6.33	

Table A38.

Study 1 Reinstatement (Reinforcement x Acquisition US x Time): PPG

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, or simple two-way interactions				
Time on the CS- in the S-US Acquisition group	0.010	1, 49	7.22	

Table A39.

Study 1 Reinstatement (Reinforcement x Reinstatement US x Time): PPG

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, simple two-way interactions, or simple main effects				

Table A40.

Study 1 Reinstatement (Reinforcement x Group x Time): PPG

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, or simple two-way interactions				
Time on the CS- in the Social/Physical group	0.024	1, 47	5.45	
Higher PPG response to CS+ than CS- in the Social/Physical group at Post-reinstatement trials 1 and 2	0.065	1, 47	3.56	

Table A41.

Study 1 Reinstatement (Reinforcement x Match x Time): P-US Expectancy

Test	P	DF	F	η^2
Time	0.000	1, 46	22.13	0.18
Time on the CS+ in the Same group	0.021	1, 46	5.74	
Time on the CS+ in the Different group	0.011	1, 46	7.03	
Time on the CS- in the Same group	0.010	1, 46	7.23	
Time on the CS- in the Different group	0.002	1, 46	10.73	

Table A42.

Study 1 Reinstatement (Reinforcement x Match x Time): S-US Expectancy

Test	P	DF	F	η^2
Time	0.014	1, 50	6.47	0.06
Time on the CS+ in the Same group	0.020	1, 50	5.79	

Table A43.

Study 1 Reinstatement (Reinforcement x Match x Time): Valence Ratings

Test	P	DF	F	η^2
No significant interactions or main effects				

Table A44.

Study 1 Reinstatement (Reinforcement x Match x Time): Arousal Ratings

Test	P	DF	F	η^2
No significant interactions or main effects				

Table A45.

Study 1 Reinstatement (Reinforcement x Match x Time): SCR

Test	P	DF	F	η^2
Reinforcement by Match interaction	0.050	1, 43	6.47	0.04

Table A46.

Study 1 Reinstatement (Reinforcement x Match x Time): ASR

Test	P	DF	F	η^2
No significant interactions or main effects				

Table A47.

Study 1 Reinstatement (Reinforcement x Match x Time): PPG

Test	<i>P</i>	DF	F	η^2
No significant interactions or main effects				

APPENDIX 3: STUDY 2 STATISTICAL OUTPUT

Table A48.

Study 2 Acquisition: P-US Expectancy

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, or simple main effects				

Table A49.

Study 2 Acquisition: S-US Expectancy

Test	<i>P</i>	DF	F	η^2
Reinforcement x Time interaction	0.038	3, 17	7.26	0.04
Reinforcement	0.029	1, 19	5.57	0.07
Higher CS+ than CS- at Acquisition trials 3 and 4	0.027	1, 19	5.77	
Higher CS+ than CS- at Acquisition trials 5 and 6	0.049	1, 19	4.44	

Table A50.

Study 2 Acquisition: Valence Ratings

Test	<i>P</i>	DF	F	η^2
No significant two-way interaction				
Reinforcement	0.001	1, 19	16.15	0.29
Higher CS+ than CS- at Post-Habituation	0.002	1, 19	13.62	
Higher CS+ than CS- at Post-Acquisition	0.010	1, 19	8.40	

Table A51.

Study 2 Acquisition: Arousal Ratings

Test	<i>P</i>	DF	F	η^2
Reinforcement x Time interaction	0.002	1, 19	13.28	0.09
Reinforcement	0.017	1, 19	6.93	0.13
Higher CS+ than CS- at Post Acquisition	0.003	1, 19	11.57	
Time on CS+	0.008	1, 19	8.95	

Table A52.

Study 2 Acquisition: SCR

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, or simple main effects				

Table A53.
Study 2 Acquisition: PPG

Test	<i>P</i>	DF	F	η^2
No significant two-way interaction				
Reinforcement	0.034	1, 19	5.28	0.06
Time	0.015	1, 19	3.84	0.07
Higher CS+ than CS- at Acquisition trials 1 and 2	0.033	1, 19	5.33	
Time on CS-	0.017	1, 19	4.58	

Table A54.
Study 2 Acquisition: ASR

Test	<i>P</i>	DF	F	η^2
No significant two-way interaction				
Time	0.005	3, 18	6.84	
Time on CS+	0.006	3, 18	6.63	
Time on CS-	0.000	3, 18	15.30	

Table A55.
Study 2 Extinction: P-US Expectancy

Test	<i>P</i>	DF	F	η^2
No significant two-way interaction				
Time	0.011	1, 20	3.87	0.19
Higher CS- than CS+ at Extinction trials 1 and 2	0.040	1, 19	5.02	
Time on CS-	0.033	1, 19	4.32	

Table A56.
Study 2 Extinction: S-US Expectancy

Test	<i>P</i>	DF	F	η^2
Reinforcement x Time interaction	0.017	3, 18	4.5	0.06
No significant main effects				
Higher CS+ than CS- at Extinction trials 1 and 2	0.050	1, 19	4.27	
Time on CS+	0.039	2, 18	3.91	

Table A57.
Study 2 Extinction: Valence Ratings

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions				
Reinforcement	0.008	1, 18	8.96	0.26
Higher CS+ than CS- at Post Acquisition	0.050	1, 19	8.39	
Higher CS+ than CS- at Post Extinction	0.010	1, 19	6.96	

Table A58.
Study 2 Extinction: Arousal Ratings

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions				
Reinforcement	0.001	1, 18	14.87	0.23
Time	0.000	1, 18	26.66	0.20
Higher CS+ than CS- at Post Acquisition	0.003	1, 19	11.56	
Higher CS+ than CS- at Post Extinction	0.005	1, 19	10.21	
Time on CS+	0.001	1, 19	16.48	
Time on CS-	0.003	1, 19	12.31	

Table A59.
Study 2 Extinction: PPG

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, or simple main effects				

Table A60.
Study 2 Extinction: SCR

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, or simple main effects				

Table A61.
Study 2 Extinction: ASR

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, or simple main effects				

Table A62.
Study 2 Reinstatement: P-US Expectancy

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions or main effects				
Time	0.056			
Time on CS- in the Physical Pain US Reinstatement group	0.032	1, 18	5.42	

Table A63.
Study 2 Reinstatement: S-US Expectancy

Test	P	DF	F	η^2
No significant three-way interaction (marginally significant)	0.084			
Reinforcement x Time	0.004	1, 18	10.65	0.06
Reinstatement US x Time	0.040	1, 18	4.93	0.12
Reinforcement x Reinstatement US interactions	0.041	3, 18	4.85	0.04
Reinforcement x Trial interactions in the Social Reinstatement US group	0.014	1, 10	9.26	0.08
Reinforcement x Reinstatement US interactions at reinstatement trials 1 and 2	0.041	1, 18	4.83	0.17
Reinstatement US x Trial interactions for CS+	0.026	1, 18	5.88	0.21
Higher CS+ than CS- in the S-US Reinstatement group at Reinstatement trials 1 and 2	0.004	1, 18	10.93	
Time on CS+ in the S-US Reinstatement group	0.005	1, 18	10.47	

Table A64.
Study 2 Reinstatement: Valence Ratings

Test	P	DF	F	η^2
Reinforcement x Reinstatement US x Time interaction	0.050	1, 17	4.28	0.05
No significant two-way interactions				
Reinforcement x Time interaction in the S-US Reinstatement group	0.042	1, 19	5.60	0.12
Reinforcement by Reinstatement US interaction at Post Reinstatement	0.004	1, 19	11.06	0.23
Reinforcement	0.005	1, 17	10.61	0.16
Reinstatement US	0.040	1, 17	4.95	0.07
Time	0.006	1, 17	9.71	0.10
Higher CS- at Post Extinction in the P-US Reinstatement than the S-US Reinstatement group	0.050	1, 18	4.45	
Higher CS+ at Post Reinstatement in the Physical Pain US Reinstatement group	0.002	1, 18	12.87	
Higher CS+ than CS- in the S-US Reinstatement group at Post Extinction	0.048	1, 18	4.54	
Higher CS+ than CS- in the Physical Pain US Reinstatement group at Post Reinstatement	0.001	1, 18	15.31	
Time on CS+ in the S-US Reinstatement group	0.012	1, 18	7.93	
Time on CS- in the Physical Pain US Reinstatement group	0.015	1, 18	7.33	

Table A65.
Study 2 Reinstatement: Arousal Ratings

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions				
Reinforcement	0.010	1, 18	8.35	0.16
Reinstatement US	0.020	1, 18	6.54	0.09
Reinforcement x Reinstatement US interaction at Extinction trials 5 and 6	0.009	1, 19	8.55	0.20
Higher CS+ than CS- at Extinction trials 5 and 6 in the Physical Pain US Reinstatement group	0.002	1, 18	14.02	
Higher CS+ than CS- in the Physical Pain US Reinstatement group at Post Extinction	0.000	1, 18	22.48	

Table A66.
Study 2 Reinstatement: ASR

Test	<i>P</i>	DF	F	η^2
No significant three-way interactions				
Reinforcement x Time interaction	0.030	1, 15	84.68	0.05
Reinforcement x Time interaction in the S-US Reinstatement group	0.040	1, 9	5.78	0.13
Reinstatement US x Time interaction for the CSM	0.033	1, 15	5.78	0.27
Time on CS+ in the Physical Pain US group	0.044	1, 15	4.83	

Table A67.
Study 2 Reinstatement: SCR

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, or simple main effects				

Table A68.
Study 2 Reinstatement: PPG

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, or simple main effects				

Table A69.
Comparing Acquisition in Study 1 and Study 2: P-US Expectancy

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects or simple two-way interactions involving study				
Higher CS- than CS+ in Study 1 at Acquisition trials 1 and 2	0.049	1, 40	4.14	
Higher CS- than CS+ in Study 1 at Acquisition trials 5 and 6	0.040	1, 40	4.53	

Table A70.

Comparing Acquisition in Study 1 and Study 2: S-US Expectancy

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A71.

Comparing Acquisition in Study 1 and Study 2: Valence Ratings

Test	<i>P</i>	DF	F	η^2
No significant three-way interaction				
Reinforcement x Study interaction	0.005	1, 38	8.94	0.08
Time x Study interaction	0.053			
Reinforcement x Time interaction in Study 1	0.009	1, 21	8.40	0.14
Higher CS+ in Study 2 than Study 1 at Post-Habituation	0.031	1, 38	5.01	
Higher CS+ in Study 2 than Study 1 at Post-Acquisition	0.030	1, 38	5.12	
Higher CS+ than CS- at Post-Habituation in Study 1	0.035	1, 38	4.76	
Higher CS+ than CS- at Post-Habituation in Study 2	0.000	1, 38	16.33	
Higher CS+ than CS- at Post-Acquisition in Study 2	0.004	1, 38	9.30	
Time on CS- in Study 1	0.001	1, 38	13.06	

Table A72.

Comparing Acquisition in Study 1 and Study 2: Arousal Ratings

Test	<i>P</i>	DF	F	η^2
Reinforcement x Time x Study interaction	0.010	1, 40	7.35	0.02
No significant two-way interactions				
Reinforcement x Time interaction in Study 2	0.002	1, 19	13.28	0.09
Higher CS+ at Post-Acquisition in Study 2 than Study 1	0.013	1, 40	6.71	
Higher CS+ than CS- in Study 2 at Post Acquisition	0.000	1, 40	19.74	
Time on CS+ in Study 2	0.001	1, 40	11.64	

Table A73.

Comparing Acquisition in Study 1 and Study 2: SCR

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A74.
Comparing Acquisition in Study 1 and Study 2: ASR

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A75.
Comparing Acquisition in Study 1 and Study 2: PPG

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interaction				
Marginally significant Reinforcement x Study	0.067			
Marginally significant Time x Study	0.065			
Study	0.043	1, 40	4.39	0.11
Higher CS- in Study 1 than Study 2 at Acquisition trials 1 and 2	0.011	1, 40	7.03	
Higher CS- in Study 1 than Study 2 at Acquisition trials 3 and 4	0.018	1, 40	6.09	
Higher CS+ than CS- in Study 2 at Acquisition trials 1 and 2	0.009	1, 40	7.50	
Time on CS- in Study 2	0.004	3, 38	5.26	

Table A76.
Comparing Extinction in Study 1 and Study 2: P-US Expectancy

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A77.
Comparing Extinction in Study 1 and Study 2: S-US Expectancy

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A78.

Comparing Extinction in Study 1 and Study 2: Valence Ratings

Test	<i>P</i>	DF	F	η^2
No significant three-way interaction				
Reinforcement x Study interaction	0.002	1, 40	10.68	0.05
No significant two-way interactions				
Higher CS+ in Study 2 than Study 1 at Extinction trials 1 and 2	0.013	1, 40	9.73	
Higher CS+ than CS- at Extinction trials 1 and 2	0.003	1, 40	6.70	
Higher CS+ than CS- at Extinction trials 3 and 4	0.005	1, 40	8.81	

Table A79.

Comparing Extinction in Study 1 and Study 2: Arousal Ratings

Test	<i>P</i>	DF	F	η^2
No significant three-way interaction				
Reinforcement x Study	0.013	1, 39	6.80	0.06
Time x Study interactions	0.008	1, 39	7.80	0.03
No significant simple two-way interactions				
Higher CS+ in Study 2 than Study 1 at Extinction trials 1 and 2	0.020	1, 39	5.85	
Higher CS+ than CS- at Extinction trials 1 and 2	0.000	1, 39	19.28	
Higher CS+ than CS- at Extinction trials 3 and 4	0.003	1, 39	9.71	
Time on CS+ in Study 1	0.029	1, 39	5.15	
Time on CS+ in Study 2	0.000	1, 39	27.51	
Time on CS- in Study 2	0.001	1, 39	12.09	

Table A80.

Comparing Extinction in Study 1 and Study 2: SCR

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A81.

Comparing Extinction in Study 1 and Study 2: ASR

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A82.
Comparing Extinction in Study 1 and Study 2: PPG

Test	<i>P</i>	DF	F	η^2
No significant two-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A83.
Comparing Reinstatement in Study 1 and 2: P-US Expectancy in P-US Reinstatement

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, or simple two-way effects				
Higher CS+ in Study 2 than Study 1 at Extinction trials 5 and 6	0.024	1, 19	5.98	
Higher CS+ than CS- in Study 2 at Extinction trials 5 and 6	0.044	1, 19	4.62	
Time on CS+ in Study 1	0.016	1, 19	6.70	
Time on CS- in Study 1	0.099	1, 19	8.47	

Table A84.
Comparing Reinstatement in Study 1 and 2: P-US Expectancy in S-US Reinstatement

Test	<i>P</i>	DF	F	η^2
There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A85.
Comparing Reinstatement in Study 1 and 2: S-US Expectancy in P-US Reinstatement

Test	<i>P</i>	DF	F	η^2
There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A86.
Comparing Reinstatement in Study 1 and 2: S-US Expectancy in S-US Reinstatement

Test	<i>P</i>	DF	F	η^2
There were no significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A87.

Comparing Reinstatement in Study 1 and 2: Valence Ratings in P-US Reinstatement

Test	P	DF	F	η^2
No significant three-way interactions				
Reinforcement x Study interaction	0.002	1, 18	12.45	0.19
No simple two-way interactions				
Higher CS+ in Study 2 than Study 1 at Reinstatement trials 1 and 2	0.022	1, 18	6.29	
Time on CS- in Study 2	0.014	1, 18	7.45	

Table A88.

Comparing Reinstatement in Study 1 and 2: Valence Ratings in S-US Reinstatement

Test	P	DF	F	η^2
No significant two- or three-way interactions, main effects, or simple two-way interactions involving Study				
Higher CS+ than CS- at Extinction trials 5 and 6 in Study 2	0.041	1, 18	4.83	
Time on CS+ in Study 2	0.007	1, 18	9.08	

Table A89.

Comparing Reinstatement in Study 1 and 2: Arousal Ratings in P-US Reinstatement

Test	P	DF	F	η^2
Reinforcement x Time x Study interaction	0.018	1, 18	6.74	0.04
No significant two-way interactions				
Higher CS+ in Study 2 than Study 1 at Post-Extinction	0.042	1, 18	4.77	
Higher CS+ than CS- in Study 1 at Post-Extinction	0.002	1, 18	12.63	
Time on CS+ in Study 2	0.002	1, 18	8.49	

Table A90.

Comparing Reinstatement in Study 1 and 2: Valence Ratings in S-US Reinstatement

Test	P	DF	F	η^2
No significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A91.

Comparing Reinstatement in Study 1 and 2: SCR in P-US Reinstatement

Test	P	DF	F	η^2
No significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A92.

Comparing Reinstatement in Study 1 and 2: SCR in S-US Reinstatement

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A93.

Comparing Reinstatement in Study 1 and 2: ASR in P-US Reinstatement

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects or simple two-way interactions involving Study				
Time on CS+ Study 1	0.012	1, 15	5.47	
Time on CS- Study 1	0.011	1, 15	8.54	
Time on CS+ in Study 2	0.041	1, 15	5.02	

Table A94.

Comparing Reinstatement in Study 1 and 2: ASR in P-US Reinstatement

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects or simple two-way interactions involving Study				
Time on CS- in Study 2	0.029	1, 21	5.47	

Table A95.

Comparing Reinstatement in Study 1 and 2: PPG in P-US Reinstatement

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A96.

Comparing Reinstatement in Study 1 and 2: PPG in S-US Reinstatement

Test	<i>P</i>	DF	F	η^2
No significant two- or three-way interactions, main effects, simple two-way interactions or simple main effects involving Study				

Table A97.

Predictors of P-US Expectancy Reinstatement Change

Outcome Variable	Significant Predictor	Significance	F	DF	Unique R² Change	Standardized Beta
CS+		Not significant				
CS-		Not significant				
CS Differential	SCR during reinstatement	0.028	5.23	1,38	0.106	-0.370
Demographics and baseline measures that were entered into the first level of the linear regression model included: age, gender, ethnicity, BDI, ASI, and Mini-SPIN						

Table A98.

Predictors of S-US Expectancy Reinstatement Change

Outcome Variable	Significant Predictor	Significance	F	DF	Unique R² Change	Standardized Beta
CS+	PPG percent change	0.043	4.36	1,35	0.105	-0.344
CS-		Not significant				
CS Differential		Not significant				
Demographics and baseline measures that were entered into the first level of the linear regression model included: age, gender, ethnicity, BDI, ASI, and Mini-SPIN						

Table A99.

Predictors of Valence Ratings Reinstatement Change

Outcome Variable	Significant Predictor	Significance	F	DF	Unique R² Change	Standardized Beta
CS+	Muscle stimulation level	.001	12.19	1,38	0.219	0.496
CS-		Not significant				
CS Differential	Muscle stimulation level	.014	6.58	1,38	0.131	0.383
Demographics and baseline measures that were entered into the first level of the linear regression model included: age, gender, ethnicity, BDI, ASI, and Mini-SPIN						

Table A100.
Predictors of Arousal Ratings Reinstatement Change

Outcome Variable	Significant Predictor	Significance	F	DF	Unique R² Change	Standardized Beta
CS+		Not significant				
CS-		Not significant				
CS Differential		Not significant				
Demographics and baseline measures that were entered into the first level of the linear regression model included: age, gender, ethnicity, BDI, ASI, and Mini-SPIN						

Table A101.
Predictors of SCR Reinstatement Change

Outcome Variable	Significant Predictor	Significance	F	DF	Unique R² Change	Standardized Beta
CS+	SCR during reinstatement	.044	5.01	1,41	0.078	0.317
CS-	SCR during reinstatement	0.041	4.44	1,41	0.084	0.330
CS Differential		Not significant				
Demographics and baseline measures that were entered into the first level of the linear regression model included: age, gender, ethnicity, BDI, ASI, and Mini-SPIN						

Table A102.
Predictors of ASR Reinstatement Change

Outcome Variable	Significant Predictor	Significance	F	DF	Unique R² Change	Standardized Beta
CS+		Not significant				
CS-		Not significant				
CS Differential		Not significant				
Demographics and baseline measures that were entered into the first level of the linear regression model included: age, gender, ethnicity, BDI, ASI, and Mini-SPIN						

Table A103.
Predictors of PPG Reinstatement Change

Outcome Variable	Significant Predictor	Significance	F	DF	Unique R² Change	Standardized Beta
CS+		Not significant				
CS-	PPG during reinstatement	0.027	5.27	1,41	0.079	0.287
CS Differential		Not significant				
Demographics and baseline measures that were entered into the first level of the linear regression model included: age, gender, ethnicity, BDI, ASI, and Mini-SPIN						

REFERENCES

- Baum M (1988). Spontaneous recovery from the effects of flooding (exposure) in animals. *Behaviour Research and Therapy*, 26(2), 185-186.
- Baeyens F, Crombez G, Van den Bergh O, Eelen P (1988). Once in contact always in contact: Evaluative conditioning is resistant to extinction. *Advances in Behaviour Research and Therapy*, 10, 179–199.
- Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4: 561-71.
- Bentz D, Michael T, de Quervain DJF, Wilhelm FH (2010). Enhancing exposure therapy for anxiety disorders with glucocorticoids: From basic mechanisms of emotional learning to clinical applications. *Journal of Anxiety Disorders*, 24, 223-230.
- Biondi M, Picardi A (1999). Psychological stress and neuroendocrine function in humans: The last two decades of research. *Psychotherapy & Psychosomatics*, 68, 114–150.
- Blaisdell A (2009). The role of associative processes in spatial, temporal, and causal cognition. In S. Wantabe, A. Blaisdell, L. Huber, & A. Young.(Eds.), *Rational animals, irrational humans* (153-172). Tokyo, Japan, *Keio University Press*.
- Blazer D, Hughes D, George LK (1987). Stressful life events and the onset of a generalized anxiety syndrome. *The American Journal of Psychiatry*, 144, 1178-1183.
- Boschen MJ, Neumann DL, Waters AM (2009). Relapse of successfully treated anxiety and fear: Theoretical issues and recommendations for clinical practice. *Australian and New Zealand Journal of Psychiatry*, 43, 89-100.
- Bolles RCF, Fanselow MS (1980) A perceptual-defensive-recuperative model of fear and pain. *Behavioral and Brain Sciences*, 3, 291–301.

- Bouton ME (1984). Differential control by context in the inflation and reinstatement paradigms. *Journal of Experimental Psychology: Animal Behavior Processes*, 10, 56–74.
- Bouton ME (1991). Context and retrieval in extinction and in other examples of interference in simple associative learning. In *Current topics in animal learning: Brain, emotion, and cognition* (eds. L. Dachowski and C.F. Flaherty), pp. 25–53. Erlbaum, Hillsdale, NJ.
- Bouton ME (1993). Context, time and memory retrieval in the interference paradigms of Pavlovian learning. *Psychological Bulletin*, 114, 90-99.
- Bouton ME, Bolles RC (1979). Role of conditioned contextual stimuli in reinstatement of extinguished fear. *Journal of Experimental Psychology: Animal Behavior Processes*, 5, 368-378.
- Bouton ME, King DA (1983). Contextual control of the extinction of conditioned fear: Tests for the associative value of the context. *Journal of Experimental Psychology: Animal Behavior Processes*, 9, 248-265.
- Bouton ME, Peck CA (1989). Context effects on conditioning, extinction, and reinstatement in an appetitive conditioning preparation. *Animal Learning & Behavior*, 17, 188-198.
- Bouton ME, Mineka S, Barlow DH (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review*, 108, 4-32.
- Bouton ME, Westbrook RF, Corcoran KA, Maren S (2006). Contextual and Temporal Modulation of Extinction: Behavioral and Biological Mechanisms. *Biological Psychiatry*, 60, 352-360.
- Brook CA, Schmidt LA (2008). Social anxiety disorder: A review of environmental risk factors. *Neuropsychiatric Disease and Treatment*, 4, 123-143.
- Brown KW, Ryan RM (2003). The benefits of being present: Mindfulness and its role in

- psychological well being. *Journal of Personality and Social Psychology*, 84, 822-848.
- Connor KM, Davidson JR, Churchill LE, Sherwood A, Foa E, Weisler RH (2000). Psychometric properties of the Social Phobia Inventory (SPIN): New self-rating scale. *British Journal of Psychiatry*, 176, 379-386.
- Connor KM, Kobak KA, Churchill LE, Katzelnick D, & Davidson JR (2001). Mini-SPIN: a brief screening assessment for generalized social anxiety disorder. *Depression and Anxiety*, 14, 137-140.
- Cook M, Mineka S (1989): Observational conditioning of fear to fear-relevant versus fear-irrelevant stimuli in rhesus monkeys. *Journal of Abnormal Psychology* 98:448–459.
- Cook M, Mineka S (1990): Selective associations in the observational conditioning of fear in rhesus monkeys. *Journal of Experimental Psychology: Animal Behavior Processes* 16:372–389.
- Cooper R, Gerlach AL (2013). Measurement of the blush. In W.R. Crozier, P.J. de Jong (Eds.). *The Psychological Significance of the Blush* (pp. 39-59). New York, NY, US: Cambridge University Press.
- Craske MG, Mystkowski JL (2006). Exposure Therapy and Extinction: Clinical Studies. In M. G. Craske, D. Hermans, D. Vansteenwegen (Eds.). *Fear and learning: From basic processes to clinical implications* (pp. 217-233). Washington, DC, US: American Psychological Association.
- Craske MG, Rachman SJ (1987). Return of fear: Perceived skill and heart-rate responsivity. *British Journal of Clinical Psychology*, 26, 187-199.
- Davis M (1989). Sensitization of the acoustic startle reflex by footshock. *Behavioral Neuroscience*, 103, 495–503.

- De Houwer J, Thomas S, Baeyens F (2001). Associative learning of likes and dislikes: A review of 25 years of research on human evaluative conditioning. *Psychological Bulletin*, 127, 853–869.
- Delamater AR (1997). Selective reinstatement of stimulus–outcome associations. *Animal Learning and Behavior*, 25, 400-412.
- Dimsdale JE, Moss J (1980). Plasma catecholamines in stress and exercise. *Journal of the American Medical Association (JAMA)*, 243, 340-342.
- Dirikx T, Hermans D, Vansteenwegen D, Baeyens F, Eelen P (2004). Reinstatement of extinguished conditioned responses and negative stimulus valence as a pathway to return of fear in humans. *Learning & Memory*, 11, 549-554.
- Dirikx T, Hermans D, Vansteenwegen D, Baeyens F, Eelen, P (2007). Reinstatement of conditioned responses in human differential fear conditioning. *Journal of Behavior Therapy and Experimental Psychiatry*, 38, 237-251.
- Dirikx T, Vansteenwegen D, Eelen P, Hermans D (2009). Non-differential return of fear in humans after a reinstatement procedure. *Acta Psychologica*, 130, 175-182.
- Eisenberger NI (2011). Why rejection hurts: What social neuroscience has revealed about the brain’s response to social rejection. To appear in: J. Decety & J. Cacioppo (Eds.). *The Handbook of Social Neuroscience* (p. 586-598). New York, NY: Oxford University Press.
- Eisenberger NI (2012) Broken hearts and broken bones: A neural perspective on the similarities between social and physical pain. *Current Directions in Psychological Science*, 21, 42-47.
- Fanselow MS (1980). Conditioned and unconditional components of postshock freezing.

- Pavlovian Journal of Biological Science*, 15, 177–182.
- Frohardt RJ, Guarraci F, Bouton ME (2000). The effects of neurotoxic hippocampal lesions on two effects of context after extinction. *Behavioral Neuroscience*, 114, 227–240.
- Garcia, J., & Koelling, R. A. (1966). Relation of cue to consequence in avoidance learning. *Psychonomic Science*, 4, 123-124.
- Glenn DE, Minor TR, Vervliet B, Craske MG (2014). The effect of glucose on hippocampal-dependent contextual fear conditioning. *Biological Psychiatry*, 75, 847-854).
- Gothelf D, Aharonovsky O, Horesh N, Carty T, Apter A (2004). Life Events and Personality Factors in Children and Adolescents With Obsessive-Compulsive Disorder and Other Anxiety Disorders. *Comprehensive Psychiatry*, 45, 192-198.
- Grey S, Sartory G, Rachman SJ (1979). Synchronous and desynchronous changes during fear reduction. *Behaviour Research and Therapy*, 17, 137-147.
- Grillon, C (2002). Startle reactivity and anxiety disorders: aversive conditioning, context, and neurobiology. *Biological Psychiatry*, 15, 958-975.
- Grillon C, Morgan III CA (1999). Fear-potentiated startle conditioning to explicit and contextual cues in Gulf War veterans with posttraumatic stress disorder. *Journal of Abnormal Psychology*, 108, 134-142.
- Haroutunian V, Riccio DC (1977). Effect of arousal conditions during reinstatement treatment upon learned fear in young rats. *Developmental Psychobiology*, 10, 25-32.
- Haroutunian V, Riccio DC (1979). Drug-induced "arousal" and the effectiveness of CS exposure in the reinstatement of memory. *Behavioral & Neural Biology*, 26, 115-120.
- Herman BH, Panksepp J (1978). Effects of morphine and naloxone on separation distress and approach attachment: Evidence for opiate mediation of social affect. *Pharmacology*,

- Biochemistry and Behavior*, 9, 213–220.
- Hermans D, Craske MG, Mineka S, Lovibond PF (2006). Extinction in Human Fear Conditioning. *Biological Psychiatry*, 60, 361-368.
- Hermans D, Crombez G, Vansteenwegen D, Baeyens F, Eelen P (2002). Expectancy-learning and evaluative learning in human classical conditioning: Differential effects of extinction. In *Advances in psychology research*, Vol. 12, (ed. S.P. Shohov), pp.17–41. Nova Science Publishers, Inc., New York.
- Hermans D, Dirikx T, Vansteenwegen D, Baeyens F, Van den Bergh O, Eelen P (2005). Reinstatement of fear responses in human aversive conditioning. *Behaviour Research and Therapy*, 43, 533-551.
- Jackson ED, Payne JD, Nadel L, Jacobs WJ (2006). Stress differentially modulate fear conditioning in healthy men and women. *Biological Psychiatry*, 59, 516-522.
- Jacobs WJ, Nadel L (1985). Stress-induced recovery of fears and phobias. *Psychological Review*, 92, 512-531.
- Johnson EO, Kamilaris TC, Chrousos GP, Gold PW (1992). Mechanisms of stress: A dynamic overview of hormonal and behavioral homeostasis. *Neuroscience and Biobehavioral Reviews*, 16, 115-130.
- Kehoe EJ, Macrae M (1997). Savings in animal learning: Implications for relapse and maintenance after therapy. *Behavior Therapy*, 28, 141-155.
- Kirschbaum C, Pirke KM, Hellhammer DH (1993). The 'Trier Social Stress Test'--a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28, 76-81.
- Klauke B, Deckert J, Reif A, Pauli P, Domschke K (2010). Life events in panic disorder—An

- update on "candidate stressors." *Depression and Anxiety*, 27, 716-730.
- Kull S, Müller BH, Blechert J, Wilhelm FH, Michael T (2012). Reinstatement of fear in humans: Autonomic and experiential responses in a differential conditioning paradigm. *Acta Psychologica*, 140: 43-49.
- LaBar KS, Phelps EA (2005). Reinstatement of conditioned fear in humans is context dependent and impaired in amnesia. *Behavioral Neuroscience*, 119, 677–686.
- Lang PJ, Bradley MM, Cuthbert BN (1990). Emotion, attention, and the startle reflex. *Psychological Review*, 97, 377-395.
- Lau JY, Lissek S, Nelson EE, Lee Y, Roberson-Nay R, Poeth K, Jenness J, Ernst M, Grillon C, Pine DS (2008). Fear conditioning in adolescents with anxiety disorders: Results from a novel experimental paradigm. *Journal of the American Academy of Child & Adolescent Psychiatry*, 47, 94-102.
- Lipp OV (2006). Human Fear Learning: Contemporary Procedures and Measurement. In M. G. Craske, D. Hermans, D. Vansteenwegen (Eds.), *Fear and learning: From basic processes to clinical implications* (pp. 37-51). Washington, DC, US: American Psychological Association.
- Lipp OV, Sheridan J, Siddle DA (1994). Human blink startle during aversive and nonaversive Pavlovian conditioning. *Journal of Experimental Psychology: Animal Behavior Processes*, 20, 380-389.
- Lissek S, Levenson J, Biggs, A. L, Johnson, L. L, Ameli, R, Pine, D. S, Grillon, C. (2008). Elevated fear conditioning to socially relevant unconditioned stimuli in social anxiety disorder. *The American Journal of Psychiatry*, 165, 124-132.
- Lovibond PF, Davis NR, O’Flaherty AS (2000). Protection from extinction in human fear

- conditioning. *Behaviour Research and Therapy*, 38, 967-983.
- MacDonald G, Leary MR (2005). Why does social exclusion hurt? The relationship between social and physical pain. *Psychological Bulletin*, 131, 202-223.
- Miller RR, Barnet RC, Grahame NJ (1995). Assessment of the Rescorla-Wagner model. *Psychological Bulletin*, 117, 363-386.
- Mineka S, Öhman A (2002). Phobias and preparedness: The selective, automatic, and encapsulated nature of fear. *Biological Psychiatry*, 52, 927-937.
- Mineka S, Zinbarg R (2006). A contemporary learning theory perspective on the etiology of anxiety disorders: It's not what you thought it was. *American Psychologist*, 61, 10-26.
- Myers KM, Ressler KJ, Davis M (2006). Different mechanisms of fear extinction dependent on length of time since fear acquisition. *Learning & Memory*, 13, 216-223.
- Mystkowski JL, Mineka S, Vernon LL, Zinbarg, RE (2003). Changes in caffeine state enhance return of fear in spider phobia. *Journal of Consulting and Clinical Psychology*, 71, 243-250.
- Neumann DL (2008). The effects of context changes on the *reinstatement* of extinguished conditioned behavior in a conditioned suppression task with humans. *Learning and Motivation*, 39, 114-135.
- Niles AN, Mesri B, Burklund LJ, Lieberman MD, Craske MG (2013). Attentional bias and emotional reactivity as predictors and moderators of behavioral treatment for social phobia. *Behaviour Research and Therapy*, 51, 669-679.
- Norton PJ, Price EC (2007). A meta-analytic review of adult cognitive-behavioral treatment outcome across the anxiety disorders. *Journal of Nervous and Mental Disease*, 195, 521-531.

- Norrholm SD, Jovanovic T, Vervliet B, Myers KM, Davis M, Rothbaum BO, Duncan EJ (2006). Conditioned fear extinction and reinstatement in a human fear- potentiated startle paradigm. *Learning and Memory*, 13, 681-685.
- Öhman A (1986). Face the beast and fear the face: Animal and social fears as prototypes for evolutionary analyses of emotion. *Psychophysiology*, 23, 123-145.
- Öhman A, Mineka S (2001). Fears, phobias, and preparedness: Toward an evolved model of fear and fear learning. *Psychological Review*, 108, 483-522.
- Pavlov IP (1927). *Conditioned Reflexes: An Investigation of the Physiological Activity of the Cerebral Cortex*. London: Oxford University Press.
- Peterson, RA, Heilbronner RL (1987). The Anxiety Sensitivity Index: Construct validity and factor analytic structure. *Journal of Anxiety Disorders*, 1, 117-121.
- Pflanzer R (1998). *Biopac Lab Experiments*. McGraw-Hill Science/Engineering/Math.
- Pijlman FTA, Herremans AHJ, van de Kieft J, Kruse CG, van Ree JM (2003). Behavioural changes after different stress paradigms: prepulse inhibition increased after physical, but not emotional stress. *European Neuropsychopharmacology*, 13, 369–380.
- Pine DS, Klein RG (2008). Anxiety disorders. In M. Rutter, D. Bishop, D. S. Pine, S. Scott, J. Stevenson, E. Taylor, A. Thapar (Eds.), *Rutter's child and adolescent psychiatry (5th ed.)* (pp. 628-647). Wiley-Blackwell.
- Rachman SJ (1978). Human fears: A three systems analysis. *Scandinavian Journal of Behaviour Therapy*, 7, 237-245.
- Rachman SJ (1979). The return of fear. *Behaviour Research and Therapy*, 17, 164-166.
- Rachman SJ (1989). The return of fear: Review and prospect. *Clinical Psychology Review*, 9, 147-168.

- Rachman SJ, Whittal M (1989). The effect of an aversive event on the return of fear. *Behaviour Research and Therapy*, 27, 513-520.
- Rescorla RA (2008). Evaluating conditioning of related and unrelated stimuli using a compound test. *Learning & Behavior*, 36, 67-74.
- Rescorla RA, Heth CD (1975). Reinstatement of fear to an extinguished stimulus. *Journal of Experimental Psychology: Animal Behavior Processes*, 1, 88-96.
- Rescorla RA, Wagner AR (1972). A theory of Pavlovian conditioning: Variations in the effectiveness of reinforcement and nonreinforcement. In A. H. Black, W. K. Pokasy (Eds.), *Classical Conditioning II: Current Research and Theory* (pp. 64-99). New York: Appleton-Century-Crofts.
- Rose MP, McGlynn FD (1997). Toward a standard experiment for studying post-treatment return of fear. *Journal of Anxiety Disorders*, 11, 263-277.
- Sandi C, Merino JJ, Cordero MI, Touyarot K, Venero C (2001). Effects of chronic stress on contextual fear conditioning and the hippocampal expression of the neural cell adhesion molecule, its polysialylation, and L1. *Neuroscience*, 102, 329-339.
- Schachtman TR, Brown AM, Miller RR (1985). Reinstatement-induced recovery of a taste-LiCl association following extinction. *Animal Learning & Behavior*, 13, 223-227.
- Schmajuk NA, Larrauri JA, LaBar KS (2007). Reinstatement of conditioned fear and the hippocampus: An attentional-associative model. *Behavioural Brain Research*, 177, 242–253.
- Shaham Y, Erb S, Stewart J (2000). Stress-induced relapse to heroin and cocaine seeking in rats: a review. *Brain Research Review*, 33, 13-33.
- Shearn D, Bergman E, Hill K, Abel A, Hinds L (1990). Facial coloration and temperature responses in blushing. *Psychophysiology*, 27, 687-693.

- Shearn D, Bergman E, Hill K, Abel A, Hinds L (1992). Blushing as a function of audience size. *Psychophysiology*, 29, 431-436.
- Sinha R (2008). Chronic stress, drug use, and vulnerability to addiction. *Annals of the New York Academy of Sciences*, 1141, 105-130.
- Sokol N, Lovibond PF (2012). Cross-US reinstatement of human conditioned fear: return of old fears or emergence of new ones? *Behaviour Research and Therapy*, 50, 313-322.
- Spielberger CD, Gorsuch RL, Lushene RE (1970). Manual for the State-Trait Inventory. *Consulting Psychologists*. Palo Alto, California.
- Steketee G (1993). Social support and treatment outcome of obsessive compulsive disorder at 9-month follow-up. *Behavioral and Cognitive Psychotherapy*, 21, 81-95.
- Steketee G, Lam JN, Chambless DL, Rodebaugh TL, McCullouch CE (2007). Effects of perceived criticism on anxiety and depression during behavioral treatment of anxiety disorders. *Behaviour Research and Therapy*, 45, 11-19.
- Stewart J (2000). Pathways to relapse: the neurobiology of drug and stress induced relapse to drug taking. *Journal of Psychiatry & Neuroscience*, 25, 125-136.
- Stewart J (2008). Psychological and neural mechanisms of relapse. *Philosophical Transactions of The Royal Society of Biological Sciences*, 363, 3147-3158.
- Thornhill R, Thornhill NW (1989). The evolution of psychological pain. In R. Bell (Ed.), *Sociobiology and the social sciences* (pp. 73–103). Lubbock: Texas Tech University Press.
- Tolin DF (2010). Is cognitive–behavioral therapy more effective than other therapies? A meta-analytic review. *Clinical Psychology Review*, 30, 710-720.

- Vansteenwegen D, Iberico C, Vervliet B, Marescau V, Hermans D (2008). Contextual fear induced by unpredictability in a human fear conditioning preparation is related to the chronic expectation of a threatening US. *Biological Psychology*, 77, 39-46.
- Vervliet B, Craske MG, Hermans D (2013). Fear extinction and relapse: state of the art. *Annual Review of Clinical Psychology*, 9, 215-248.
- Wade SL, Monroe SM, Michelson LK (1993). Chronic life stress and treatment outcome in agoraphobia with panic attacks. *The American Journal of Psychiatry*, 150, 1491-1495.
- Wagner AR, Brandon SE (1989). Evolution of a structured connectionist model of Pavlovian conditioning (AESOP). In SB Klein (Ed.) *Contemporary learning theories: Pavlovian conditioning and the status of traditional learning theory* (pp. 149-189). Hillsdale, NJ, England: Lawrence Erlbaum Associates, Inc, xiii, 322 pp.
- Westbrook RF, Jordanova M, McNally G, Richardson R, Harris JA (2002). Reinstatement of fear to an extinguished conditioned stimulus: Two roles for context. *Journal of Experimental Psychology: Animal Behavior Processes*, 28(1), 97-110.
- Wilcox RR, Keselman HJ (2003): Modern robust data analysis methods: Measures of central tendency. *Psychol Methods* 8: 254-274.
- Wilson A, Brooks DC, Bouton ME (1995). The role of the rat hippocampal system in several effects of context in extinction. *Behavioral Neuroscience*, 109, 828-836.